Perspectives in vestibular diagnostics and therapy

Abstract

Vestibular diagnostics and therapy ist the mirror of technological, scientific and socio-economics trends as are other fields of clinical medicine. These trends have led to a substantial diversification of the field of neurotology.
The improvements in diagnostics have been characterized by the introduction of new receptor testing tools (e.g., VEMPs), progress in imaging (e.g., the endolymphatic hydrops) and in the description of central-vestibular neuroplasticity.
The etiopathology of vestibular disorders has been updated by geneticists (e.g., the description of the COCH gene mutations), the detection of structural abnormalities (e.g., dehiscence syndromes) and related disorders (e.g. migraine-associated vertigo).
The therapeutic options were extended by re-evaluation of techniques known a long time ago (e.g., saccus exposure), the development of new approaches (e.g., dehiscence repair) and the introduction of new drug therapy concepts (e.g., local drug delivery).
Implantable, neuroprosthetic solutions have not yet reached experimental safety and validity and are still far away. However, externally worn neuroprosthetic solution were introduced in the rehab of vestibular disorders (e.g., VertiGuard system).
These and related trends point into a medical future which is characterized by presbyvertigo as classical sign of the demographic changes ahead, by shortage of financial resources and a medico-legally overregulated, even hostile environment for physicians in clinical medicine.

Keywords: Vestibular diagnostics, vestibular therapy, presbyvertigo, neurofeedback training

Arneborg Ernst[1]

1 HNO-Klinik, Unfallkrankenhaus Berlin, Germany

1. Socio-economic background for neurotology in Germany today

The modern Western society creates the framework for clinical medicine. The society defines the ethical and legal standards, science and technology contribute to its progress and the health economy has to take care that the political decision makers and the insurance system are willing to cover the costs. So far in theory, but how does the German social and health care system impact neurotology? The following theses should be discussed and considered in more detail:

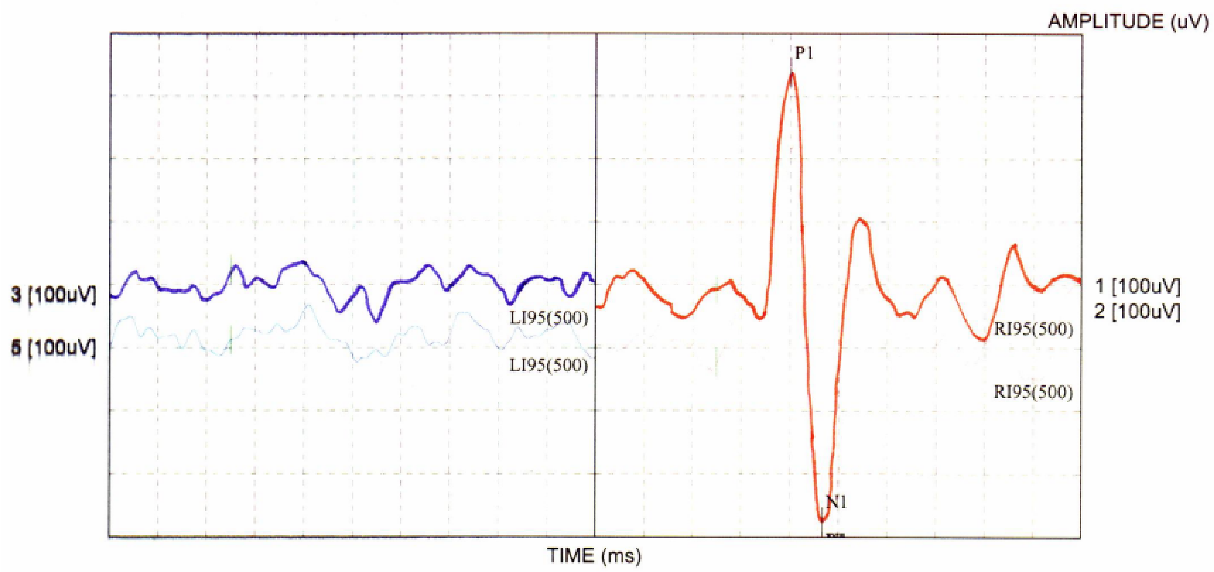
- New diagnostic tools enable a precise diagnostics of each vestibular receptor and individualize therapy.
- Recent progress in imaging and neurogenetics reveal the background of complex vestibular disorders, improve the understanding of central-vestibular one and describe new disease entities.
- Non-invasive therapeutic approaches have been refined and improved and will dominate vestibular therapy despite a re-evaluation of surgical measures.
- The rehabilitation of vestibular disorders and the prevention of falls are major topics in health politics due to a strong demand from the ongoing changes in the epidemiology of the ageing Western societies (the “turning age pyramid”).
- The individualization of our societies with strong demands from single patients is in sharp contrast to the real fiscal expenses for the health economy and will reinforce the “prioritization debate”. This conflict is overshadowed by legal decisions [1] which demand a thorough consideration of all aspects of the patient’s daily life when making treatment decisions.

The following chapters are to explicitly outline these theses. Established therapy standards (e.g., repositioning maneouvers in BPPV) will not be specifically mentioned (this not a textbook), but the developmental lines of neurotology into the future will be pointed out.

2. Recent trends in vestibular diagnostics

2.1. Diagnostics of vestibular diagnostics

The vestibular end organ consists of three semicircular canals (SCC), the otolith organs (maculae sacculi and utriculi) and the endolymphatic sac (ES). Within the last



LATENCIES (ms)						
VEMP						
	P1	N1	P2	N2	P3	N3
1	15.25	21.93	***	***	***	***
2	21.93	21.93	***	***	***	***

WAVEFORMS												
VEMP												
	Date	Record	Intensity	Ear	Rate	Stimulus	Gain	High	Low	Acc	Rej	Epoch
1	09.11.2006	1.2	95dB nHL	Right	5.1/s	500 Hz-A	5000	2 Hz	500 Hz	106	0	100ms
2	09.11.2006	2.2	95dB nHL	Right	5.1/s	500 Hz-A	5000	2 Hz	500 Hz	113	0	100ms
3	09.11.2006	3.1	95dB nHL	Left	5.1/s	500 Hz-A	5000	2 Hz	500 Hz	109	0	100ms
5	09.11.2006	5.1	95dB nHL	Left	5.1/s	500 Hz-A	5000	2 Hz	500 Hz	106	0	100ms

Figure 1: Typical picture of a saccular dsiorder oft he left side (no VEMPs), but normal VEMP pattern oft he right side (P1/N1)

few years, each of those receptors could have been diagnosed separately apart from the formerly well-known, usual tests (e.g., caloric VOR testing, Romberg's, Unterberger's test) [2], [3], [4].

The head thrust test (HTT) was described for the first time by Halmagyi and Curthoys [5] for the lateral SCC, but can be applied in a modified version for the superior and posterior SCC as well [6]. The saccadic eye bulb movement on the diseases side is registered nowadays with the search coil technique [7], [8], but can be used as bedside test as well [9].

While the SCC are largely affected in vestibular neuritis [10], in canal paresis [4], [11], the otolith organs have been described to be affected separately for the first time just recently [3]. These disorders can be accompanying in Meniere's disease (MM), in case of canalolithiasis, in vestibular neuritis, in posttraumatic disorders after blunt head trauma or microsurgery of the ear (e.g., cochlear implantation, CI) [12], [13], [14], [15].

Utricular disorders have the more severe clinical picture with difficulties in walking and continuous dizziness (an increase in body sway in the roll plane) [16], [17]. Those disorders can be screened for with the subjective, haptic vertical test (SHV), but a unilateral, quantitative testing has to perform eccentric rotation (with adjustment of the subjective, visual vertical) [18].

Saccular disorders (Figure 1) are tested for with vestibular evoked mygenic potentials (VEMP) [19], [20]. The saccule is derived phylogenetically from the hearing organ so that it reacts to tonal stimuli which also works in those patients who have a SNHL or deafness even (Table 1). In addition to acoustic stimuli, galvanic, vibratory ones can also be applied [20], [21] and the VCR can also be registered from the SCM (oVEMP) [3].

The VCR arch goes along the superior (utricle) and inferior (saccule) vestibular nerve and is synaptically connected to other nuclei of the CNS, so that central vestibular impairments can lead to a negative test result (e.g. in acoustic neuroma, in neurovascular conflicts within the CPA). This phenomenon can be used as tool in intraoperative, neurophysiological monitoring in otoneurosurgery [19], [22] and in CI surgery [13], [15], [23].

While the postural stability was frequently assessed by dynamic posturography in former times [24], mobile systems are now underway (e.g., VertiGuard® D) to better assess body sway in stance and gait tasks [2], [25]. This is a great advantage since those systems away from the lab situation can better assess the risks of everyday life (e.g., walking over stairs, moving while turning the head) in the prevention of falls and falls-related injuries [26].

For more details on testing the ES see chapter 2.3 and for review [27], [28].

2.2. Diagnostics of central-vestibular disorders

Central vestibular structures have rceived more attention within the scientific community after the description of the parieto-insular, vestibular cortex (PIVC) in the 1990s

Table 1: Average P1/N1 amplitudes in VEMP recordings – elicited by air and bone conduction – with respect to age and gender [due to 20]

	VEMP-amplitude in µV			
age group	air conduction	bone conduction	male	female
20-40	73.8 ± 45.5	60.2 ± 33.2	69.8 ± 51.4	65.8 ± 34.6
41-60	45.0 ± 33.2 *	34.6 ± 17.2 *	37.5 ± 22.9 *	42.1 ± 30.0 *
>60	35.8 ± 20.8 *	35.9 ± 22.9	37.2 ± 18.3	32.8 ± 28.5 *

* indicates significant differences ($p<0.05$)

by PET/fMRI [29]. The results of healthy volunteers could subsequently be transferred to specific vestibular disorders [30], [31]. In this way, mechanisms of neural plasticity underlying the central compensation after vestibular neuritis were described and their interaction with the visual and somatosensory cortex [29], [31]. Moreover, the consequences of isolated CNS deficits (e.g., cerebral infarction, tumours, hemispheric degeneration) on the vestibular system were elucidated [32], [31] and spezial imaging techniques (e.g., "voxel-based morphometry") became a clinical mainstay [33].

2.3. Neuroimaging

The following lines will not cover functional imaging techniques as described in 2.2, but the recent progress in imaging of the petrous bones and surrounding structures.

Those bony structures are examined by thin-sliced CT scans at 1 mm [34], so that minor malformations – e.g., dehiscence syndromes of the SCC (SCDS) [35] – can be exactly localized and categorized [36] (Figure 2). The same holds true for multiple labyrinthine malformations [34] so that the dehiscence syndromes might be hereditary (e.g., mutations of the COCH gene) [37]. Koo demonstrated recently that a prominent Sinus petrosus superior can also induce a SCDS [38].

Another new application for bony structures is rotational volume tomography (RVT) which is particulaerly helpful in localizing CI electrodes and to reduce the X ray load in children [39].

When using MRI technology to evaluate fluid-filled spaces (e.g., the cochlea), the FLAIR sequences (in T1/T2 weighted pictures) have become most important [40]. The same holds true for the internal auditory canal (IAC) to differentiate cholesterol granulomas [41], to detect an endolymphatic hydrops [42], [43] or to localize tumours of the ES [44]. For years, ECoG has been the neurophysiological method-of-choice before those MRI techniques became available [45], [46]. When the IAC and the CPA are affected by haemangiomas, a neuritis which has to be differentiated against a neurovascular conflict or a small acoustic neuroma, FLAIR sequences are evenly helpful [40].

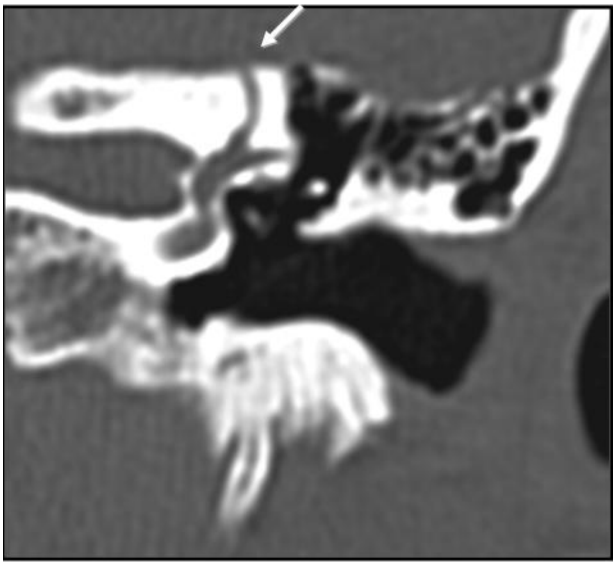


Figure 2: Dehiscence of the superior semicricular canal (SCDS) in thin-slice CT of the petrous bone

3. Recent trends in vestibular research

The refined diagnostic techniques have contributed to a large extent to deeper insights into the pathophysiology of the vestibular system. New, hitherto unclassified disorders were defined (e.g., migraine-associated vertigo, MAV). Those patients complain of dizziness and vertiginous spells [47] without a pathological profile upon neurotological testing [48]. MAV is frequently associated with classical migraine due to the HIS classification and visual and vestibular disorders can occur without headache [47]. A hereditary background seems to be likely, but the genes responsible have not yet been identified [49]. The medical therapy of MAV corresponds to the guidelines of migraine treatment [50], [51].

In contrast to MAV, genetic studies have revealed that SCDS is linked to a mutation of the COCH gene [37]. Additional genetic studies could evidence that connexion-26 mutations can be responsible for an otolith dysfunction [52] and are associated to the HID/KID syndrome [53]. A close correlation between the geno- and phenotype in NF-2 patients could also be established [54]. Those findings were the most important contributions to the genetics of vestibular disorders since the small number of diseased people make linkage studies hardly feasible (with the exception of ataxia) [55].

Prebyvertigo is a well-known, but not yet epidemiologically fully characterized entity. In contrast to presbycusis which has been investigated in detail, presbyvertigo should become a mainstay in vestibular research over the next decade. The prevention of falls [56] is one global task of the health care systems in the Western world. Several features are associated with presbyvertigo, e.g., ageing of the vestibular receptors and the visual system [57], the reduced strength of the musculo-skeletal system, declining speed of neuronal processing, cardiovascular co-morbidity [58].

The extended diagnostic tools described above have accumulated knowledge about the nature of posttraumatic vertigo (e.g., after blunt head trauma, due to microsurgery of the middle ear or cochlear implantation). It was shown that the dizziness or rotational vertigo complaints can be based on separate otolith disorders, SCC paresis, delayed EL hydrops and can occur parallel (e.g., utricular loss and BPPV) or subsequently (e.g., UVL and delayed EL hydrops after 6 months) [12], [14], [59], [16], [17], [23].

4. Recent trends in the therapy of vestibular disorders

4.1. Surgical therapy

The description of dehiscence syndromes of the SCC has led to the development of surgical techniques to close those "third windows" [60]. The direct, transmastoidal approach will lead to a complete remission of the vertiginous complaints [61] with preserved hearing [62], [63]. These "canal plugging" techniques are based on the occlusion of a longer distanced segment of the SCC affected. This surgical approach can also be applied in cupulolithiasis as sequaelae of a re-occuring canalolithiasis [62]. An alternative, middle fossa approach has been described, particularly for SCDS [64].

A recently published paper suggests the occlusion of the lateral SCC for Meniere's disease and reports good hearing preservation with relief of vertiginous spells [65]. In general, the scientific community has re-evaluated the benefits of endolymphatic sac surgery in Meniere's disease (or in delayed EL hydrops) over the last decade [66]. The recently published COCHRANE review on this topic [67] does not yet cover this development [68], [69] which was initiated by a statistical re-evaluation of old, previously published data [70], [71], [72]. This was lately confirmed by a series from the House Ear Institute which reports the long-term benefits of endolymphatic sac surgery and the advantages against local gentamycin therapy with respect to vertigo relief and preservation of hearing [72].

This confirms also the classical Tübingen concept of Meniere's treatment (Figure 3) as published by Klaus Jahnke as early as 1984 in Germany [73]. The surgical concepts can be modified and transferred to the treatment of EL sac tumours.

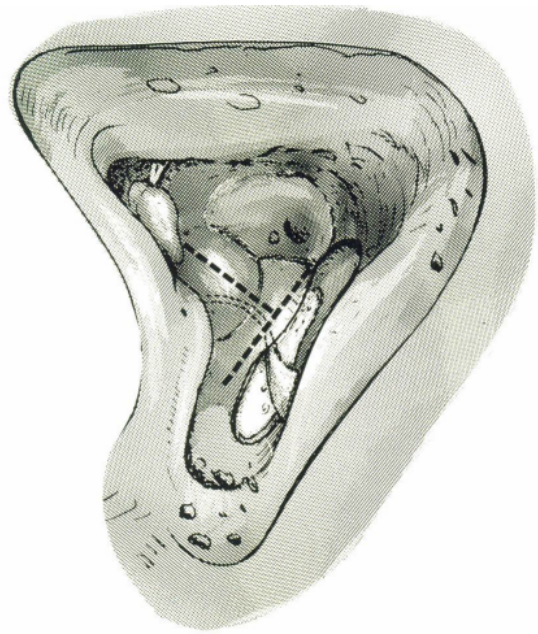


Figure 3: Surgical approach for the exposition oft he endolymphatic sac (due to PLESTER)

The microsurgical removal of an acoustic neuroma was the most reasonable suggestion to a patient when she/he was counselled in the 80s and 90s. Nowadays, stereotactic radiotherapy on one hand [74] and the knowledge about the growth patterns of small, intracanalicular tumours have to be taken into account on the other hand [75]. Moreover, medicolegal experts demand that the whole range of possible treatment alternatives (e.g., including herbal medicine) have to be explained [75], [76]. The great expectations of the patients nowadays (no surgery, full preservation of all cranial nerves, minimal therapy, no surgery) are in sharp contrast to the real clinical outcome, regardless of the therapy [74]. The litigation risk for the MD who has to counsel the patient is greatly increased when the patient denies any therapy (and was not fully counselled about the consequences) [76]. A careful "watch & scan" approach in small tumours can be an alternative for those patients [75].

If it comes to implantable neuroprosthetics of the vestibular system as known from the auditory system, a clinical application is not within sight because of the complexicity of the stimuli to be applied [77], [78].

4.2. Drug therapy

Drug therapy of vestibular disorders has considered a number of substance classes which have not dramatically changed over the last few years. However, the local application of drugs into the middle ear (via the tympanic membrane or drug delivery system) [79] is an extension

of the therapeutic applications and can be easily combined with surgery, e.g., in bilateral Meniere's disease [80], [81], [82].
The application of intravenous corticosteroids in vestibular neuritis should begin as soon as possible after the onset of the disease since this should lead to a complete remission in 62% of the patients [10].
In Meniere's disease, the local application of corticosteroids and/or genatmycin is an integral part of drug therapy [82], but off-label use of betahistidin has proven effectiveness as well [66].
In MAV, the prophylaxis is of vital importance (done as in classical migraine) with the additional application of triptanes in the acute attack [51].
Experimental studies with isolated vestibular hair cells have confirmed that the antivertiginous effect of calcium antagonists (cinnaricine) is also based on a modulation of the transmitter output within the labyrinth [83].

5. Recent trends in vestibular rehabilitation

Uncompensated vestibular disorders (e.g., after vestibular neritis or removal of an acoustic neuroma) [4], patients with multiple lesions or after stroke have undergone so-called vestibular exercises for decades [11], [84], which is done alone or under supervision [85]. The main targets of those approaches are to improve central compensation and to activate the vestibulo-spinal and opto-vestibular pathways [86]. The exercises should be preceded by intense counselling without drug therapy in parallel [84]. In the elderly, home exercises can improve postural stability and reduce the risk to fall [87].

Figure 4: Vibrotactile neurofeedback training system VertiGuard® in action

A new trend in vestibular rehab is the introduction of neurofeedback-based systems [25]. The stance and gait exercises of the patient are accompanied by a second sensory stimulus which provides additional information on the spatio-temporal movements and the body sway (Figure 4). This sensory stimulus can be viobrotactile, but acoustic feedback has also been introduced (with the problem of SNHL in the elderly) [88], [89]. Acoustic feedback, however, is lab-based which limits its applications. The galvanic feedback has no proven efficacy so that this approach seems to be ineffective [90].
The presently available vibrotactile system (VertiGuard®) has proven efficency (Figure 5) with proven long-term stability as demonstrated in patients after acoustic neuroma removal, in presbyvertigo and Parkinson's disease [26], [91].

6. Future perspectives. Quo vadis neurotology?

The socio-economic background of clinical medicine in general and of neurotology in particular – as outline in the introduction – has led to the situation in Germany that:

- despite remarkable progress in diagnostisc, these new tools are mainly lab tools as yet (e.g., otolith diagnostics) since they are not at all reimbursed,
- modern neuroimaging (MRA, CT of the petrous bones) has a strictly limited budget so that it is not always applied if required,
- modern vestibular microsurgery is limited to very few neurotological centers since the surgical procedures are not very cost-efefctive under the newly implement DRG rules,
- prevention of falls is a major topic of the health economy, but its practical management (e.g., vestibular exercises or similar approaches) has to be covered by the individual elderly,
- even neurological departments suffer from this low-budget approach to neurotology by the health care system (e.g., stroke unit are much more cost-effective under DRG rules).
- the frequently demanded gold standard of evidence-based medicine (double-blind, placebo-controlled) [67] will be hard to fulfil in neurotology – also in the future – due to the small number of patients for the single disorders (apart from BPPV) and for ethical concerns.
- quality control can be easily demanded, but is not easy to implement under the current constraints.

The strong and egocentric demands of some young patients could be best channelled into a service culture of medical care where you get what you pay for, worst-case scenario is a decline of neurotological service down to the level of GPs. We should go for the first and fight against the latter.

Acknowledgements

Supported by BMWiT (FKz. KF2227701), BMBF (FKz. 01EZ0754), the Sonnenfeld Foundation (Berlin) and ZNS Foundation (Bonn).

Figure 5: Typical evaluation of pre/post results after neurofeedback training with stance and gait tasks

References

1. Andreas M, Debong B, Bruns W. Handbuch Arztrecht. Nomos, Baden-Baden.
2. Allum JHJ, Carpenter MG. A speedy solution for balance and gait analysis: angular velocity measured at the centre of body mass. Current opinion in Neurology. 2005;18:15-21. DOI: 10.1097/00019052-200502000-00005
3. Helling K. Funktionsstörungen der Otolithenorgane: Klinische Aspekte und Therapieoptionen. HNO. 2008;56:996-1002. DOI: 10.1007/s00106-008-1806-y
4. Halmagyi GM, Weber KP, Curthoys IS. Vestibular function after acute vestibular neuritis. Restor Neurol Neurosci. 2010;28:37-46.
5. Halmagyi GM, Curthoys IS. A clinical sign of canal paresis. Arch Neurol. 1998;45:737-739. DOI: 10.1001/archneur.1988.00520310043015
6. Viciana D, Ferrer J, Palma MJ, Zapata C, Lopez-Escamez JA. Dynamic visual acuity during head-thrust test in canal planes in healthy subjects and patients with vestibular neuritis. Acta Otolaryngol. 2010;130(11):1260-6.
7. Lehnen N, Aw ST, Todd MJ, Halmagyi GM. Head impulse test reveals residual semicircular canal function after vestibular neurectomy. Neurology. 2004;62:2294-2296.

8. MacDougall HG, Weber KP, McGarvie LA, Halmagyi GM, Curthoys IS. The video head impulse test: diagnostic accuracy in peripheral vestibulopathy. Neurology. 2009;73:1134-41. DOI: 10.1212/WNL.0b013e3181bacf85
9. Jorns-Häderli M, Straumann D, Palla A. Accuracy of the bedside head impulse test in detecting vestibular hypofunction. J Neurol Neurosurg Psychiatry. 2007;78:1113-1118. DOI: 10.1136/jnnp.2006.109512
10. Strupp M, Brandt T. Vestibular neuritis. Semin Neurol. 2009;29:509-519. DOI: 10.1055/s-0029-1241040
11. Brandt T, Huppert T, Hüfner K, Zingler VC, Dieterich M, Strupp M. Long-term course and relapses of vestibular balance disorders. Restor Neurol Neurosci. 2010;28:69-82.
12. Basta D, Clarke A, Ernst A, Todt I. Stance performance under different sensorimotor conditions in patients with post-traumatic otolith disorders. J Vest Res. 2007;17:25-32.
13. Basta D, Singbartl F, Todt I, Clarke A, Ernst A. Vestibular rehabilitation by auditory feedback in otolith disorders. Gait & Posture. 2008. DOI: 10.1016/j.gaitpost.2008.01.006 DOI: 10.1016/j.gaitpost.2008.01.006
14. Basta D, Todt I, Ernst A. Postural control in otolith disorders. Human Movement Sciences. 2005;24:268-279. DOI: 10.1016/j.humov.2005.04.002
15. Jacot E, Van Den Abbeele T, Debre HR, Wiener-Vacher SR. Vestibular impairments pre- and post-cochlear implant in children. Int J Pediatr Otorhinolaryngol. 2009;73:209-217. DOI: 10.1016/j.ijporl.2008.10.024
16. Ernst A, Basta D, Seidl RO, Todt I, Scherer H, Clarke A. Management of posttraumatic vertigo. Otolaryngol Head & Neck Surg. 2005;132:554-558. DOI: 10.1016/j.otohns.2004.09.034
17. Ernst A, Seidl RO, Nölle C, Pudszuhn A, Ganslmeier A, Ekkernkamp A, Mutze A. Hör- und Gleichgewichtsstörungen nach Kopfanpralltraumen. Trauma Berufskrankheit. 2001;3:27-31. DOI: 10.1007/s100390000343
18. Helling K, Schönfeld U, Scherer H, Clarke AH. Testing utricular function by means of on-axis rotation. Acta oto-laryngol (Stockh). 2006;126:587-593. DOI: 10.1080/00016480500450008
19. Basta D, Todt I, Ernst A. Normative data for: P1./N1 latencies of VEMP induced by air- or bone conducted tone burst. Clin Neurophysiol. 2005;116:2216-2219. DOI: 10.1016/j.clinph.2005.06.010
20. Basta D, Todt I, Ernst A. Characterization of age-related changes in VEMPs. J Vest Res. 2007;17:93-98.
21. Ernst A, Todt I, Basta D. The application of VEMP in otoneurosurgery. Otolaryngol Head & Neck Surg. 2006;135:286-290. DOI: 10.1016/j.otohns.2006.03.006
22. Ernst, A. Chirurgische Behandlung von Gleichgewichtserkrankungen. HNO Praxis heute. 2007;27:79-85.
23. Todt I, Basta D, Ernst A. Does the surgical approach in cochlear implantation influence the occurrence of postoperative vertigo? Otolaryngol Head & Neck Surg. 2008;138:8-12. DOI: 10.1016/j.otohns.2007.09.003
24. Allum JHJ, Zamani F, Aitkin AL, Ernst A. Differences between trunk sway characteristics foam support surface and on the Equitest ankle-sway-referenced support surface. Gait & Posture. 2002;16:264-270. DOI: 10.1016/S0966-6362(02)00011-5
25. Basta D, Ernst A. Moderne Rehabilitation von Gleichgewichtsstörungen mit Hilfe von Neurofeedback-Trainingsverfahren. HNO. 2008;56:990-995. DOI: 10.1007/s00106-008-1805-z
26. Basta D, Ernst A. Neurofeedback-based vestibular rehab in vestibular disorders. J Vest Res. 2010; (submitted).
27. Yamamoto M, Teranishi M, Naganama S, Otake H, Sugiura M, Iwata T, Yoshida T, Katayama N, Nakata S, Sone M, Nakashima T. Relationship between the degree of endolymphatic hydrops and electrocochleography. Audiol Neurootol. 2010;15:254-260. DOI: 10.1159/000258681
28. Perez-Fernandez N, Montes-Jovellar L, Cervera-Paz J, Domenech-Vadillo E. Auditory and Vestibular Assessment of Patients with Ménière's Disease Who Suffer Tumarkin Attacks. Audiol Neurootol. 2010;15:399-406. DOI: 10.1159/000310899
29. Brandt T, Dieterich M. The vestibular cortex. Its locations, functions, and disorders. Ann N Y Acad Sci. 1999;871:293-312. DOI: 10.1111/j.1749-6632.1999.tb09193.x
30. Dieterich M. Functional brain imaging. A window into the visio-vestibular systems. Curr Opin Neurol. 2007;20:12-18. DOI: 10.1097/WCO.0b013e328013f854
31. Dieterich M, Brandt T. Funtional imaging of peripheral and central vestibular disorders. Brain. 2008;131:2538-2552. DOI: 10.1093/brain/awn042
32. Kim HA, Lee H. Isolated vestibular nucleus infarction mimicking acute peripheral vestibulopathy. Stroke. 2010;41:1558-1560. DOI: 10.1161/STROKEAHA.110.582783
33. Zu Eulenburg P, Stoeter P, Dieterich M. Voxel-based morphometry depicts central compensation after vestibular neuritis. Ann Neurol. 2010;68:241-249. DOI: 10.1002/ana.22063
34. Wang JR, Parmes LS. Superior semicircular canal dehiscence associated with external, middle, and inner ear abnormalities. Laryngoscope. 2010;120:390-393. DOI: 10.1002/lary.20726
35. Minor LB, Solomon D, Zinreich JS, Zee DS. Sound- and/or pressure-induced vertigo due to bone dehiscence of the superior semicircular canal. Arch Otolaryngol Head neck Surch. 1998;124:249-258.
36. Chi FL, Ren DD, Dai CF. Variety of audiologic manifestations in patients with superior semicircular canal dehiscence. Otol Neurotol. 2010;31:2-10. DOI: 10.1097/MAO.0b013e3181bc35ce
37. Hildebrand MS, Tack D, Deluca A, Hur IA, Van Rybroek JM, McMordie SJ, Muilenburg A, Hoskinson DP, Van Camp G, Pensak ML, Stoper IS, Huygen PL, Casavant TL, Smith RJ. Mutation in the COCH gene is associated with superior semicircular canal dehiscence. Am J Med Genet. 2009;149A:280-285. DOI: 10.1002/ajmg.a.32618
38. Koo JW, Hong SK, Kim DK, Kim JS. Superior semicircular canal dehiscence syndrome by the superior petrosal sinus. J Neurol Neurosurg Psychiatry. 2010;81:465-467. DOI: 10.1136/jnnp.2008.155564
39. Kurzweg T, Dalchow CV, Bremke M, Majdani O, Kureck I, Knecht R, Werner JA, Teymoortash A. The value of digital volume tomography in assessing the position of cochlear implant arrays in temporal bone specimens. Ear Hear. 2010;1:413-419. DOI: 10.1097/AUD.0b013e3181d3d6b6
40. Petrovic BD, Futterer SF, Hijaz T, Russell EJ, Karagianis AG. Frequency and diagnostic utility of intralabyrinthine FLAIR hyperintensitiy in the evaluation of internal auditory canal and inner ear pathology. Acad Radiol. 2010;17:992-1000. DOI: 10.1016/j.acra.2010.04.002
41. Kim HC, An YS, Ahn JH. Petrous apex cholesterol granuloma presenting as endolymphatic hydrops: a case report. Clin Exp Otorhinolaryngol. 2009;2:151-154. DOI: 10.3342/ceo.2009.2.3.151
42. Kasai S, Teranishi M, Katayama N, Sugiura M, Nakata S, Sone M, Naganawa S, Nakashima T. Endolymphatic space imaging in patients with delayed endolymphatic hydrops. Acta Otolaryngol. 2009;129:1169-1174. DOI: 10.3109/00016480802691143

43. Naganawa S, Ishihara S, Iwano A, Sone M, Nakashima T. Three-dimensional (3D) visualization of endolymphatic hydrops after intratympanic injection of Gd-DTPA: optimization of a 3D-real inversion-recovery turbo spin-echo (TSE) sequence and application of a 32-channel head coil at 3T. J Magn Reson Imaging. 2010;31:210-214. DOI: 10.1002/jmri.22012

44. Diaz RC, Amjad EH, Sargent EW, Larouere MJ, Shaia WT. Tumors and pseudotumors of the endolymphatic sac. Skull Base. 2007;17:379-393. DOI: 10.1055/s-2007-991116

45. Ge X, Shea JJ. Transtympanic electrocochleography: a 10-year experience. Otol Neurotol. 2001;22:465-470.

46. Katayama N, Yamamoto M, Teranishi M, Naganawa S, Nakata S, Sone M, Nakashima T. Relationship between endolymphatic hydrops and vestibular-evoked myogenic potential. Acta Otolaryngol. 2010;130:917-923. DOI: 10.3109/00016480903573187

47. Neuhauser H, Leopold M, von Brevern M, Arnold G, Lempert T. The interrelations of migraine, vertigo, and migrainous vertigo. Neurology. 2001;56:436-441.

48. Cha YH. Migraine-associated vertigo: diagnosis and treatment. Semin Neurol. 2010;30:167-174. DOI: 10.1055/s-0030-1249225

49. Cha YH, Kane MJ, Baloh RW. Familial clustering of migraine, episodic vertigo, and Ménière's disease. Otol Neurotol. 2008;29:93-96. DOI: 10.1097/mao.0b013e31815c2abb

50. Baier B, Winkenwerder E, Dietrich M. “Vestibular migraine”: effects of prophylactic therapy with various drugs. A retrospective study. J Neurol. 2009;256:436-442. DOI: 10.1007/s00415-009-0111-3

51. Brandt T, Zwergal A, Strupp M. Medical treatment of vestibular disorders. Expert Opin Pharmacother. 2009.

52. Todt I, Hennies HC, Basta D, Ernst A. Vestibular dysfunction in patients with mutations of connexin 26. Neuroreport. 2005;16:1179-1181. DOI: 10.1097/00001756-200508010-00009

53. Todt I, Hennies HC, Küster W, Smolle J, Rademacher G, Mutze S, Basta D, Seidl RO, Ernst E. Neurotological and neuroanatomical changes in the Connexin-26 related HID/KID syndrome. Audiol Neuotol. 2006;11:242-248. DOI: 10.1159/000093110

54. Selvanathan SK, Shenton A, Ferner R, Wallace AJ, Huson SM, Ramsden RT, Evans DG. Further genotype-phenotype correlations in neurofibromatosis 2. Clin Genet. 2010;77:163-170. DOI: 10.1111/j.1399-0004.2009.01315.x

55. Jen JC. Recent advances in the genetics of recurrent vertigo and vestibulopathy. Curr Opin Neurol. 2008;21:3-7. DOI: 10.1097/WCO.0b013e3282f41ca0

56. Furman JM, Raz Y, Whitney SL. Geriatric vestibulopathy assessment and management. Curr Opin Otolaryngol Head Neck Surg. 2010;18(5):386-91. DOI: 10.1097/MOO.0b013e32833ce5a6

57. Walther LE, Westhofen M. Presbyvertigo-aging of otoconia and vestibular sensory cells. J Vestib Res. 2007;17:89-92.

58. Katsarkas A. Dizziness in aging: the clinical experience. Geriatrics. 2008;63:18-20.

59. Basta D, Todt I, Göpel F, Ernst A. Loss of saccular function after cochlear implantation. Audiology & Neurotology. 2008;13:187-192. DOI: 10.1159/000113509

60. Agrawal SK, Parnes LS. Human experience with canal plugging. Ann N Y Acad Sci. 2001;942:300-305. DOI: 10.1111/j.1749-6632.2001.tb03754.x

61. Crane BT, Lin FR, Minor LB, Carey JP. Improvement in autophony symptoms after superior canal dehiscence repair. Otol Neurotol. 2010;31:140-146. DOI: 10.1097/MAO.0b013e3181bc39ab

62. Seo T, Hashimoto M, Saka N, Sakagami M. Hearing and vestibular functions after plugging surgery for the posterior semicircular canal. Acta Otolaryngol. 2009;129:1148-1152. DOI: 10.3109/00016480802609582

63. Fiorino F, Barbieri F, Pizzini FB, Beltramello A. A dehiscent superior semicircular canal may be plugged and resurfaced via the transmastoid route. Otol Neurotol. 2010;31:136-139. DOI: 10.1097/MAO.0b013e3181b76b9e

64. Westhofen M. Surgical treatment of vestibular vertigo: methods and indications. HNO. 2008;56:1003-1010. DOI: 10.1007/s00106-008-1807-x

65. Charpiot A, Rohmer D, Gentine A. Lateral semicircular canal plugging in servere Ménière's disease: a clinical prospective study about 28 patients. Otol Neurotol. 2010;31:237-240. DOI: 10.1097/MAO.0b013e3181ca85a2

66. Westhofen M. Menière's disease: evidence and controversies. HNO. 2009;57:446-454. DOI: 10.1007/s00106-009-1915-2

67. Pullens B, Giard JL, Verschuur HP, van Benthem PP. Surgery for Ménière's disease. Cochrane Database Syst Rev. 2010;20: CD005395.

68. Park JJ, Chen YS, Westhofen M. Impact of endolymphatic mastoid shunt surgery on saccule and lateral semicircular canal function. Eur Arch Otorhinolaryngol. 2010;267(1):21-6. DOI: 10.1007/s00405-009-0998-4

69. Hu A, Parnes LS. 10-year review of endolymphatic sac surgery for intractable meniere disease. J Otolaryngol Head Neck Surg. 2010;39:415-421.

70. Durland WF Jr, Pyle GM, Connor NP. Endolymphatic sac decompression as a treatment for Meniere's disease. Laryngoscope. 2005;115:1454-1457. DOI: 10.1097/01.mlg.0000171017.41592.d0

71. Ghossaini SN, Wazen JJ. An update on the surgical treatment of Meniere's disease. J Am Acad Audiol. 2006;17:38-44. DOI: 10.3766/jaaa.17.1.5

72. Lee L, Pensak ML. Contemporary role of endolymphatic mastoid shunt surgery in the era of transtympanic perfusion strategies. Ann Otol Rhinol Laryngol. 2008;117(12):871-5.

73. Cohen JE, Spektor S, Valarezo J, Felling Y, Umansky F. Endolymphatic sac tumor: staged endovascular-neurosurgical approach. Neurol Res. 2003;25:237-240. DOI: 10.1179/016164103101201436

74. Bassim MK, Berliner KI, Fisher LM, Brackmann DE, Friedman RA. Radiation therapy for the treatment of vestibular schwannoma: a critical evaluation of the state of the literature. Otol Neurotol. 2010;31:567-573.

75. Cheung SW, Aranda D, Driscoll CL, Parsa AT. Mapping clinical outsomes expectations to treatment decisions: an application to vestibular schwannoma management. Otol Neurotol. 2010;31:284-293. DOI: 10.1097/MAO.0b013e3181cc06cb

76. Bruns W. Der Patient muss über das Risiko der Nichtbehandlung ausreichend aufgeklärt werden. ArztRecht. 2010;45:183-187.

77. Wall C, Weinberg MS. Balance prosthesis for postural control. IEEE BME. 2001;22:84-90.

78. Merfeld DM, Priesol A, Lee D, Lewis RF. Potential solution to several vestibular changes facing clinicians. J Vestib Res. 2010;20:71-77.

79. Salt AN, Plontke SK. Principles of local drug delivery to the inner ear. Audiol Neurootol. 2009;14:350-360. DOI: 10.1159/000241892

80. Gouveris H, Lange G, Mann WJ. Intratympanic gentamicin treatment after endolymphatic sac surgery. Acta Otolaryngol. 2005;125:1180-1183. DOI: 10.1080/00016480510043909

81. Nabi S, Parnes LS. Bilateral Ménière's disease. Curr Opin Otolaryngol Head Neck Surg. 2009;17:356-362. DOI: 10.1097/MOO.0b013e3283304cb3

82. Nguyen KD, Minor LB, Della Santina CC, Carey JP. Vestibular function and vertigo control after intratympanic gentamicin for Ménière's disease. Audiol Neurootol. 2009;14:361-372. DOI: 10.1159/000241893

83. Hassler T, Homann G, Duong Dinh TA, Jüngling E, Westhofen M, Lückhoff A. Pharmacological modulation of transmitter release by inhibition of pressure-dependent potassium currents in vestibular hair cells. Naunyn Schmiedebergs Arch Pharmacol. 2000;380:531-538. DOI: 10.1007/s00210-009-0463-3

84. Bronstein AM, Lempert T. Management of the patient with chronic dizziness. Restor Neurol Neurosci. 2010;28:83-90.

85. Szturm T, Ireland DJ, Lessing-Turner M. Comparison of different exercise programs in the rehabilitation of patients with chronic peripheral vestibular dysfunction. J Vest Res. 1994;4(6):461-479.

86. Horak FB. Postural compensation for vestibular loss and implications for rehabilitation. Restor Neurol Neurosci. 2010;28:57-68.

87. Kamide N, Shiba Y, Shibata H. Effects on balance, falls, and bone mineral density of a home-based exercise program without home visits in community-dwelling elderly women: a randomized controlled trial. J Physiol Anthropol. 2009;28:115-122. DOI: 10.2114/jpa2.28.115

88. Hegemann J, Honegger F, Kupper M, Allum JHJ. The balance control of bilateral peripheral vestibular loss subjects and its improvement with auditory prosthetic feedback. J Vest Res. 2005;15:1-8.

89. Ernst A, Singbartl F, Basta D, Seidl RO, Todt I, Eisenschenk A. Postural stabilization through auditory feedback training in patients with posttraumatic otolith disorders: a pilot study. J Vest Res. 2008;17:137-147.

90. Danilov YP, Tyler ME, Skinner KL, Hogle RA, Bach-Y-Rita P. Efficacy of electrotactile vestibular substitution in patients with peripheral and central vestibular loss. J Vest Res. 2007;17:119-130.

91. Basta D, Steinhagen-Thiessen E, Gövercin M, Ernst A. Ageing effects in human postural control: Normative data to predict falls. Gait & Posture. 2010; (in press).

Corresponding author:

Prof. Dr. Arneborg Ernst
HNO-Klinik, Unfallkrankenhaus Berlin,
Warener Str. 7, 12683 Berlin, Germany,
Tel. +49-30-5681-4301, Fax +49-30-5681-4303
arneborg.ernst@ukb.de

Please cite as

Ernst A. Perspectives in vestibular diagnostics and therapy . GMS Curr Top Otorhinolaryngol Head Neck Surg. 2011;10:Doc05.
DOI: 10.3205/cto000078, URN: urn:nbn:de:0183-cto0000783

This article is freely available from

http://www.egms.de/en/journals/cto/2011-10/cto000078.shtml

Published: *2012-04-26*