Vertigo, Dizziness, and Tinnitus After Otobasal Fractures

Lóránt Heid,[1] Claus-Frenz Claussen,[3] Michael Kersebaum,[3] Elemér Nagy,[1] Gábor Bencze,[1] and Beáta Bencsik[2]

[1]Central Military Hospital and [2]Department of Otorhinolaryngology Head and Neck Surgery, Semmelweis University, Budapest, Hungary, and [3]Neurootologisches Forschungsinstitut der 4-G-Forschung e.V., Bad Kissingen, Germany

Abstract: Some 15% of temporal bone fractures are produced by blows to the occiput. The fracture line begins in the posterior fossa, at or near the foramen magnum, and crosses the petrous ridge through the internal auditory canal or the otic capsule. Thus, it is called a *transverse fracture*. In cases of transverse fractures of the temporal bone, due to automobile accidents or other causes of head injury, the labyrinth is involved more frequently than in longitudinal fractures. Severe vertigo with severe or total hearing loss is not uncommon in such injuries. In milder injuries, labyrinthine "concussion" may occur, with transitory auditory-vestibular symptoms. The force that causes the fracture is so great that it not only fractures the base of the skull but may cause a lesion of the brainstem, resulting in a combined peripheral and central lesion. We evaluated 61 patients (50 [81.97%] male, 11 [18.03%] female) with neurootological complaints of sequelae of otobasal fractures. Of these, 40.98% complained of tinnitus and 52.82% of hearing loss. Reviewing our experimental neurootometric investigations, we identified pathological processes on 75.41% of the butterfly calorigrams and 72.13% of the stepping craniocorpograms, as well as in 32.79% and 39.34% of subjects on right- and left-ear bone-conduction audiometry, respectively.

Key Words: craniocorpography; dizziness; electronystagmography; hearing loss; temporal bone fractures; vertigo

Anatomically speaking, a fracture is a break or a crack in a bone or in ossified cartilage. However, when the skull is subject to fracture, we can expect not only local complications but general cephalic complications due to a concussion of the adjacent brain structures or of the entire brain. At the lower part of the temporal bone, the middle ear is cracked open from above. The mastoid process and the temporal bone form the otobasis of the human skull. The temporal bone contains the ear with its organs of hearing and balance. Also, the facial nerve passes through this area. Within these canals are hair cells (i.e., mechanoreceptors similar to those that form the organ of Corti). Diseases of the inner ear include disturbances caused by injuries.

Otobasal fractures are subdivided into longitudinal and transverse fractures, according to the x-ray findings. However, affected patients are also differentiated into those suffering posttraumatically solely from hearing disturbances or solely from equilibrium disturbances and the major group suffering from hearing disturbances together with vertigo and disequilibrium. By means of this study, we evaluated 61 patients who suffered otobasal fracture due to a traumatic event.

PATIENTS AND METHODS

From our neurootological data bank (Neurootological Data Evaluation–Claussen [NODEC]), we chose 61 patients suffering from a posttraumatic otobasal

Reprint requests: Dr. Lóránt Heid, Central Military Hospital, Róbert-Károly krt. 44, H-1134 Budapest, Hungary.

This article is based on a lecture presented at the Thirtieth Congress of the Neurootological and Equilibriometric Society, Porto, Portugal, April 3–5, 2003. This study was sponsored by Grant Projekt: D. 1417, through the LV A Baden-Württemberg, Stuttgart, Germany.

fracture. The complaints of these patients have been reported and recorded by our systematic neurootological history chart (i.e., the NODEC). This chart contains questions regarding vertigo, nausea, hearing complaints (e.g., loss of hearing and tinnitus), visual disturbances, taste and smell disturbances, and disturbances of the facial nerve, as well as questions about major disease backgrounds (e.g., trauma, cardiovascular disorders, diabetes mellitus, kidney disorders). Our drug treatment also was recorded. Additionally, we questioned the patients for harmful habits, such as smoking and drinking.

All patients underwent a systematic neurootometric and equilibriometric analysis that included neurosensory network analysis by means of polygraphic electronystagmography (ENG) together with electrocardiographic (ECG) recordings and vestibular spinal test recordings by craniocorpography (CCG), for charting head and trunk movements.

In comparing the test results of the weaker supraliminal caloric warm test with the stronger supraliminal perrotatory stimulus, we can establish the so-called vestibular stimulus response intensity comparison (VESRIC). This combination of vestibuloocular nystagmus tests provides us with a tool for estimating the dynamics of processing and switching within the brainstem and higher centers of vestibuloocular nystagmus regulation in terms of parallel action and of recruitment and decruitment phenomena.

Testing of the retinoocular pathways include optokinetic tracking tests on one hand and optokinetic nystagmus evaluation on the other. However, these tests are not included in the statistical part of this study. For testing the vestibulospinal pathways, we used a CCG recording of the head and shoulders. The images received appeared strongly as radar images of a head and trunk floating through time and space.

RESULTS

This study centered on a special sample of 61 patients suffering from otobasal fractures. For this patient group, we have complete individual descriptions within our files. In addition, we have set up a NODEC that comprises a list and statistical analysis of the entire sample of 61 patients. Therein, we have noted descrip-

Table 2. Subjective History of Study Patients Suffering from Otobasal Fracture

Symptom	Percentage of Patients Affected
Vestibular symptoms	
Instability	39.34
Blackout	26.23
Falling	22.95
Rotating	32.79
Lifting	8.20
Rocking	39.34
Vegetative or nausea symptoms	
Collapse	1.64
Vomitus	8.20
Retching	1.64
Malaise	26.23
Sweating	4.92
Duration of single vertigo attacks	
Days	1.64
Hours	8.20
Minutes	27.87
Seconds	52.46
Visual disturbances	
Oscillopsia II	1.64
Amaurosis	0.0
Oscillopsia, jerking	1.64
Oscillopsia	0.0
Double vision	9.84
Loss of acuity	22.95
Hearing symptoms	
Ear surgery	6.56
Deafness	11.48
Hearing loss	50.82
Tinnitus	40.98
Whistling	9.84
Humming	3.28
Accompanied diseases	
Paralysis	8.20
Seizures	3.28
Depression	3.28
Hypertension	6.56
Hypotension	21.31
Cardiac insufficiency	3.28
Diabetes mellitus	3.28
Kidney disorders	6.56
Types of accidents	
Traffic	40.98
Work-related	31.15
Sports	9.84
Home-related	16.39
Accompanying fractures	
Skull fracture, occipital	4.92
Skull fracture, frontal	9.84
Rhinobasal fracture	13.11
Otobasal fracture	100.00

Table 1. Biographical Data of 61 Patients with Neurootological Complaints After Otobasal Fractures

Gender		Age (yr)		Height (cm)		Weight (kg)		Systolic BP (mm Hg)		Diastolic BP (mm Hg)	
M	F	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD
50 (81.97%)	11 (18.03%)	33.8	15.79	172.01	7.39	72.29	11.95	130	28.28	90	0.1

BP = blood pressure; SD = standard deviation.

Table 3. Statistical Evaluation of the Vestibuloocular Caloric Test in 61 Patients After Otobasal Fractures

Parameter	Mean	SD
Spontaneous nystagmus, supine		
Right-beating, frequency	0.60	0.42
Left-beating, frequency	0.37	0.37
Right-beating, amplitude	50.20	24.61
Left-beating, amplitude	30.00	35.27
ECG	77.88	12.98
Caloric nystagmus, 44°C right		
Frequency	1.25	0.75
Culmination	63.69	16.68
ECG	76.77	12.95
Caloric nystagmus, 30°C right		
Frequency	1.51	0.69
Culmination	74.60	63.55
ECG	78.64	12.93
Caloric nystagmus, 44°C left		
Frequency	1.35	0.61
Culmination	64.32	19.19
ECG	76.5	11.88
Caloric nystagmus, 30°C left		
Frequency	1.31	0.71
Culmination	66.76	18.40
ECG	78.12	12.96

Note: Recordings obtained with electrocardiography (ECG) and electronystagmography.
ECG = electrocardiography; SD = standard deviation.

tive statistics of age, height, weight, and the like for all persons entered into this study (Table 1).

As regards the history of patients suffering from otobasal fracture complaints, parameter frequencies as seen in Table 2 have been found (via NODEC). The results of the polygraphic ENG together with the three-channel ECG represented the experimental investigations using the caloric test (Table 3).

To obtain a synoptic overview of the most frequent

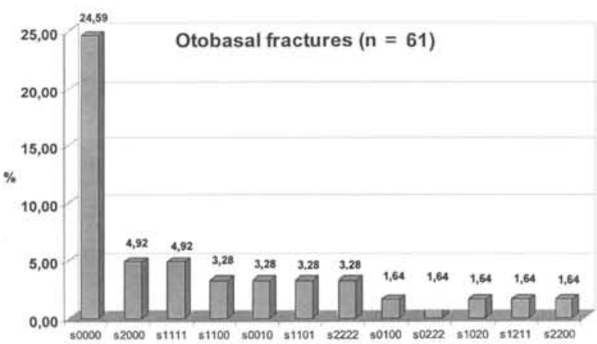

Figure 1. Caloric butterfly test ranked and sorted according to the occurrences of the 12 most frequent patterns (n = 61), expressed by trinary coding (0 = normal; 1= inhibited; 2 = disinhibited) with four digits (position I = right warm; position II = right cold; position III = left warm; position IV = left cold).

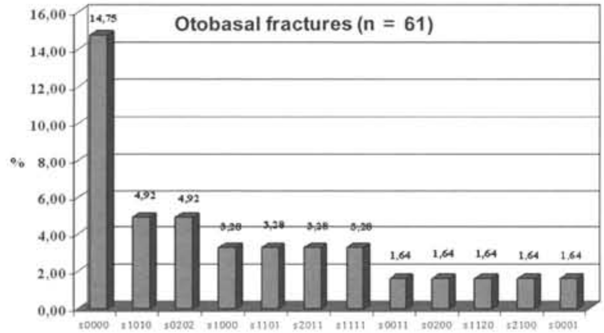

Figure 2. Dynamic vestibular stimulus response intensity comparison (VESRIC) ranked and sorted according to the occurrences of the 12 most frequent patterns (n = 61). Combining the caloric warm response with the perrotatory part of the rotatory intensity-damping test establishes the VESRIC, which can be expressed by trinary coding (0 = normal; 1= inhibited; 2 = disinhibited) with four digits (position I = right warm; position II = perrotatory right; position III = left warm; position IV = perrotatory left). The test results can be categorized into three groups within each of which are three subdivisions. The first group contains the parallel behavior; the second group contains the so-called recruiting phenomena; and within the third group are the so-called decruitment phenomena of VESRIC.

combination of caloric responses, all the butterfly patterns were transcribed by trinary code and thereafter statistically evaluated (Fig. 1). The results of the VESRIC are shown in Figure 2.

Table 4 contains the statistical evaluation of the binaural vestibuloocular per- and postrotatory test results (rotatory intensity-damping test [RIDT]) with simultaneous ENG and ECG recording. The results are charted using the mean and standard deviation for 61 patients with otobasal fracture. CCG served for objectively and quantitatively measuring the responses of the vestibulospinal standing test and the stepping test (Table 5). The results of audiometric evaluation by speech audiometry are represented in Table 6.

DISCUSSION

Some 15% of temporal bone fractures are produced by blows to the occiput. The fracture line begins in the posterior fossa, at or near the foramen magnum, and crosses the petrous ridge through the internal auditory canal or the otic capsule. It is thus called a *transverse fracture*. In transverse fractures of the temporal bone, due to automobile accidents or other causes of head injury, the labyrinth is involved more frequently than in longitudinal fractures. Severe vertigo with severe or total hearing loss is not uncommon with such injuries. In milder injuries, labyrinthine "concussion" may occur, with transitory auditory-vestibular symptoms. The force

Table 4. Statistical Evaluation of the Binaural Vestibuloocular Perrotatory and Postrotatory Test (RIDT) with Simultaneous ENG and ECG Recording in 61 Patients Suffering from Otobasal Fractures

RIDT	Mean	SD
Perrotatory nystagmus, right		
Frequency	46.21	24.29
Culmination	23.65	6.86
ECG	74.22	10.93
Perrotatory nystagmus, left		
Frequency	42.78	18.65
Culmination	24.03	6.02
ECG	74.44	10.21
Postrotatory nystagmus, right		
5-second	10.80	5.71
30-second	50.32	28.02
ECG	72.23	21.79
Postrotatory nystagmus, left		
5-second	11.00	4.98
30-second	53.02	22.87
ECG	78.46	11.14
Spontaneous nystagmus, sitting, eyes closed		
Right frequency	0.37	0.35
Left frequency	0.49	0.31
ECG right	76.55	10.23
Spontaneous nystagmus, sitting, eyes open		
Right frequency	0.33	0.27
Left frequency	0.31	0.23
ECG left	74.11	11.11

ECG = electrocardiogram; ENG = electronystagmography; RIDT = rotatory intensity-damping test; SD = standard deviation.

that causes the fracture can be so strong that it not only results in the fracture of the base of the skull but also may cause a lesion of the brainstem, resulting then in a combined peripheral and central lesion. According to Alexander and Scholl, 15% of the patients having suffered this kind of trauma demonstrate signs of cerebral injury, and 33% have a hearing loss.

The *longitudinal fracture* is a linear break through the floor of the middle cranial fossa passing parallel and adjacent to the anterior margin of the petrous pyramid. Approximately 80% of temporal bone fractures usually are of this type. Facial weakness or paralysis occurs in fewer than 25% of such cases. Characteristically, the fracture line lacerates the tympanic membrane. Therefore, bleeding from the external auditory canal can be experienced. Cerebrospinal otorrhea is common and usually subsides in a few days as the fracture heals. Sensorineural hearing loss can occur, with the characteristics of inner-ear concussion.

Table 5. Statistical Evaluation of the Main Parameters of Stepping and Standing Craniocorpography (CCG) in 61 Otobasal Fracture Patients

Parameter	Mean	SD
CCG, stepping		
Lateral sway (cm)	12.28	4.94
Longitudinal displacement (cm)	74.91	39.27
Angular deviation (degrees of angle)	−3.65	72.88
Body spin (degrees of angle)	−1.44	91.99
CCG, standing		
Lateral sway (cm)	3.72	1.61
Longitudinal sway (cm)	8.81	2.96

SD = standard deviation.

Table 6. Statistical Evaluation of Speech Audiometry in 61 Otobasal Fracture Patients

Parameter	Mean	SD
Speech audiometry, right		
Numbers	25.60	28.01
Words	71.41	21.15
Loss of discrimination	13.28	30.05
Speech audiometry, left		
Numbers	23.30	22.15
Words	71.10	20.10
Loss of discrimination	7.68	24.39

SD = standard deviation.

In a transverse fracture, the break line is perpendicular to the long axis of the petrous pyramid. In nearly 50% of cases, the facial nerve is lacerated, and the resultant palsy may be permanent and require surgical solution. Bleeding of the ear seemingly is uncommon, although hematotympanum is a frequent finding. Cerebrospinal fluid may continue to fill the middle ear even after the blood is absorbed and is drained through the Eustachian canal. The fracture passes through the vestibule of the inner ear, causing extensive destruction of the membranous labyrinth and usually complete loss of cochlear and vestibular function. This then causes severe vertigo lasting for several days, with accompanying nausea and vomiting. It subsides gradually in a period of perhaps 2–3 weeks. Unsteadiness and a tendency to sway to the side of the involved ear may persist for months. A mild spontaneous nystagmus with the quick component to the opposite side may persist for many months or years or even may persist indefinitely. The tendency toward compensation is shown in follow-up CCGs and especially in VESRIC.

For this study, we evaluated the neurootological histories of and important neurootological findings in 61 patients who suffered from otobasal skull fractures. Table 1 reflects the typical conditions for an accident group: First, many more men (50 [81.97%]) than women (11 [18.03%]) are involved. Second, as can be observed also in other accident patient groups, is the low mean age of 33.8 years. These accidents occurred 40.98% of the time in traffic and 31.15% of the time at work. The smaller group of other accidents included sports accidents (9.84%) and home accidents (16.39%) according to the NODEC.

All the patients demonstrated otobasal fractures. Additionally, in 8 patients (13.11%), head trauma was so severe that rhinobasal fractures also were seen (according to the NODEC). Furthermore, frontal skull fractures occurred additionally in 6 patients (9.84%) and occipital skull fractures in 3 (4.92%.) Thus, in 27.87% of patients (17), otobasal fractures were among multiple cranial fractures incurred. The most prominent vertigo complaints were the feeling of instability and the feeling of a rocking vertigo (both symptoms in 24 patients [39.34%]), followed by a rotating vertigo (in 20 patients [32.79%]) and blackouts (in 16 patients [26.23%]). The vegetative and nausea symptoms showed a high incidence of malaise (16 patients [26.23%]) but a lower incidence of vomitus (5 patients [8.20%]).

With respect to the duration of the single vertigo attacks, 32 patients (52.46%) reported that the dizzy spells lasted for only seconds. All in all, this is a short-lasting vertigo, with instability and rocking sensations and a certain amount of malaise. With respect to visual disturbances, we found much double vision (6 patients [9.84%]) but a low proportion of oscillopsias. Loss of visual acuity occurred in only 22.95% (14 patients).

The number of subjective hearing symptoms was much higher than the amount of visual symptoms. This can easily be understood, as this patient sample was selected for a history of otobasal fractures. Subjective hearing loss occurred in 31 patients (50.82%), and an additional 7 patients (11.48%) experienced deafness. Twenty-five patients (40.98%) suffered from posttraumatic tinnitus, and 4 (6.56%) underwent ear surgery for correction of traumatic changes in the temporal bone. Among the other accompanying symptoms and diseases, we found two major groups: Thirteen patients (21.31%) suffered from hypotension, whereas only 4 (6.56%) had hypertension. Five patients (8.2%) complained of palsy.

In analyzing the objective and quantitative results of neurootometric testing, we found that statistical evaluation of the ENG results of caloric nystagmus analysis showed a high value for spontaneous nystagmus, especially for right-beating nystagmus. However, the caloric nystagmus mean frequencies were within the normal range, although they showed a tendency toward a disinhibition as regards standard deviation (see Table 3).

At the same time that the ENG was recorded during the caloric vestibuloocular test, the ECG was recorded and evaluated for heart rates during the response culmination phase. We found negative vestibular cardiac downregulation of the heart rate, especially the downregulation lost with respect to the means in the two caloric cold responses. Tabulating all the caloric butterfly patterns after transferring them from the graphic design into a numerical design by trinary coding, we observed (see Fig. 1) that only 15 patients (24.59%) showed a completely normal caloric response. In ranking the next twelve most frequently found caloric butterfly patterns, we saw a mixture of central, peripheral, and combined vestibuloocular dysfunctions. The per- and postrotatory nystagmus of the RIDT (see Table 4) demonstrated an elevated level of the means, although these remained within the normal range. The standard deviations, however, exhibit a broad spread, especially for perrotatory and postrotatory right-beating nystagmus.

Despite the failure in vestibular cardiac rate downregulation under the vestibuloocular stimulus (see Table 3), this vestibular vegetative reaction is beneficial for the ECG heart rate, counted during the perrotatory right- and left-beating reaction and the postrotatory right-beating reaction but not during the postrotatory left nystagmus (see Table 3). Concerning the postrotatory vestibular cardiac reaction, usually we see (for both postrotatory responses) upregulation as compared to the pretest situation in the spontaneous phase.

Putting the caloric warm responses and the perrotatory nystagmus responses together into the VESRIC, we found (see Fig. 2) that only 14.75% of all the responses exhibited a normal pattern. In other words, nearly 52 patients (85%) showed vestibuloocular pathology as a typical sign of the 60.80% of all patients suffering from an otobasal fracture. The first three pathological patterns—s1010, s0202, and s1000—of recruitment behaviors showed a certain lability in the regulation, although in two of them, there was a tendency for recovery. One pattern typically exhibits a labile central disinhibition. Parallel peripheral inhibitions are followed in patterns s1101 and s2011. With respect to the vertigo history, more pathology was found in the equilibriometric tests based on nystagmus, as expected.

On analysis of the vestibulospinal pathways (e.g., by the stepping CCG; see Table 4), the two indicator symptoms for deviations—angular deviation and body spin—showed highly pathological results. Lateral sway, which usually is greatly increased in cerebello-pontomedullary dysfunctions, remained in the normal range, even for the sum of the mean plus standard deviation. The two important parameters of longitudinal and lateral sway of the standing CCG remained fairly normal. In summarizing the stepping CCGs (Fig. 3), we found all in all that only 27.87% (17) were normal, meaning that some 72% (44) of all the stepping CCGs results were pathological. The results of the trinary coded stepping CCGs gave us a mixed picture of typical peripheral lesions, as in patterns s0010, s0011, and s0020. Purely central pathology was exhibited in patterns s2200 and s0200.

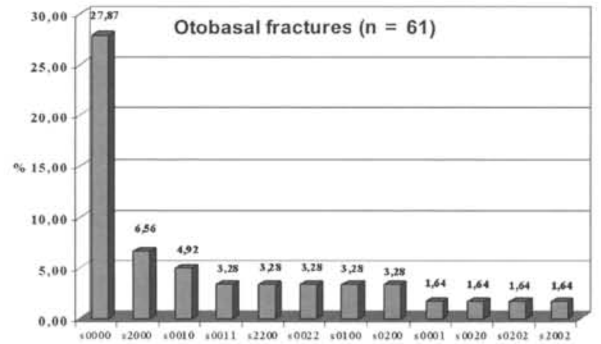

Figure 3. Stepping craniocorpography (CCG) patterns ranked and sorted according to the occurrences of the 12 most frequent patterns, which are expressed by trinary coding (0 = normal; 1 = inhibited; 2 = disinhibited) with four digits (position I = displacement; position II = lateral sway; position III = angular deviation; position IV = body spin).

CONCLUSION

As regards otobasal fractures as a severe trauma to the inner-ear system and the head, patient medical history is typical, showing vertigo, nausea, and hearing symptoms. In the group of patients having visual symptoms, double vision was especially prominent. However, the statistical significance of these symptoms is higher in other disease groups that we have investigated particularly. However, the incidence of dysfunction in the vestibuloocular pathways was extremely high. Only 9 patients (14.75%) showed no pathological changes on the vestibuloocular tests by calorization and perrotatory stimuli. The incidence of pathologically changed stepping CCGs was very high, into the range of 72%.

Patients' hearing capacity as tested by speech audiometry also showed pathology, which corresponds to the high incidence of hearing symptoms, including hearing loss, deafness, and tinnitus. Otobasal fractures lead to subjective statoacoustic complaints, which are reflected also in the results of objective and quantitative measurements of the vestibuloocular, vestibulospinal, and hearing pathways.

BIBLIOGRAPHY

Barány R. Untersuchungen über den vom Vestibularapparat des Ohres reflektorisch ausgelösten rhythmischen Nystagmus und seine Begleiterscheinung. *Mschr Ohrenheilk* 40:193–297, 1906.

Barány R, Wittmaack K. *Funktionelle Prüfung des Vestibularapparates*. Jena: Fischer-Verlag, 1911.

Black FO, Lilly DJ, Nashner LM, et al. Quantitative diagnostic test for perilymph fistulas. *Otolaryngol Head Neck Surg* 96:125–134, 1987.

Bodo G, Heid L. The Different Types of Perilymph Fistula. In C-F Claussen, MV Kirtane (eds), *Vertigo, Nausea, Tinnitus and Hypoacusia Due to Head and Neck Trauma*. Amsterdam: Elsevier Science, 1991:159–160.

Brunner H, Schönbauer. Commotio Auris Internae (Innenohrerschütterung). In G Alexander, O Marburg (eds), *Handbuch der Neurologie des Ohres*. Wien: Urban & Schwarzenberg, 1928.

Claussen C-F. Das belastungsabhängige Dysaequilibrium. *Proc NES* 6:15–74, 1979.

Claussen C-F. *Der schwindelkranke Patient—Grundlagen der Neurootologie und Äquilibriometrie*. Hamburg: Werner Rudat, 1992.

Claussen C-F. *Presbyvertigo, Presbyataxie, Presbytinnitus*. Berlin: Springer-Verlag, 1985.

Claussen C-F. *Schwindel—Symptomatik, Diagnostik, Therapie*. Hamburg: Werner Rudat, 1981.

Claussen C-F. Über eine Gleichgewichtsfunktionsprüfung mit Hilfe der Cranio-Corpo-Graphie (CCG) und Polarkoordinaten im Raume. *Arch Klin Exp Ohr Nas Kehlk Heilk* 196:256–261, 1970.

Claussen C-F, Aust G, Schäfer WD, von Schlachta I. *Atlas der Elektronystagmographie*. Hamburg: Werner Rudat, 1986.

Claussen C-F, Bergmann de Bertora JM, Bertora GO. *Otoneurooftalmologia*. Berlin: Springer-Verlag, 1988:1–124.

Claussen C-F, Claussen E. Forschungsbericht Cranio-Corpo-Graphie (CCG)—Ein einfacher objektiver und quantitativer Gleichgewichtstest für die Praxis. Schriftenreihe des Hauptverbandes der gewerblichen Berüfsgenossenschaften eV, D 5205 St. Augustin 1986.

Claussen C-F, De Sa JV. *Clinical Study of Human Equilibrium by Electronystagmography and Allied Tests*. Bombay: Popular Prakashan, 1978.

Claussen C-F, Tato JM. *Equilibriometria Practica*. Buenos Aires: Hasenclever, 1973.

Claussen C-F, von Schlachta I. Butterfly chart for caloric nystagmus. *Arch Otolaryngol* 96:371–375, 1972.

Czigner J, Szabados E. Does Delayed Labyrinthine Hydrops Develop in Consequence of Head Trauma? In C-F Claussen, MV Kirtane (eds), *Vertigo, Nausea, Tinnitus and Hypoacusia Due to Head and Neck Trauma*. Amsterdam: Elsevier Science, 1991:161–165.

English GM. *Otolaryngology: A Text Book*. New York: Harper & Row, 1976.

Fitzgerald G, Hallpike CS. *Studies in Human Vestibular Function: Observation on the Directional Preponderance of Caloric Nystagmus Resulting from Cerebral Lesions*. 1942. Fee G.A. Traumatic perilymphatic fistulas. *Arch Otolaryngol* 88:477, 1968.

Fort E, Fort J. Hallazgos otoneurologicos en fracturas de base de cranneo. *Proc NES* 5:93– 106, 1976.

Goodhill V. *Ear Diseases, Deafness and Dizziness*. Hagerstown, MD: Harper & Row, 1979.

Hallpike CS. *The Caloric Tests*. 1956.

Hemenway WG, Black FO, Grimm R, Pesznecker S. Traumatic Perilymph Fistula. In C-F Claussen, MV Kirtane (eds), *Vertigo, Nausea, Tinnitus and Hypoacusia Due to*

Head and Neck Trauma. Amsterdam: Elsevier Science, 1991:167–170.

Henriksson NG. Speed of slow component and duration of caloric nystagmus. *Acta Otolaryngol Suppl (Stockh)* 125:1956.

Henriksson NG, Jahneke JB, Claussen C-F. *Vestibular Disease and Electronystagmography*. Lund, Sweden: Studentlitteratur, 1969.

Kessler L. Functional complications after the otobasal fracture. *Excerpta Medica* 929:113–117, 1991.

Megighian D, Barion U, Tosatto A. Die Kompensationsmechanismen nach einseitigen akuen Labyrinthstörungen in Abhängigkeit vom Lebensalter. *Proc NES* 12(13):75–77, 1988.

Mizukoshi K, Watanabe Y, Shojaku H, Aso S. Vertigo after head and inner ear injuries—with special reference to neurotological findings. *Excerpta Medica* 1087:91–94, 1995.

Mungai A. *Kopf-Hals Trauma und objektive aequilibriometrische Befunde* [inaugural dissertation]. Würzburg: Würzburg Universität, 1984.

Pialoux P. Les fractures du rocher et leur traitement. *Proc NES* 8:285–290, 1981.

Romberg H. *Lehrbuch der Nervenkrankheiten*. Berlin: Springer-Verlag, 1848:184–191.

Ruttin E. *Klinik der serösen und eitrigen Labyrinthentzündungen*. Wien: Josef Safar, 1922.

Schuknecht HF. A clinical study of auditory damage following blows to the head. *Ann Otol* 59:331, 1950.

Schuknecht HF. *Pathology of the Ear*. Cambridge, MA: Harvard University Press, 1974.

Tato JM, Tato JM. Terapia de las afecctiones vestibulares perifericas. *Proc NES* 4:181–189, 1975.

Unterberger S. Neue objektive registrierbare Vestibulariskörperdrehreaktionen, erhalten durch Treten auf der Stelle. Der Tretversuch. *Arch Ohr Nas Kehlk Heilk* 145:273–282, 1938.

Wittmaack K. Über die traumatische Labyrinthdegeneration. *Arch Ohr Nas Kehlk Heilk* 131:59–124, 1932.