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Treatment of the Syndrome of the Slowed Down Brainstem with Vertigoheel

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Summary

The syndrome of the slowed down brainstem embraces conceptually a group of patients who suffer subjectively from vertigo with the particular symptoms of staggering, noise in the ears, hearing defects and a general depressive state with decrease in mental powers. Objectively there is in these patients conspicuous prolongation both of cumulative latency of the vestibular nystagmus reaction and latency prolongation of the acoustically evoked brainstem potentials. These findings represent an indication for degenerative symptoms in the stimulus conduction of the relevant system stimuli in the brainstem. We assume degenerative features depending upon the metabolism in the neuronal network of the brainstem to be the cause of the syndrome of the slowed down brainstem. These changes are of a general nature, since they concern both the vestibular and the acoustic system. The term of the syndrome of the slowed down brainstem resulted from practical clinical observations. The popular expression "slow on the uptake" corresponds to it in patients with pathway degeneration, for instance.

The homeopathic remedy **Vertigoheel** contains in its administered form per tablet cocculus D4, 210mg; conium D3, 30mg, ambergris D6, 30mg; petroleum D8, 30mg. **Vertigoheel** is well tolerated. Its time and again providing of clear subjective relief in vertigo and nausea and in part also in tinnitus corresponds in systematic neurotological investigations to the improvement in objective findings of the equilibrium functional system and also in the auditory path.

Extensive neurotometric functional measurements have been developed to analyze the effect. These include neurotological anamnesis NODEC, examining the vestibulo-spinal function with the aid of the craniocorpography of the stepping and standing test, examining the vestibulo-ocular system with the aid of polygraphic electronystagmography, topodiagnostic neurotological characteristic diagnostics and acoustically evoked brainstem potentials (AEBP). The results of the neurotometric functional tests named are evaluated according to the following 3 categories:

1. Assessment by inspection of typical graphical elements of the recorder curves.
2. Measurement and calculation of representative reaction intensities, such as of the central nystagmus rate or the amplitudes of the evoked brainstem potentials.
3. Measurement of temporal reaction patterns, such as the vestibular cumulative latency and the latency time pattern of the acoustically evoked brainstem potentials (AEBP).

This method of observation enables different pathomechanisms to be differentiated and verified in the functional area.

We observe quantitatively calculated nystagmus inhibition and disinhibition conditions usually after cross-connective lesions, in which one or the other neurotransmitter exercises a too strong inhibiting or disinhibiting influence on the cerebral pathways. In the case of the syndrome of the

slowed down brainstem, we must assume that the entire activity in the neuronal network is restricted.

In daily neurotological practice, we have used **Vertigoheel**, the combination preparation from ambergris, cocculus, conium and petroleum, in 40.2% of 1031 firstly examined vertigo patients. 33.7% received **Vertigoheel** tablets and 6.5% **Vertigoheel** liquid. 163 patients from this group come into the special group of the syndrome of the slowed down brainstem. 52.3% of them were treated with **Vertigoheel**, 45.4% with tablets and 6.9% with liquid.

Out of the 377 patients treated with **Vertigoheel** tablets, 207 patients = 54.91% became free of complaints in the course of therapy and also by combination with other drugs.

In a selective therapy study with **Vertigoheel** on 40 patients with neurotological examination before and after 14 day therapy with 3 **Vertigoheel** tablets 3 times daily, 57.5% reported an improvement in symptoms. Synoptic calorific butterfly characteristic evaluation of the monaural vestibulo-ocular reaction showed an improvement by 32.5%. In the case of the vestibulo-spinal changes associated with disturbances to the gait and head-body deviations, an improvement by 22.5% was observed. A clear transit time improvement could also be achieved in the evaluation of the acoustically evoked brainstem potentials in the entire ponto-medullar course from the acoustic nuclei up to the lower quadrigeminal region.

In a second study directed towards the special question of the action of the components petroleum, ambergris, cocculus and conium, 4 groups of 15 persons each were formed from 60 vertigo, nausea and tinnitus patients. These were treated for 14 days separately either with petroleum D6, ambergris D4, cocculus D2 or conium D1. Neurotological examinations before and after treatment include questions regarding anamnesis, measurement of the vestibulo-ocular nystagmus function, measurement of the vestibulo-spinal head-body staggering function with the aid of craniocorpography, examining the retino-ocular nystagmus regulation with the aid of optokinetics and examinations on the auditory path in the brainstem region with the aid of acoustically evoked brainstem potentials (AEBP). In the analysis of the action of the components of **Vertigoheel**, an exclusively positive or exclusively negative effect could be ascertained in no single substance. It was observed comparatively that the strongest positive effect is produced by cocculus. This is followed by conium followed by petroleum. Ambergris produces the weakest effect. Negative effects are observed most frequently in the case of petroleum and to a lesser degree in ambergris, cocculus and conium. When comparing the study of the total remedy with the study of its components, it must be assumed that the components in **Vertigoheel** develop additional healing success by synergistic effects when they are combined to form a single remedy.

The fact that the most active component, cocculus, acts in opposition to the inhibitory neurotransmitter of the brainstem gamma aminobutyric acid appears to be important for treatment of the syndrome of the slowed down brainstem, which is characterized by objective slowing down of the reactions of brainstem regulating processes. Among other things, the activity promoting effect with stimulus conduction acceleration of **Vertigoheel** on the syndrome of the slowed down brainstem can be explained by this.

I. Introduction

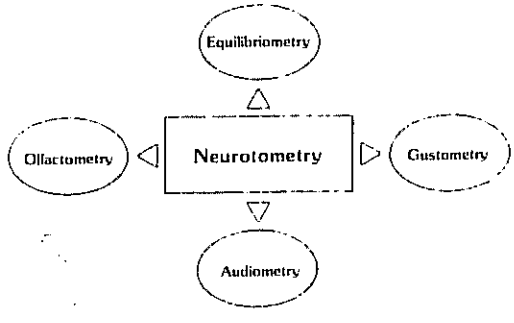
Neurotology has developed at the limits of ENT medicine, neurology and ophthalmology. The classical medical examination methods are the technique of anamnesis, in which symptoms are verbally described and inspection, in which symptoms are converted into pictures. Functional analysis has been added in this century as the third side of medical diagnostics. It requires observation of changes in parameters with time and its representation with the aid of functional graphs and characteristics. Life is, among other things, a function of time. Functional analytical medical diagnostic methods are today an important component in the repertoire of practically every physician. These include electrocardiography, electroencephalography, electromyography, electronystagmography etc.

Neurotology concerns itself with the examination and treatment of the senses of the head. It concentrates on the regions of hearing, equilibrium, smelling and tasting. It is extremely analytically oriented. Metrical examination methods in this respect are summarized under the term "neurotometry" (fig. 1).

The sensory functions of hearing and equilibrium are depicted predominantly electrophysiologically by time curves. Evaluation of these time curves is grouped basically into 3 areas. On one hand, the graphical elements of the curves as such are linked with clinical meaning. On the other hand, reaction intensity is measured metrically. Frequency distributions of signals and signal amplitudes are available to a particular extent. Finally we recognize thirdly that the initiated reactions proceed at different speeds in the sense organs, the nerves and in the brain. We determine the temporal changes in the regulating process with the aid of different latency times.

With the aid of three part neurophysiological reaction analysis, we observe as a sign of disease not only complete

Fig. 1: *Diagram of neurotometry*



functional paralyses of the equilibrium or of the hearing, or quantitative reaction inhibitions or disinhibitions with excessive nystagmus beats, reduced nystagmus beats, reduced nystagmus or hearing potential amplitudes, but also the delayed reaction processes in reactions which are otherwise quantitatively still in the standard or in the vicinity of the standard.

From the abundance of data on surveying a neurotological symptom data bank, summarizing the symptoms under the term of syndrome of the slowed down brainstem suggests itself. The patients suffer subjectively from vertigo, staggering, tinnitus, hearing defects and a general depressive condition with decrease in mental powers. It is observed objectively that the calorific vestibular test standardized over a long period leads to delayed reactions with prolonged cumulative latency. A similar picture is seen on evaluating the perrotatory vestibular test. There as well there is prolonged perrotatory cumulative latency. If now for comparison the analysis of the audioencephalograms of the acoustically evoked brainstem potentials is referred to, then a delayed reaction process is also observed there.

II. Neurotometric Functional Measurements

Equilibrium functional analysis in patients with vertigo is based essentially on anamnesis and equilibriometry. Both the analysis of nystagmus and the analysis of the head-body movements (fig. 2) form part of equilibriometry. A more or less accurately dosable stimulus is applied vestibularly. This is the sensory part of the measuring system. There then follows a motor reaction of the eye as so-called vestibulo-ocular nystagmus, or else as resulting head-body adjustment reactions. This corresponds to the vestibulo-spinal part. In addition there are today numerous other sensomotor stimulus-reactions chains, such as retino-ocular nystagmus.

The neuroanatomical diagram of the auditory path (fig. 3) is of significance for the neurotological examination with computer assisted evaluation. By comparing the functional behaviour of vestibulo-ocular, vestibulo-spinal and retino-ocular regulation loops with the reactions of the auditory path, it is possible to develop a network of functional topological neurotological analyses which enable disturbances to be localized and treated selectively.

1. The neurotological anamnesis NODEC

Anamnesis is indispensable for medical diagnosis of every type. Differentiated neurotological functional diagnostics must also be concerned firstly with the subjective complaints stated by the patient. We use the clinical anamnesis system NODEC (fig. 4) for recording neurotological symptoms. This covers a single page questionnaire. The anamnesis questionnaire contains questions regarding the type of vertigo, accompanying vegetative or nausea reactions and the mechanisms triggering vertigo. Further time data of the patient concerning the duration of the complaint in total and of the individual attacks of vertigo are asked for. In this case a geometrical time system from seconds through minutes to hours, days, weeks etc. is used. Further, it is asked whether the vertigo occurs uniformly over a lengthy period or whether it rises and falls.

Moreover, the questionnaire contains numerous questions regarding symptoms of the other cranial nerves, such as smelling, seeing, hearing, facial, trigeminus and tasting disturbances. Hearing defects are subdivided according to hearing loss and subjective noises in the ear. Further, a closely defined range of important basic symptoms is asked about.

Fig. 2: *Input and output diagram of equilibriometry with special reference to the vestibulo-ocular and to the vestibulo-spinal reflex arc.*

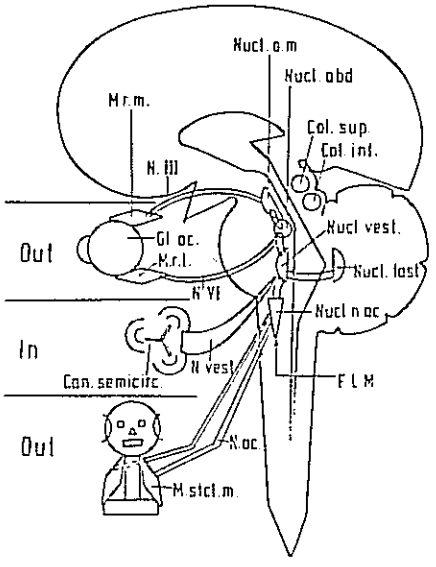
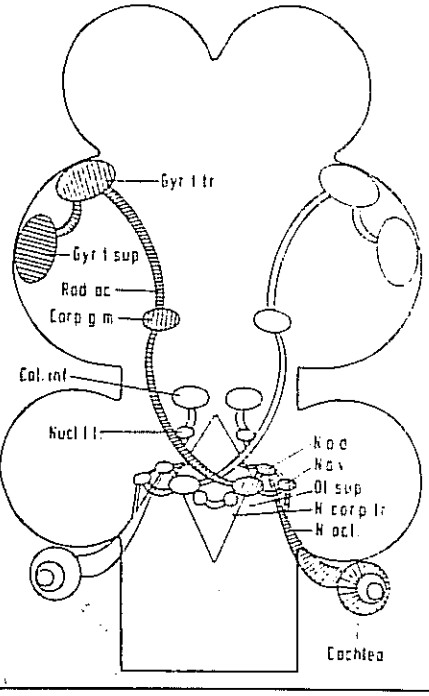


Fig. 3: *Diagram of the human auditory path. A plain section of the human brain from the vertex to the foramen magnum is superimposed on the diagram.*



Such basic complaints are primarily the cardio circulatory complaints, metabolic illnesses such as diabetes mellitus, kidney disease, further neurological complaints and the condition after head-neck trauma. Finally the preprinted neurotological anamnesis NODEC III also occupies itself with habits regarding stimulants and pharmaceuticals.

For the case of repeat examinations, the preprinted anamnesis has a 5-stage scale of subjective self assessment of the course of the disease. It is possible to add handwritten entries for special features.

The neurotological anamnesis NODEC has proven to be a very sensitive indicator for determining a change in symptoms to the better or to the worse in the course of 17 years use. However, it is unsuitable as a topodiagnostic instrument. Therapy which should also have the subjective health of the patient in view cannot be successful in the field

Fig 4: *Systematically recorded neurotological anamnesis NODEC III*

1) Vertigo symptoms:
   - a) vestibular vertigo ( )
   - b) lifting feeling ( )
   - c) turning feeling ( ) to right ( ) to left ( )
   - d) tendency to fall ( ) to right ( ) to left ( )
   - e) becoming black before the eyes ( )
   - f) uncertainty ( )

2) Vegetative symptoms:
   - a) outbreaks of sweat ( )
   - b) nausea ( )
   - c) retching ( )
   - d) vomiting ( )
   - e) collapse ( )

3) Cause of vertigo:
   - a) Kinetosis — ship, airplane, railway, car ( )
   - b) turning the head ( )
   - c) bending down ( )
   - d) standing up ( )
   - e) changing direction of view ( )

4) Duration of complaints:
   - a) for hours ( )
   - b) for days ( )
   - c) for weeks ( )
   - d) for months ( )
   - e) for years ( )
   - f) for decades ( )

5) Duration of the individual attack:
   - a) 1 - 2 seconds ( )
   - b) minutes ( )
   - c) hours ( )
   - d) days ( )
   - e) weeks ( )
   - f) months ( )
   - g) long lasting uniformly ( )
   - h) rising and declining, long duration ( )

6) Disturbances of sense of smell:
   - a) anosmia on the right ( ) on the left ( )
   - b) hyosmia on the right ( ) on the left ( )
   - c) cacosmia on the right ( ) on the left ( )
   - d) nasal breathing impediment on the right ( ) on the left ( )

7) Disturbances of vision:
   - a) unsharpness ( )
   - b) double images ( )
   - c) impressions of motion ( ) jerkily ( )
   - d) blindness on the right ( ) on the left ( )

8) Ear symptoms:
   - a) tinnitus on the right ( ) on the left ( )
   - b) hearing loss on the right ( ) on the left ( )
   - c) deafness on the right ( ) on the left ( )
   - d) condition after ear surgery on the right ( ) on the left ( )

9) Disturbances of sense of taste
   - a) ageusia ( )
   - b) parageusia ( )
   - c) hypogeusia ( )

10) Signs of trigeminus
   - on the right ( )
   - on the left ( )

11) Facial paresis
   - a) peripheral on the right ( ) on the left ( )
   - b) central on the right ( ) on the left ( )

12) Head-neck trauma
   - a) traffic accident ( )
   - b) accident at work ( )
   - c) sports accident ( )
   - d) domestic accident ( )

13) Neurological diseases ( )

14) Angiocardiopathies:
   - a) hypertension ( )
   - b) hypotension ( )
   - c) arteriosclerosis ( )
   - d) cardiac insufficiency ( )
   - e) condition after cardiac infarction ( )

15) Diabetes mellitus: ( )

16) Kidney diseases: ( )

17) Pharmaceuticals or stimulants
   - a) alcohol ( )
   - b) nicotine ( )
   - c) caffeine ( )
   - d) salicylates ( )
   - e) streptomycin ( )
   - f) gentamycin ( )
   - g) contraceptives ( )
   - h) sedatives ( )
   - i) antivertiginous agents ( )
   - j) other ( )

18) .....examination, diseases:
   - a) unchanged ( )
   - b) slightly improved ( )
   - c) clearly improved ( )
   - d) slightly worsened ( )
   - e) clearly worsened ( )

19) Miscellaneous:

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of neurotology without such an anamnesis. This applies not only for acute organic diseases but also for degenerative conditions of multicausal etiology, such as the syndrome of the slowed down brainstem.

In connection with vertigo, nausea and tinnitus, we observe as most frequent underlying disease a cardio-circulatory complaint on evaluation of the anamnesis. Nearly every second patient suffers from vertigo as an effect of a cardio circulatory disease. Vertigo, nausea and tinnitus are here quasi alarm signs of commencing degeneration in the equilibrium sensory system. Metabolic diseases such as diabetes mellitus and kidney disease are also relevant. Degenerative symptoms involving the cervical spine play only a minor role (Table 1). Even more seldom are the known morphological diseases from the textbooks, such as acoustic nerve neurinoma, ponsglioma, brain abscess, thromboses of a sinus etc. Even meningitis and encephalitis are relatively infrequent events. Nevertheless in diagnosis the last named serious diseases should always be paid adequate attention.

In the neurotological department of the University of Wurzburg, model data banks in which the data from anamneses, functional tests and morphological examinations are collected have been used for many years. The typical distribution pattern for vertigo and nausea symptoms in 10,335 patients of the neurotological data bank NODEC IV, which covers patients of all age classes and both sexes, is shown below in Table 2.

Table 1: *Distribution of selected underlying diseases in patients of the neurotological data bank NODEC III (n = 10,279)*

Disease	NODEC III (n = 10,279)
Hypotension	23.5%
Hypertension	12.4%
Cardiac insufficiency	11.3%
Diabetes mellitus	5.1%
Kidney disease	6.3%
Cervical spine disease	4.9%

Table 2: *Vertigo and nausea symptoms from NODEC IV (n = 10,335)*

Symptoms	NODEC IV (n = 10,335)
vestibular vertigo	39.1%
lift vertigo	5.3%
turning vertigo	35.9%
tendency to fall	19.7%
becoming black in front of the eyes	19.7%
uncertainty	35.2%
outbreaks of sweat	11.9%
nausea	30.1%
retching	3.7%
vomiting	15.1%
collapse	5.8%

Hearing and equilibrium are represented by 6 measuring points in the receptor region of the inner ear. The ear symptoms in neurotological patients are shown in Table 3.

Table 3: *Ear symptoms from NODEC IV (n = 10,335)*

Symptoms	NODEC IV (n = 10,335)
noise in the ears	44.8%
hearing reduction	53.3%
deafness	8.2%
condition after ear surgery	5.7%

Comparison of tables 2 with 3 shows that vertigo and nausea symptoms are strongly mixed with hearing defect symptoms in neurotological patients.

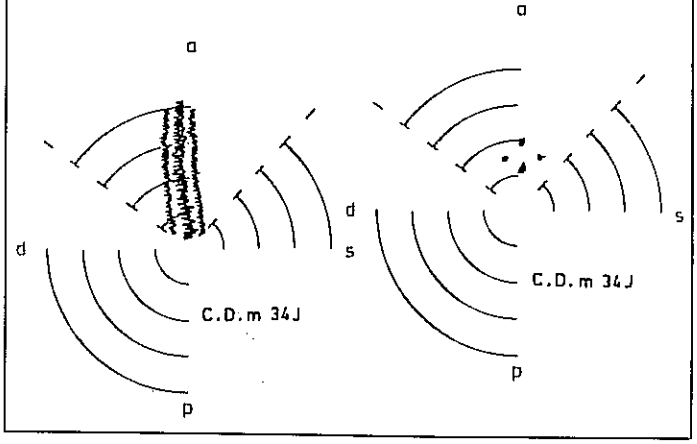
2. Craniocorpography (CCG)

Craniocorpography is a photo-optical recording method for head and shoulder movements in a view from above. The head and shoulders are marked with electric bulbs for this purpose. The light path movements are recorded with an instant picture camera which is equipped with multiple exposure. For the quantitative evaluation of the recorded head and shoulder movement patterns, a polar coordinate phantom is superimposed into the picture at headheight in a second exposure (fig. 5).

The stepping test according to Unterberger and Fukuda with 80 - 100 steps on the spot is performed as a very sensitive vestibulo-spinal test, during which the eyes of the subject are covered with a sleeping mask. This test reacts most sensitively to vestibular disturbances. The evaluation parameters are:

1. The deviation length from the start to the final position.
2. The deviation angle from the starting direction to the final direction, i.e. the angular deviation.

Fig. 5: *Clinical example of a craniocorpogram*
a = anterior, p = posterior, d = dexter, s = sinister



3. The rotation of the body about its own axis, i.e. the body's own spin.
4. The deviation width of the head during each individual step on the spot, i.e. the lateral deviation width.

We perform the standing test according to Romberg in combination with the stepping test. This is very rigid and insensitive for vestibular disturbances. The patient is requested to stand for 3 minutes in an upright position (for younger patients) or 1 minute (for older or atactic patients). The eyes are covered with a sleeping mask once again. The following parameters are assessed for evaluation:

1. The deviation in anterior/posterior direction.
2. The deviation width in lateral direction.
3. Twisting of the head axis in relation to the shoulder axis, the so-called wry-neck angle.
4. Typical head and shoulder light spot configurations.

It should be emphasized in particular under item 4 that there is a typical pattern for Parkinson's disease, in which the head deviations (in anterior/posterior direction elongated) points to a nodding head movement. The a/p deviations of the shoulder light paths are clearly less in contrast.

Clinical investigations and the evaluation of post-mortem findings or inspection findings in neuroradiology or neurosurgery as well as operation findings in otosurgery show that there are typical reaction patterns in craniocorpography, the stepping test and the standing test for peripheral and central vestibular disturbances (fig. 6).

Fig. 6: *Topodiagnostic diagram of the craniocorpography pattern of the stepping test and standing test.*

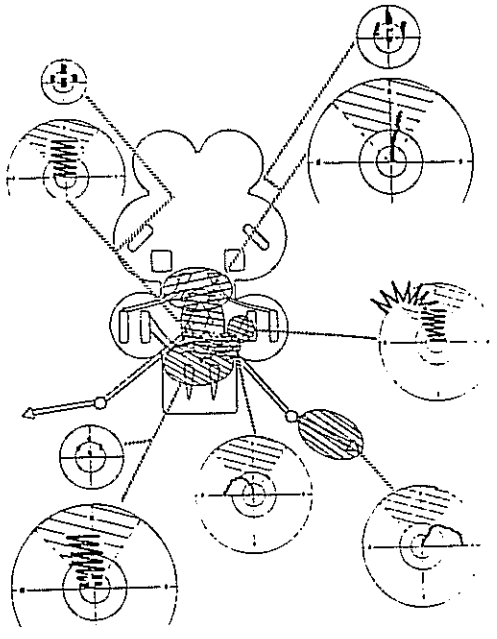
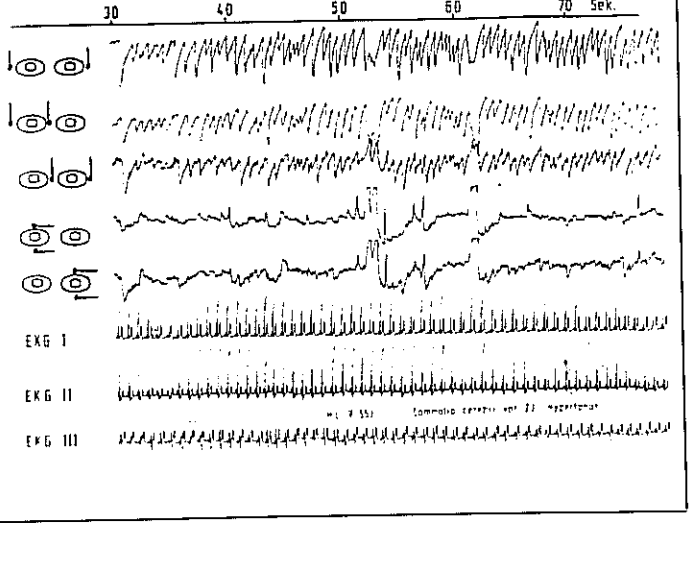


Fig. 7: *Example of a polygraphic 5 track electronystagmogram with simultaneous 3 track ECG for recording the vestibulo-cardiac reactions.*



3. Electronystagmography (ENG)

Whereas craniocorpography is a robust equilibrium test of screening character, electronystagmography is a very subtle examination procedure. In electronystagmography, the background conditions must be kept carefully constant. The recorded potentials are only in the microvolt range, for electronystagmography is based on bioelectrical inherent eye marking. We record with EEG sensitive amplifiers the potential changes across 5 differential electrode pairs generated by eye movements.

Our electrode pattern looks like the following (fig. 7):

1. Derivation of the horizontal eye sum potential by pick-off from one electrode pair in both outer corners of the eye.
2. Pickoff of the right side monocular eye movement pattern from an electrode pair at the right outer corner of the eye and above the root of the nose.
3. Derivation of the left horizontal monocular eye movement pattern from an electrode pair above the root of the nose and the left outer corner of the eye.
4. Pickup of the monocular vertical eye movements of the right eye through an electrode pair on the right cheek and the right forehead.
5. Pickup of the vertical eye movement pattern of the left eye monocularly through an electrode pair on the left cheek and the left forehead.

For the analysis of nystagmus movements, we have recently been using the NYDIAC computer assisted system which acquires the nystagmus parameters simultaneously with the recording on all 5 recording tracks with regard to beat direction, beat number, amplitude, cumulative latency etc., subsequently prints them out in a table separately for both eyes, produces histograms for all tracks for the tempor

al course of the amplitude, the frequency and the speed of the slow phase and in addition outputs synoptic reaction patterns for the clinical work on a data plotter. Any desired segment of the curves can also be output by plotter after digital-analog conversion.

As test patterns we use spontaneous and fixation nystagmus, optokinetic nystagmus predominantly with visual pendulum sequence but also with the linear tracking test of the type of the kite diagram, monaural vestibulo-ocular nystagmus with evaluation through the butterfly diagram, binaural perrotatory and postrotatory nystagmus with evaluation through the L-diagram of the RIDT and complex equilibrium function tests of temporal and spatial stimulus interference of the type of the caloric adaptation cyclogram and of the calorization pendulum interference test. Moreover, the coordination or synchronization of both eyes can be tested simultaneously with the aid of multitrack recording.

In addition, the test results can be related to one another which is used, for example, in the vestibular stimulus reaction strength comparison, which compares the caloric thermal reaction with the ipsidirectional perrotatory reaction in each case. Just as there is physiological spontaneous nystagmus, there are physiological coordination deviations of the movement patterns of both eyes on comparison of the monocular recording tracks. We have therefore had to introduce limit values for the identification of pathological eye movement dissociations.

Simultaneously with the electronystagmogram, we regularly record the electrocardiogram in an electrode layout according to Einthoven (fig. 7). This enables us to record and assess vestibulo-vegetative reactions parallel to vestibulo-ocular reactions.

4. The caloric vestibular test

The vestibulo-ocular system is examined monaurally with the aid of the caloric vestibular test (from the fundamental work by Robert Barany). Physically stimulating the opposite ear as well is avoided by stimulation using a thermal gradient. The lateral semicircular canal must be brought into a vertical position for caloric stimulation. This is done by lifting the patient by 30 degrees in relation to the horizontal on a positioning table.

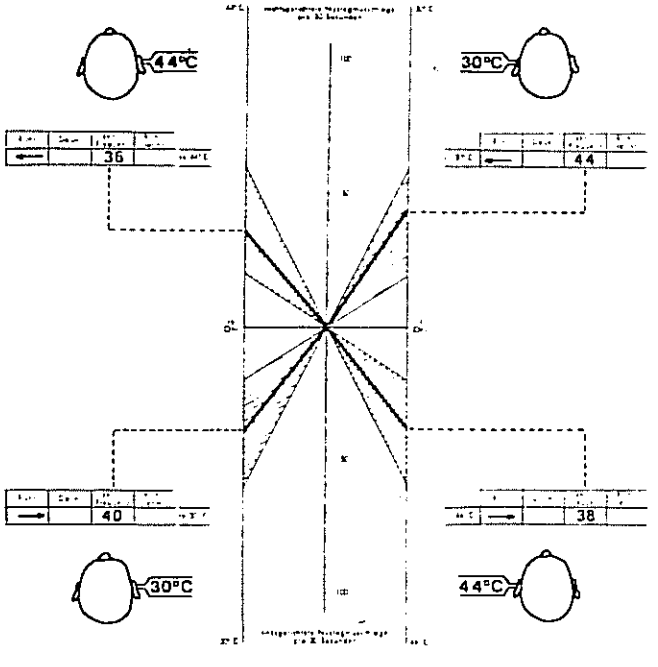
20 ml. water at 30 or 44 degrees C is syringed into the auditory meatus over a period of 30 seconds in each case through an auditory meatus catheter. A plastic bag is attached to the external ear to collect the water running out. This is to prevent the patient receiving additional stimulus by manipulations to the eardrum or by moisture resulting from syringing water running past, which activate muscle potentials which become visible as artifacts in the electronystagmogram.

The produced nystagmus reaction is recorded by means of multi-channel electronystagmography performed simultaneously on both eyes monocularly, horizontally and vertically for 3 minutes (fig. 7). The ECG is recorded simultaneously. Thus it is possible to continuously observe the vestibulo-vegetative reactions. ECG monitoring also provides a criterion for interrupting the test in good time in cases of threatening collapse, increased extrasystoles or too strong bradycardia.

In the evaluation of the caloric electronystagmograms, we proceed from the maximum reaction dynamics to a standard stimulus. The culmination of the caloric nystagmus phenomenon in the ENG curve of the eye examined in each case is sought for. From this, two values result, namely the cumulative latency, i.e. the duration up to occurrence of the strongest reaction, and the maximum nystagmus beat number. The characteristics of the maximum reaction dynamics are summarized in a 4 field diagram, which orders the nystagmus reactions according to stimulated ear and according to nystagmus beat direction. The diagram resulting from this looks like a butterfly. This method is therefore also called butterfly calorigram, according to Claussen (fig. 8).

Fig. 8:

Butterfly Vestibulometry
according to Claussen



Mathematical statistical methods, which code trinarily the individual reactions of the 4 caloric tests in the butterfly diagram by 4 indices have been introduced for the overall statistical evaluation.

Based on these patterns it is possible to establish statistical categories for typical peripheral vestibular disturbances and for typical central lesion patterns. The central reaction patterns can still be differentiated in many ways with regard to the site of origin in the region of the IVth ventricle, of the mesencephalon, of the diencephalon and of the cortex of the temporal lobe. In addition it can be determined whether it is a question of a functional disturbance of the so-called inhibition type (fig. 9) or of the disinhibition type (fig. 10). The results of this examination serve in particular following localization of the disturbance for the initiation and monitoring of differential therapeutic measures.

The cumulative latency is the time which passes from starting syringing up to the centre of the frequency maximum of the caloric nystagmus. It plays a role in the definition of the syndrome of the slowed down brainstem as special functional aspect of the temporal course of reaction. Cumulative latency is determined regularly in every quantitative nystagmus evaluation. If prolonged, it gives information on the central location of a vestibulo-ocular disturbance, the nystagmus beat rate maximum which can quite possibly still be within the standard. The lower and upper standard limits of cumulative latency are shown in table 4 for all 4 caloric vestibular reactions.

Table 4: *Lower and upper standard limits in seconds of the caloric cumulative latency in NODEC III (n=10,001)*

Caloric reaction	Cumulative latency in seconds lower standard limit/upper standard limit	
right 44 degrees C	50.0	87.6
right 30 degrees C	50.1	88.3
left 44 degrees C	50.3	87.7
left 30 degrees C	49.8	88.4

Fig. 9: *Topodiagnostic diagram of peripheral and central vestibular inhibition conditions*

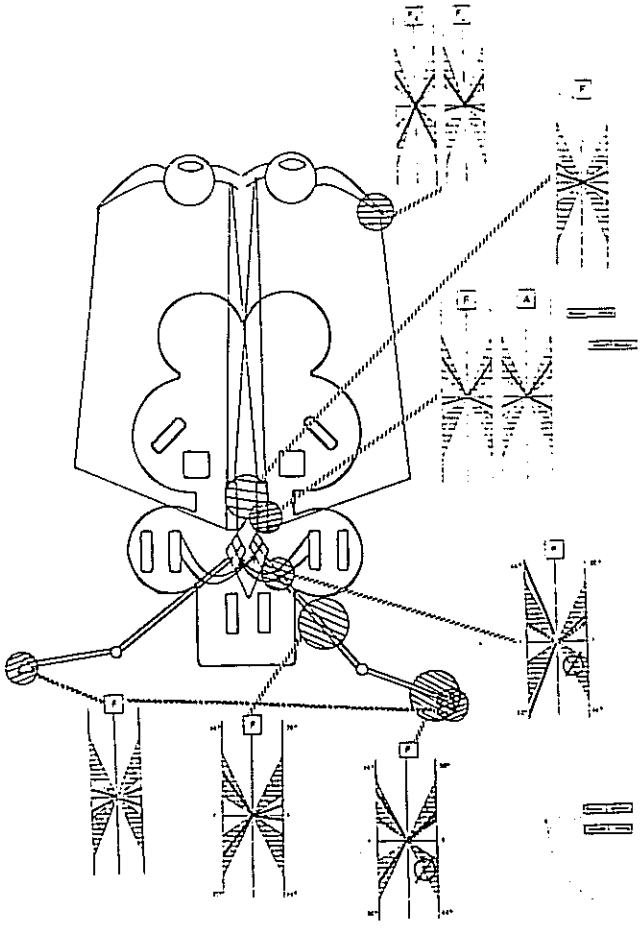
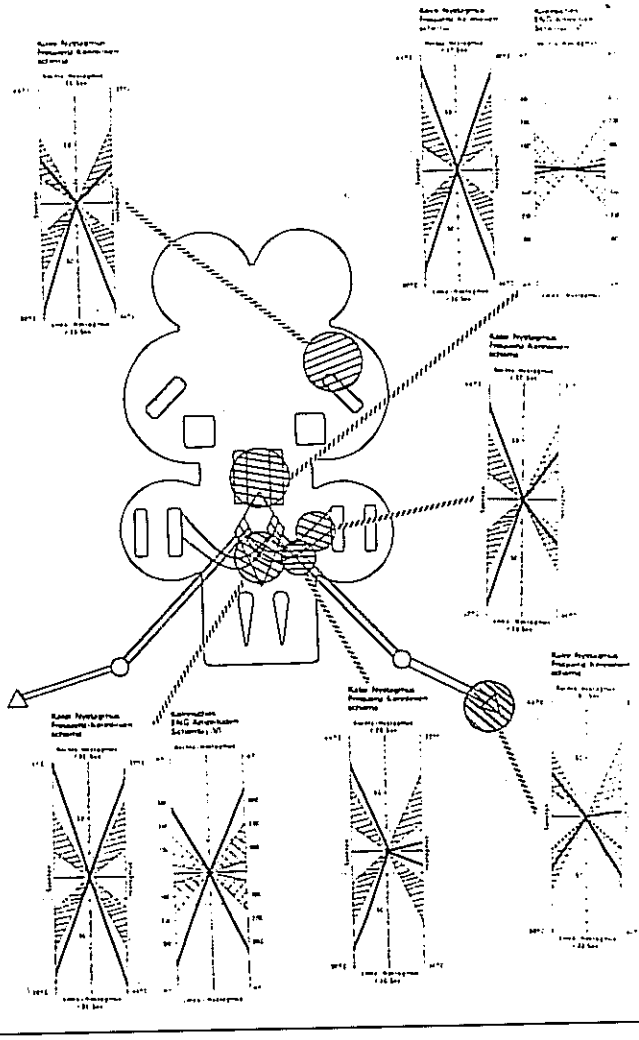


Fig. 10: *Topodiagnostic diagram of peripheral and central vestibular disinhibition conditions*



5. Acoustically evoked brainstem potentials (AEBP)

The auditory path (fig. 3) is examined with the aid of conventional psychophysical sound threshold and speech audiograms and also with the aid of audioencephalograms. Acoustically evoked potentials are termed cortical and subcortical, electrical voltage differences which stand in a temporally definable relationship to external acoustic stimuli and can be made visible as wave form by electronic signal processing. The duration of their latency or the temporal sequence of their appearance after the acoustic stimulus is used for practical classification into early, medium and late acoustically evoked brain potentials. The early or also acoustically evoked brainstem potentials are very reliable with regard to the analysis of their graphical elements. Their closeness to the stimulated receptor also permits good topographical localization (fig. 11).

We use 2 channel systems regularly for recording the evoked brain potentials. The pickoff electrodes are located for the right and left side of the head in each case at a finger's width next to the vertex and above the mastoid.

We use biphase sound clicks for triggering the acoustically evoked brainstem potentials (AEBP). Depending upon the sound threshold, their intensity is adjusted so that they are firstly 60 dB above the sound threshold. Afterwards they are increasingly or decreasingly amplified or attenuated in 20 dB steps. We use click rates of 10 to 20 per second. 2000 clicks are evaluated in total as a rule. The modern "Bad Kissingen" computer assisted unit makes it possible to lay down in the screen dialog rejection criteria which reject EEG sections which cannot be evaluated according to filter and amplitude aspects. In this way studies on the acoustically evoked brainstem potentials can be performed regularly in nearly all adult patients without sedation of the patients solely with surface electrodes as noninvasive measurements. The computer determines the activity in a time window of 10 ms from start of stimulus. Evaluation takes place on the screen by the examiner identifying the typical wave patterns. Positive deflections are directed upwards in the recorder curves. Cursor marks are placed on the identified graphical elements of the waves. The computer then calculates the latencies of the individual waves in ms from start of stimulus and also the interpeak latencies, i.e. the running time of the stimulus conduction inside the individual sections of the auditory path.

Acoustically evoked brainstem potentials have developed topodiagnostically to an important examination method in the hand of the neurotologist in that the individual wave patterns can be assigned to typical structures of the auditory path. Wave I is attributed to stimuli in the ganglion of the vestibulocochlear nerve (fig. 11). This is followed by wave II. This can also be two-peaked. It arises because of stimulation in the ventral and dorsal cochlear nuclei. Stimulation of the superior olivary complex follows on this. This leads to the significant wave III. The subsequent wave IV is frequently unclear and can also be only weakly pronounced. It is assigned to the nucleus of the lateral lemiscus. The 10 ms window of the acoustically evoked brainstem potentials is dominated by the large biphase wave V which is attributed to the nuclei of the inferior quadrigeminal body. Wave VI corresponds to stimu-

Fig. 11: *Diagram of the longitudinal section of the human brain with drawn in auditory path and electrodes for deriving the acoustically evoked brainstem potentials (AEBP) with adjacent curve of the acoustically evoked brainstem potentials.*

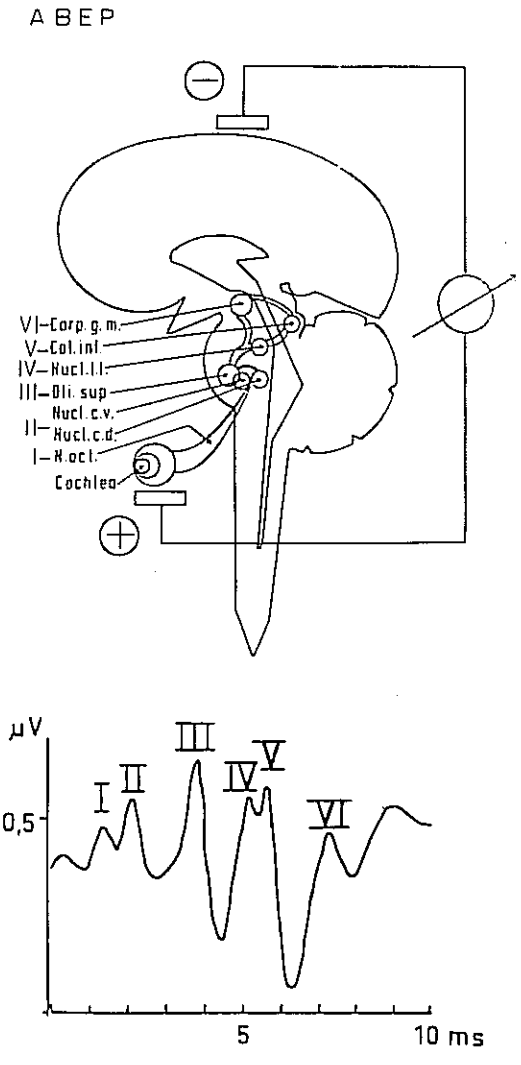


Table 5: *Standard ranges of the latency times of the acoustically evoked brainstem potentials.*

Graphical element	standard range of the latencies in ms
wave I	1.5 - 1.9
wave II	2.5 - 3.0
wave III	3.6 - 4.1
wave IV	4.8 - 5.2
wave V	5.5 - 6.0
wave VI	only - 7.6

lation of the medial geniculate body. At this point in time, the acoustic stimulation has already left the posterior cranial fossa. The standard ranges of the latency times of the acoustically evoked brainstem potentials used by us are shown in table 5.

On inhibition of a cochlear because of a pronounced sensory neural hearing loss, the lack of all brainstem waves, ipsilaterally and contralaterally, is observed as a rule. In a less severe hearing loss, wave V is still displayed, nevertheless as a rule with considerably prolonged latency time. Pontine disturbances, on the other hand, are characterized by a clear latency time prolongation of wave V. Wave I is in this case as a rule within the standard and wave III can be delayed in the case of distal lesions or it can still be within the standard in the case of proximal lesions. Disturbances in the midline of the brainstem result in the latencies of waves IV, V and VI in particular falling apart in the ipsilateral and contralateral stimulation pattern comparison.

The amplitudes of the graphical elements are assessed by us only by rough inspection. Here it is important that a certain size relation of waves among one another is maintained. Wave V forms typically the largest deflection, followed by wave III which is attributed to the superior olivary nucleus system.

The stimulation of the vestibulocochlear nerve itself then follows from the size (wave I). The acoustic nuclei form smaller and multiform waves as wave II and the lateral lemniscus nucleus as wave IV.

III. Neurosensory reactions

Neurosensory reactions are recorded objectively and quantitatively with the aid of different methods in neurotometry. Sensomotor reactions, for instance, can be recorded as head-body movement patterns with the aid of craniocorpography. The craniocorpogram is in this case a total graphical element which in turn consists of subelements. At first sight, the curve pattern of the stepping test craniocorpogram can be assigned to a certain disturbance. The peripheral vestibular disturbance (fig. 6) is characterized by a lateral deviation towards the disturbed vestibular periphery in the stepping test. The lateral deviations are not broadened.

In central disturbances of the medulla oblongata, in particular in the region of the accessory nucleus, there are broadened lateral deviations on stepping on the spot. The standing test, on the other hand usually shows in this case a normal small head and shoulder deviation pattern (fig. 6).

In diffuse brainstem disturbances, e.g. in extended pathway degeneration in the course of multiple sclerosis, in conditions after traumatic brainstem contusions, in extended arteriosclerosis with following cerebral sclerosis, diffuse and coarsened deviation patterns are observed both on stepping on the spot and on standing (fig. 6). The graphical elements of nystagmus curves and of audioencephalograms can similarly also be classified coarsely with regard to normal and pathological reaction patterns. Moreover we observe in the time/intensity curves of electronystagmography and audioencephalography after data processing editing of the curves, two further important parameters for the functional analysis and for the topodiagnostic classification of the disturbance. On one hand, in nystagmus analysis, the reaction strength can be determined in the shape of nystagmus quantity distribution of the beat rates or of the amplitudes of the slow nystagmus phases. On the other hand, data on reaction strength can be determined from acoustically evoked brainstem potentials with the aid of the wave amplitudes. In the case of nystagmus analysis, the method of reaction strength measurement is very successful while the information content in the case of acoustically evoked brainstem potentials is not so high.

The quantitative method of temporal reaction propagation enables the phenomenon of slowed down reaction propagation to be investigated in the time/intensity curves by latency time measurements. It is said in popular terms concerning a person with slow reactions: "He is slow on the uptake". Since reaction propagation in the nerve system is also linked to the metabolism of the involved pathway system, conclusions can be drawn regarding degeneration in connection with metabolic dysregulation in particular in cases of temporal reaction delay. The 3 basic categories for the assessment of neurosensory reactions, namely graphical elements, quantitatively measurable reaction strength and temporally measurable reaction propagation are discussed further below.

1. Typical electrophysiological graphical elements

a) Nystagmus patterns

The primary examination result can be seen in the curve shape of the recorded nystagmus beats. There are also disturbances which can be observed directly on the nystagmus beats.

The nystagmus signal which can be recorded with all types of nystagmography has a typical triangular shape (fig. 12). The two rising sides have different angles of inclination. The side with the stronger inclination designates the fast nystagmus phase, the side with the lesser inclination the slow nystagmus phase. The angle which the fast nystagmus phase forms with the zero line indicates the speed of the fast nystagmus phase taking into account the paper speed and the calibrated eye amplitude, and the angle which the slow nystagmus phase forms with the zero line indicates the speed of the slow nystagmus phase. The perpendicular line from the peak point to the zero line designates the nystagmus amplitude. The duration of the individual nystagmus beat can be measured on the zero line between the start of the fast nystagmus phase and the end of the slow nystagmus phase or vice versa.

The nystagmus beat can be directed either to the right or to the left and either upwards or downwards. In order to make clearly visible in the curve diagram these direction factors which refer alone to the fast nystagmus phase, the electronystagmogram is oriented in such a way that an upwards directed fast phase is plotted in the horizontal traces in the case of nystagmuses beating to the right and a downwards pointing fast phase is plotted for nystagmuses beating to the left. Upwards beating nystagmus points upwards in the verti-

cal trace of the ENG and downwards beating nystagmus points downwards.

In disturbances of the nystagmus signal itself, so-called square waves can occur. In this case the eye jerks with a fast phase into a lateral position. After it has remained there a short time, it jumps in turn with a fast phase to the opposite side. The slow eye movement phases are therefore lacking in such movement series. They occur in labile control positions of the brainstem, such as after nicotine or strychnine poisoning.

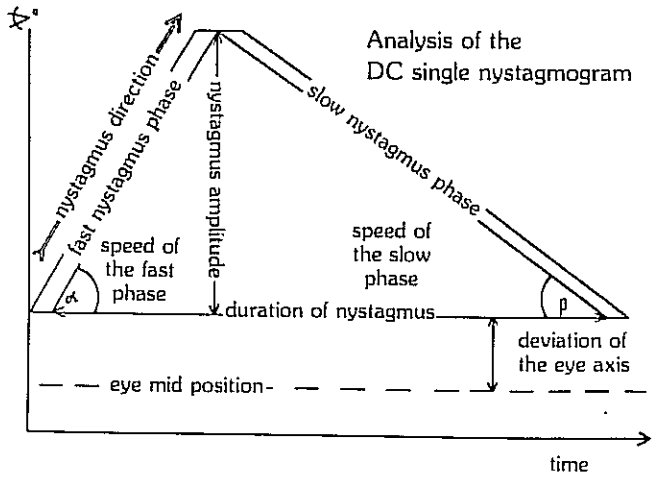
In the case of undulations, on the other hand, the fast nystagmus phase is missing. Here the eye wanders with slow phase in one direction. After an oscillating transition, it moves back again to the starting position. Undulations can be an expression of paralysis of the nystagmus generator, which occurs, for instance, in barbiturate intoxication, alcohol intoxication, etc.

Nystagmus can be at high frequencies with fine beats. We then talk about so-called "petite ecriture". This disturbance is expression of a change in the mesencephalic nystagmus generator. So-called nystagmus dysrhythmia exists if coarse beat nystagmuses alternate in time with fine beat nystagmuses or also nystagmus pauses during experimentally generated nystagmus series. Dysrhythmia is an expression of alternating instability in the brainstem.

Assessment of the nystagmus coordination behaviour by inspection is also very important. For this purpose the horizontal traces of both eyes in the polygraphic ENG are compared with one another. It is observed time and again that the nystagmus beats are not in the same direction but are phasewise or continuously in opposite directions and also that the fast nystagmus phases can beat towards the nose or towards the temples. In the first case one talks about so-called convergent nystagmus dissociation and in the second case about divergent nystagmus dissociation.

Previously, such disturbances were primarily brought into connection with multiple sclerosis and so-called internuclear ophthalmoplegia. Today we also know based on animal experiments that there are physiological nystagmus dissociations. However, these are limited in time and quantity. Increased nystagmus dissociations indicate degeneration of the nystagmus regulation, in particular in its paramedian pontine control component.

Fig. 12: *Diagram of the nystagmus signal with the individual components of nystagmus fine analysis.*



b) Wave pattern of the brainstem evoked potentials

In the case of acoustically evoked brainstem potentials, in contrast to nystagmus patterns, it is not a question of the derivation of an indicator potential of a single defined current source but of a calculated intensity distribution pattern after deriving 2000 audioencephalograms each of 10 ms duration. In principle these potentials form postsynaptic discharges which are calculated from the diffuse EEG between 2 electrodes which are favourable for projection. We have reported earlier in this article on the topographical assignment of the waves I - VI.

The typical pattern of acoustically evoked brainstem potentials is shown in fig. 11. In addition there is as conspicuous deviation the case of general amplitude reduction or amplitude inhibition. This pattern as a rule indicates that either the stimulus loudness was not sufficient or else that the stimulated ear is deaf. Excessive waves I with subsequent amplitude reduction of waves II - V point to a disturbance in the cerebellopontine angle.

Disturbances in the olivary nucleus system lead among other things to amalgamation of the complex of wave III. Isolated amalgamation of wave V is observed among other things in multiple sclerosis. However, apart from amalgamation of wave V, amalgamation of wave III can also occur, wave V after amalgamated wave III possibly being highly delayed but shaped correctly.

In pathway degenerations, brainstem traumata, inflammatory diseases, metabolic disturbances, cardiovascular disturbances, etc., widely differing deformations of the wave patterns can be observed depending upon the site of the lesion. But it is still difficult to draw up a binding system of pathological and simultaneously dignostically unequivocal types without including latency time assessment at the same time.

2. Representative reaction intensities

a) The central nystagmus frequency

From a topodiagnostic viewpoint, the expressiveness of caloric vestibular testing with characteristic display in the butterfly system according to Claussen is unexcelled for studying the vestibulo-ocular system. We attribute the highest diagnostic information content to this test.

Using the frontobasal flat section diagram through the floor of the IVth ventricle shown in fig. 9 with special accentuation of the medial longitudinal bundle, both vestibular nerves with the terminal cupular organs of the semicircular canals, the old cereballar inhibiting bundles of the nucleus fastigii through the juxtarestiform body to the vestibular nuclei of the opposite side, the envisaged assignment of reaction patterns with inhibiting characteristic can be made. The typical pattern of vestibular receptor failure with spontaneous vestibular nystagmus to the opposite side is located to the right below (dashed characteristic in the diagram). A cold reaction is simulated by the spontaneous vestibular nystagmus. On separation of the superior proximal vestibule from the ganglion, as occurs in a petrous bone fracture, for instance, spontaneous vestibular nystagmus frequently is not present. Then both hot and cold inhibition are observed on the side concerned (centre, below).

If both vestibular receptors fail, e.g. as a result of intoxication of the inner ear with aminoglucosidic antibiotics, then the pattern of the small butterfly is obtained (below, left). A common feature of all three patterns described is that the nystagmus reactions occur coordinated as expressed by the direction arrows at the lower right edge of the picture below.

In cerebellopontine angle tumors with pressure on the vestibulocochlear nerve and the juxtarestiform body, the pattern of a peripheral vestibular inhibition is observed on the side of the tumor, whereas on the opposite side due to absence of cerebellar restraint on the vestibular nucleus, disinhibition of both caloric reactions occurs (right picture margin, 2nd pattern line from below).

Central vestibular inhibitions must be differentiated from peripheral ones. Whereas in peripheral vestibular inhibitions the caloric reactions are blocked with reference to the stimulation of one ear, it is found that in central vestibular inhibitions, nystagmus reactions are inhibited in a certain beat direction. These disturbances arise in the case of lesions in the actual nystagmus generator, which forms the nystagmus beats on interaction of the oculomotor nucleus with the central paramedian pontine reticular formation. We also call this pattern the type of nystagmus inhibition direction predominance, both for the nystagmus frequency and frequently for nystagmus amplitude. One beat direction is blocked in caloric stimulation. In the present case, it is a question of blockage on the left side. This becomes in addition more conspicuous by increased nystagmus dissociations in monocular reaction recording. These dissociations can beat both divergently and convergently. The pattern of nystagmus inhibition direction predominance is shown to the right at the margin in the 3rd pattern line from below in fig. 9 together with the direction symbols for nystagmus dissociation. The nystagmus frequency butterfly is marked with "F" and the nystagmus amplitude butterfly with "A".

Total blockage of the nystagmus function in the mesencephalic nystagmus generator represents the bilateral inhibition pattern in the line lying above. This small butterfly requires additional parameters for its differentiation from the small nystagmus butterfly in the case of bilateral receptor intoxication, such as of accompanying conspicuous nystagmus dissociation. In addition, increased staggering in the standing test craniocorpogram with normal deviation width in the stepping test craniocorpogram is frequently found in these cases (fig. 6, right above).

Abducens paresis or paresis of the lateral rectus muscle also produces the pattern of nystagmus inhibition direction predominance on monocular recording on one eye. However, on multichannel recording, a normal vestibulo-ocular reaction is detected in the opposite eye without difficulty. In this way this pattern of effector inhibition can be differentiated with certainty from that of nystagmus generator inhibition.

Reaction disinhibition of vestibulo-ocular nystagmus of peripheral and central origin is summarized schematically in fig. 10 for different topographical lesion sites. The inhibited cold reaction under normal hot reaction indicates disinhibition of the information production in the receptor. This pattern shown in fig. 10 on the right below shows that cold syringing does not succeed in pushing down the information tone in the disturbed receptor to a level below that of the

opposite side. Such patterns occur in particular in connection with ischemic stimuli. This is the actual type of Meniere's disease.

In the lower pattern row of fig. 10, the pattern of the cerebellopontine angle tumor is located in the 2nd position from the right. In this case, both the superior vestibular nerve and the juxtarestiform body, which produces the restraint from the nucleus fastigii from the cerebellum to the vestibular nuclei of the opposite side, are severely disturbed. Because of this, inhibition of both caloric vestibular reactions results ipsilaterally and disinhibition of the vestibular reactions contralaterally.

Pure disturbance of the restraining function of the vestibular nucleus of the opposite side is shown on the right margin of the diagram in the 2nd diagram row from below. This pattern is observed frequently in the dorsolateral oblongata syndrome. It exists there frequently in connection with arteriosclerosis of the posterior inferior cerebellar artery, in the so-called PICA syndrome. Reference is also made in this connection to the pattern located to the right in the centre of the diagram in fig. 6 in the craniocorpogram.

One can differentiate between 2 groups in the case of bilateral disinhibition of nystagmus in the brainstem. To the left below in fig. 10 are to be found the frequency butterfly calorigram (on the far left) and the amplitude butterfly calorigram (second from the left) of the deep brainstem disinhibition. In arteriosclerosis, synringobulbia, deep seated focuses of demyelinisation of multiple sclerosis, both inhibition of the nystagmus frequency and of the nystagmus amplitude occur. We attribute this to reduced restraint of the vestibular nucleus on both sides by the cerebellum.

The actual disinhibition of the nystagmus generator causes the already long known ENG pattern of "petite ecriture" (cf. chapter III.1.a). In normal eye deflection, the nystagmus generator performs many small switching steps. The butterfly pair with the frequency butterfly disinhibited on all sides and the inhibited amplitude butterfly shown to the right above in fig. 10 results accordingly. This pattern frequently arises in ischemic conditions after a blood pressure drop in the entire circulatory system. The fresh cardiac infarction also belongs to this. Based on purely subjective symptoms, such cases with vertigo, nausea, vomiting, hearing loss and tinnitus can by all means be falsely classified as Meniere's disease.

Finally the pattern of disinhibition direction predominance has to be mentioned under the disinhibition conditions. This is the actual pattern of so-called "nystagmus preponderance" which Fitzgerald and Hallpike have described in contrast to peripheral vestibular inhibition. We observe this pattern in temporoparietal degenerations, posttraumatic epilepsies and hemorrhages in the lateral sulcus of the cerebrum. Occasionally a temporal lobe tumor exists in connection with this pattern. The pattern of caloric nystagmus disinhibition direction predominance is shown on the left above in fig. 10. The presented functional neurotological topodiagnostics have been worked out in detail for caloric vestibular testing based on representative reaction intensities. It represents the backbone of our neurotological topodiagnostics to which we add improvements which are based on further neurotological methods presented here.

b) The amplitudes of the evoked brainstem potentials

In analogy to the nystagmus amplitude and to the nystagmus frequency, the amplitudes of the acoustically evoked brainstem potentials (AEBP) represent a representative reaction intensity. Whereas in the triangular nystagmus pattern it is possible to proceed from a baseline from which the amplitudes are measured numerically, in the wave pattern of the acoustically evoked brainstem potentials, there must be established a system of reference points between which the wave heights are measured. According to the proposals of Maurer, waves I and V are measured from the positive peak to the following negative valley. Waves II, III, IV, VI and VII are, on the other hand, determined from the negative valley to the following positive peak. It results from this procedure that the rising side of wave I (fig. 11) is frequently masked by the stimulus artifact and the descending flank of IV is absorbed by wave V.

Since it is a question of a calculated potential without a systematic zeroline, numerous standard variants must be taken into account making the amplitude measuring method very unpractical. Maurer indicates that in most cases there results a common base for waves IV and V with a valley which does not extend to the baseline. As further variant, there is frequently a wave in which the separate peaks can no longer

be identified. Then it is a question of a combined complex from wave IV and V. Separation of the peaks by another electrode projection can possibly be brought about by electrode repositioning. Amplitude measurement is subjected to numerous influences, such as stimulus loudness, the age of the patient, selection of the derivation points, the headphones, the stimulus forms, the stimulus rate and the polarity of the stimulus. Accurate determination of a low electrode skin resistance is very important. Filters and amplifiers further play a large part. Therefore numerical measurement of the absolute amplitudes has not yet become established with us.

3. Temporal reaction pattern

a) Vestibular cumulation latencies

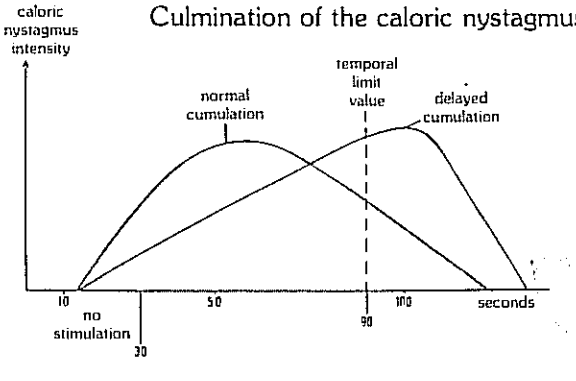
The monaural caloric vestibulo-ocular nystagmus reaction can be viewed as an important indicator reaction of the brainstem function and of the connected vestibular receptors. Fig. 13 shows diagrammatically the build up and decline of the postcaloric nystagmus reaction I with normal cumulation latency (left curve) and delayed cumulation latency (right curve). The standard values of the caloric cumulation latencies are reproduced in section II.4.

We observe clearly shortened caloric cumulation latencies as a rule only in conditions after surgical removal of the mastoid and middle ear structures, as they occur after surgical removal of cholesteatomas. The thermal gradient on calorization with water can then act so directly on the lateral semicircular channel that an overquickly appearing caloric nystagmus reaction occurs.

The phenomenon of slowed down caloric reactions is of essentially greater significance. In this case the pulsation rate of the so-called central nystagmus frequency is within the standard in the culmination range. However, the run up to development of the postcaloric reaction maximum requires a too long period so that the reaction reaches the culmination point only with a delay. We observe such delayed reactions in more than 90% of the cases only in connection with central or combined peripheral and central functional disturbances of the equilibrium. They are an expression of delayed stimulation conduction in the brainstem and thus also expression of a delay in the actual formation of the nystagmus pulsation series development. The phenomenon can occur on one side but also on both sides.

The low correlation of the delayed nystagmus culmination with the central nystagmus disinhibition or inhibition indicates that we are dealing here with an important further indicator of central pathology.

Fig. 13: *Diagram of the different patterns of the culmination phenomenon of the postcaloric nystagmus reaction. It can be observed that the cumulation latencies of the two displayed curves are of very different size.*



b) Latency time pattern of acoustically evoked brainstem potentials (AEBP)

Determining the latency time of the different wave patterns is the most important measure for the evaluation of the calculated acoustically evoked brainstem potentials (AEBP). The standard ranges used by us are summarized in table 5.

The standard ranges of the acoustically evoked brainstem potentials stated by us in table 5 refer to adults. It is known that the latency time values of AEBP in the newborn, infants and schoolchildren are delayed according to the maturing of the brain. Waves III, IV and V in particular are delayed in this case. The stimulation rate also plays a role in the prolongation of the latencies of the acoustically evoked potentials. We use currently as a rule click frequencies with a stimulation rate of 10 - 20 Hz. The delay in the acoustically evoked brainstem potentials is related by us in each case to the regional anatomy of their place of origin. Therefore it is also of significance that not only the individuals waves are determined but also the interpeak latencies in our standardized evaluation programs of the "Bad Kissingen" unit from Medelectronic (table 6).

IV. The syndrome of the slowed down brainstem

Against the background of the pathodiagnostic and localisatory indicators discussed in chapter III, there is a group of patients in which both the cumulation latency of the caloric vestibular test and the latencies of the acoustically evoked brainstem potentials are prolonged.

This is an expression for degenerative impairment in the stimulus conduction of the relevant system stimuli in the brainstem. In contrast to the basic distribution of the neurotological group NOBKIb from Bad Kissingen, more men than women appear to suffer from the syndrome of the slowed down brainstem (table 7).

Table 6: *Standard values of the interpeak latencies of the acoustically evoked brainstem potentials (AEBP).*

Interpeak latencies	standard range in ms
I - III	2.0 - 2.6
I - V	3.8 - 4.4
III - V	1.8 - 2.5

As far as the age distribution is concerned, the mean value and standard deviation of the subgroup with syndrome of slowed down brainstem coincide with the same parameters of the total group.

Vertigo and hearing loss are the salient symptoms in the field of statoacoustic complaints (table 8).

Among the further subjective complaints, defective vision with 65.03% is the most frequent followed by very frequently occurring cardiac insufficiency symptoms with 30.06% (table 9).

With the aid of equilibriometry, extensive functional pathology could be exposed in the patients. Caloric reaction retardation is common to all patients with the syndrome of slowed down brainstem (table 10). In around one third of the patients in each case, vestibular inhibition of peripheral or central origin, vestibular recruitments of the type of the catching up phenomenon or of latent brainstem disinhibition, disinhibition of the optokinetics or pathological nystagmus dissociation is ascertained.

In summary, objectifiable disturbed equilibrium is ascertained in all 163 first examined patients with the syndrome of slowed down brainstem. The seat of the main disturbance can be localized in more than 90% of all cases. Nevertheless, it must be admitted that in 6 cases = 3.68%, the diagnostic indicators speak predominantly for peripheral vestibular disturbance (table 11). Latency time prolongations, nystagmus dissociations and certain optokinetic disturbances are indeed still present here. However, they determine the diagnosis so weakly that we must assume predominantly peripheral vestibular disturbance. Such conditions occur in particular in arteriosclerosis and in the condition after cardiac infarction. We also observe them in virus infections. In such cases of inner ear pathology, the acoustically evoked brainstem potentials, as a rule of all waves, are prolonged in their latency time.

Provided the waves can be detected and the interpeak latencies determined, it is ascertained that the latter have normal values in the sense of table 6.

However, it is clear in the case of the patients who fall statistically under the syndrome of the slowed down brainstem that 52.15% suffer from a pure central disturbed equilibrium. In addition there are 37.42% in whom a clearly pronounced diffuse neurosensory disturbance exists both in the receptor area and in the brainstem area. We summarize the latter under the term of combined peripheral and central disturbed equilibrium.

More than 80% of our patients with the syndrome of slowed down brainstem show sound perception deafness in

Table 7: *Age and sex distribution in first examined neurotological patients of the KOBKIb data bank.*

Parameter	all vertigo patients	syndrome of the slowed down brainstem
number	1031	163
men	44.1%	57.0%
women	55.9%	43.0%
age	58.3 +/- 18.3 y	58.6 +/- 18.5 y

Table 8: *Statoacoustic complaints in 163 patients with syndrome of slowed down brainstem (NOBKIb)*

Complaints	n	%
vertigo	156	95.71%
nausea	97	59.51%
tinnitus	93	57.06%
hypoacousis	135	82.82%
deafness	2	1.23%
pressure in the ear	17	10.43%

Table 9: *Further complaints in 163 patients with syndrome of slowed down brainstem (NOBKIb)*

Complaints	n	%
cardiac insufficiency	49	30.06%
condition after skull trauma	24	14.72%
diabetes mellitus	9	5.52%
paresthesias	7	4.29%
depressions	2	1.23%
headaches	28	17.18%
stupor	7	4.29%
exhaustion conditions	6	3.68%
defective vision	106	65.03%
flickering in front of the eyes	17	10.43%
double images	8	4.91%
disturbed sense of smell	1	0.61%
disturbed taste	2	1.23%

Table 10: *Equilibriometric findings in 163 first examined patients with the syndrome of slowed down brainstem (NOBKIb)*

Finding	n	%
vestibular inhibition	59	36.20%
vestibular recruitment	61	37.42%
vestibular decruitment	8	4.91%
caloric reaction retardation	163	100.00%
pontine nystagmus disinhibition	27	16.56%
mesencephalic nystagmus inhibition	9	5.52%
disinhibition of the optokinetics	50	30.67%
nystagmus dissociation	58	35.58%
pathological downwards nystagmus	12	7.36%
standing ataxia	39	23.93%
CCG deviations of the Parkinson type	4	2.45%
stepping ataxia	23	14.11%
atactic side deviation	33	20.25%
inhibited side deviation	35	21.47%

the audiometric check. In 57.67%, it is a question of cochleobasal high tone deafness. Both traumatic deafness and age induced degenerative hearing defects of the presbycousis type belong here. The type of pure sound conduction deafness resulting from middle ear lesions is extremely rare. In all patients, latency time prolongation of the acoustically evoked brainstem potentials is ascertained according to definition (table 12). However, in around one third of the patients, deformation and destabilization of the graphical elements both of the acoustically evoked brainstem potentials and of the acoustically evoked brain cortex potentials is observed. These disturbances can consist in particular in the case of acoustically evoked brainstem potentials (AEBP) of the extinction of individual wave complexes or else only in shifting the amplitude relation between wave V, wave III and wave I.

We assume metabolism-dependent degeneration effects in the neuronal network of the brainstem to be the cause of the syndrome of the slowed down brainstem. These changes must be of a general nature, since they affect both the vestibular and the acoustic system. Metabolic alterations because of hypoxidosis, ischemia, thixotropy deviations and general degeneration due to age come into question as cause. Further, it must be considered that disturbances of the protein and energy metabolism have also occurred there.

On the other hand, we observe pure nystagmus inhibition and disinhibition conditions after cross-connection lesions in which one or the other neurotransmitter tonus exercises a too strong inhibiting or disinhibiting influence on the neuronal pathways and control circuits. In the case of the slowed down brainstem, we rather assume that the total activity in the neuronal network has been brought low.

This also applies in particular for the presynaptic and postsynaptic sodium ion and chlorine ion channels.

The term of the syndrome of the slowed down brainstem was born from practical clinical observation. The popular expression "slow on the uptake" corresponds to it.

Table 11: *Equilibriometric diagnosis in 163 first examined patients with the syndrome of slowed down brainstem (NOBKIb)*

Functional diagnoses	n	%
objectifiable disturbed equilibrium	163	100.00%
peripheral vestibular disturbance	6	3.68%
central disturbed equilibrium	85	52.15%
combined peripheral and central disturbed equilibrium	61	37.42%

Table 12: *Audiometric findings in 163 first examined patients with the syndrome of the slowed down brainstem (NBOKIb)*

Finding	n	%
cochleobasal high tone deafness	94	57.67%
cochleoapical low tone deafness	2	1.23%
pancochlear deafness	32	19.63%
combined middle and inner ear deafness	7	4.29%
pure sound conduction deafness	1	0.61%
deformation and destabilization of the acoustically evoked brainstem potentials	49	30.06%
latency time prolongations of the acoustically evoked brainstem potentials	163	100.00%
pronounced wave I disturbances	3	1.84%
pronounced wave III disturbances	13	7.98%
pronounced wave IV disturbances	2	1.23%
deformation and destabilization of the acoustically evoked brain cortex potentials	52	31.90%
hypermobile tympanogram	13	7.98%
inhibited tympanogram	7	4.29%
retracted tympanogram	19	11.66%

V. The treatment of vertigo, nausea and tinnitus with the combination preparation from petroleum, ambergris, cocculus and conium: **Vertigoheel**

1. The clinical application of **Vertigoheel**

Because of its good compatibility and its clear subjective relief described time and again in vertigo and nausea, we have occupied ourselves more closely with the combination preparation from petroleum, ambergris, cocculus and conium, namely **Vertigoheel**. It is possible to demonstrate

Table 13: *Therapy with a combination preparation from conium, cocculus, ambergris and petroleum (**Vertigoheel**) in first examined neurotological patients (NOBKIb)*

Group	Cocculus compositum tablets		liquid	
	n	%	n	%
all vertigo patients (n = 1031)	347	33.70%	67	6.50%
all patients with vertigo, nausea, tinnitus and hearing loss (n = 506)	149	29.50%	36	7.10%
peripheral vestibular disturbance (n = 132)	29	21.97%	6	4.60%
central equilibrium disturbance (n = 410)	148	36.10%	23	5.61%
combined peripheral and central disturbance (n = 307)	123	40.07%	25	8.10%
syndrome of the slowed down brainstem (n = 163)	74	45.40%	11	6.90%

numerous objective improvements in symptoms with the aid of neurotological methods.

In the case of 1031 vertigo patients of the neurotological patient group NOBKIb of all age classes, we have seen the indication for treatment with **Vertigoheel** in 40.2% of all patients after the first examination (table 13.) **Vertigoheel** was used here most frequently in the case of the patients with the syndrome of slowed down brainstem, namely in 52.2%.

Among the most important other medicines which are used in combination with **Vertigoheel** or alone in the syndrome of the slowed down brainstem, dihydro-ergotoxine in the shape of Orphol plays an outstanding part. It is attempted with this medicine, exactly as with calcium antagonists, to influence in a regulative way the vascular neurotransmitters of the cerebral vessels. Meclozine and dimenhydrinate play a significantly smaller role as antivertiginous agents compared with **Vertigoheel**. They together are used only half as frequently (table 14).

It can be seen from table 13 that the overwhelming percentage of the patients is treated with **Vertigoheel** tablets, namely 3 x 1 per day. The smaller proportion, in particular older patients with dry mucous membranes and swallowing difficulties, receive **Vertigoheel** liquid, 3 x 20 drops. The patients basically prefer tablets to the liquid form, since measuring out the dose is simpler with the tablets. As can be seen from table 15, we have also prescribed **Vertigoheel** tablets for around 12% of the patients in whom disturbed equilibrium cannot be objectified with our methods. As a rule we then attempt to influence the combination of vertigo and tinnitus with tinnitus predominating (table 17). Among the further complaints of all 377 patients who present an indication for treatment with **Vertigoheel** tablets based on their first examination, the first place is taken by defective vision followed by cardiac insufficiency and headaches (table 17).

Out of the 377 patients treated with **Vertigoheel** tablets, 207 patients = 54.91% became free of complaints

Table 14: *Therapy in 163 first examined patients with the syndrome of the slowed down brainstem (NOBKIb)*

Medicine	n	%
conium, cocculus, ambergris, petroleum (**Vertigoheel**)	85	52.2%
meclozine (Peremesin)	20	12.3%
dimenhydrinate, nicotinic acid, vitamin B_6 (Vertigo Vomex)	23	14.1%
meclofenoxate (Helfergin)	9	5.5%
naftidrofurylhydrogenoxalate (Dusodril)	4	2.5%
pentifyllin, vitamin A, vitamin E (Cosaldon A and E)	21	12.9%
vincamin (Vincamin)	31	19.0%
dihydroergocornin, dihydroergocristin, dihydroergocryptin (Orphol)	84	51.5%

Table 15: *Equilibriometric diagnoses in 377 patients with indication for therapy with* **Vertigoheel** *tablets (NOBKIb)*

Functional diagnoses	n	%
objectifiable equilibrium disturbance	332	88.06%
peripheral vestibular disturbance	29	7.69%
central equilibrium functional disturbance	156	41.38%
combined peripheral and central disturbance	128	33.95%

Table 16: *Statoacoustic complaints in 377 patients with the indication to therapy with* **Vertigoheel** *tablets (NOBKIb)*

Complaints	n	%
vertigo	366	97.08%
nausea	213	56.50%
tinnitus	242	64.19%
hypoacousis	334	88.59%
deafness	6	1.59%
pressure in the ears	25	6.63%

Table 17: *Further complaints in 377 patients with indication for therapy with* **Vertigoheel** *tablets (NOBKIb)*

Complaints	n	%
cardiac insufficiency	167	44.30%
condition after skull trauma	43	11.41%
diabetes mellitus	42	11.14%
paresthesias	8	2.12%
depressions	9	2.39%
headaches	84	22.28%
stupor	13	3.45%
exhaustion conditions	21	5.57%
defective vision	283	75.07%
flickering in front of the eyes	52	13.79%
double images	14	3.71%
disturbed sense of smell	9	2.39%
disturbed taste	6	1.59%

in the course of treatment by **Vertigoheel** and also by the combination with other medicines, so that they no longer required our neurotological diagnosis and therapy. Similar conditions resulted from treatment with **Vertigoheel** liquid. Nevertheless, it must be taken into account that a number of patients firstly change from **Vertigoheel** liquid to **Vertigoheel** tablets.

2. A study with the medicine Vertigoheel in selected vertigo, nausea and tinnitus patients

The homeopathic medicine **Vertigoheel** made from petroleum, ambergris, cocculus and conium, has proven both its effectiveness and its good compatibility in a first study in the investigation on selected vertigo, nausea and tinnitus patients. We have reported on this in various places. These results are the basis of using **Vertigoheel** consistently in the neurotological therapy of vertigo, nausea and tinnitus patients as we have described in chapter V.1.

In the first therapy study with **Vertigoheel**, the examination was performed on 40 patients before and after 14 day treatment with 3 x 3 tablets of **Vertigoheel** per day. The group of patients was composed of 55% men and 45% women (table 18). Among the subjective vertigo complaints, the feeling of uncertainty and rotating vertigo ranked clearly ahead of vestibular vertigo, inclination to fall and the lifting feeling (table 19). All symptoms show a constant improvement in symptoms under therapy. The nausea symptoms (table 20) also improve during the 14 day treatment with **Vertigoheel** over all individual symptoms. The subjective self estimation of the patients produces in 57.5% the feeling of an improvement in symptoms, which extends up to complete elimination of the symptoms. The patients were treated exclusively with **Vertigoheel** during the study.

It is also shown that in those patients in whom attacks of vertigo continue to occur, the period of the individual attacks is clearly reduced.

Objective equilibriometric tests prove in the first **Vertigoheel** study that the combined medicine made from conium, cocculus, ambergris and petroleum improves both the vestibulo-ocular and the vestibulo-spinal reactions. Thus, in the synoptic caloric butterfly characteristic evalua-

Table 18: *First therapy study with conium, cocculus, ambergris and petroleum (* ***Vertigoheel****) in otherwise untreated vertigo and nausea patients*

Group parameter	first study
number	40
men	55%
women	45%
age	48.2 +/-14.7 y

Table 19: *First therapy study with conium, cocculus, ambergris and petroleum (* ***Vertigoheel****) in 40 vertigo and nausea patients*

vertigo symptoms	before treatment	after treatment	difference
staggering	37.5%	32.5%	- 5.0%
lifting feeling	17.5%	7.5%	-10.0%
rotating vertigo	55.0%	35.0%	-20.0%
falling to the right	15.0%	5.0%	-10.0%
falling to the left	22.5%	15.0%	- 7.5%
black out	32.5%	17.5%	-15.0%
uncertainty	67.5%	60.0%	- 7.5%

Table 20: *First therapy study with conium, cocculus, ambergris and petroleum (* ***Vertigoheel****) in 40 vertigo and nausea patients*

nausea symptoms	before treatment	after treatment	difference
outbreaks of sweat	32.5%	15.0%	-17.5%
nausea	40.0%	15.0%	-25.0%
retching	17.5%	7.5%	-10.0%
vomiting	17.5%	7.5%	-10.0%
collapse	7.5%	2.5%	- 5.0%

Table 21: *1st therapy study with conium, cocculus, ambergris, and petroleum (* ***Vertigoheel****) in 40 vertigo and nausea patients*

Subjective self estimate	after treatment
improvement in symptoms	57.5%
no improvement	42.5%

Table 22: *1st therapy study with conium, cocculus, ambergris and petroleum (* ***Vertigoheel****) in 40 vertigo and nausea patients*

Caloric characteristic pattern	before treatment	after treatment	difference
normal caloric reactions	15.0%	47.5%	+32.5%
pathological caloric reactions	85.0%	52.5%	-32.5%

tion of the monaural vestibulo-ocular reaction, an improvement by 32.5% is observed (table 22). In the case of the vestibulo-spinal signs of disease accompanied by disturbances in gait and head-body deviations, an improvement by 22.5% is observed (table 23). A clear improvement in transit time can also be verified in the evaluation of the acoustically evoked brainstem potentials in the entire pontomedullar course from the acoustic nuclei up to the lower quadrigeminal region.

The treatment study with **Vertigoheel** in 40 vertigo, nausea and tinnitus patients has therefore shown that this medicine exercises a neuropharmacological effect on subjective complaints and also on the objectively measurable functional changes of the brainstem. At the end of the study, the question naturally arose to what extent are the changes named caused by the 4 individual components petroleum, ambergris, cocculus and conium. For this reason we performed a second study with the special question regarding the action of the components of **Vertigoheel**.

3. A study with the components petroleum, ambergris, cocculus and conium of the medicine Vertigoheel in selected vertigo, nausea and tinnitus patients

For the second study, 4 groups of 15 persons each were formed from 60 vertigo, nausea and tinnitus patients (table 24). Special tablets were prepared for the treatment:

1. Petroleum D 6 bound to cornstarch, magnesium stearate and lactose.
2. Ambergris D 4 bound to cornstarch, magnesium stearate and lactose.
3. Cocculus D 2 bound to cornstarch, magnesium stearate and lactose.
4. Conium D 1 bound to cornstarch, magnesium stearate and lactose.

The patients were treated in each case with 3 x 1 of these tablets for 14 days. An extensive neurotological examination, as described in chapter III took place before the start and after conclusion of treatment.

Table 23: *1st therapy study with conium, cocculus, ambergris and petroleum (* **Vertigoheel** *) in 40 vertigo and nausea patients*

CCG pattern	before treatment	after treatment	difference
normal CCG pattern	25.0%	47.5%	+22.5%
pathological CCG pattern	75.0%	52.5%	-22.5%

Table 24: *Characteristics of the 4 groups of the 2nd therapy study on petroleum, ambergris, cocculus and conium (constituents of* **Vertigoheel** *)*

Parameter	group I	group II	group III	group IV
active ingredient	petroleum	ambergris	cocculus	conium
number	15	15	15	15
men	6	5	7	7
women	9	10	8	8
age	39.25 +/- 11.26	39.40 +/- 15.81	37.80 +/- 18.40	42.23 +/- 10.80

The study produces a large number of classified findings. It starts with the psychophysical findings of the anamnesis and the subjective self estimate (table 25). Evaluation of the objective equilibriometric measurements follows. These include vestibulo-ocular nystagmus with spontaneous nystagmus, caloric nystagmus frequency, caloric cumulation latency and caloric nystagmus amplitude. Following these, the vestibulo-spinal equilibriometric test is evaluated. This is craniocorpography of the stepping test and of the standing test. The retino-ocular system is also assessed. Finally assessment of the stimulus conduction of the acoustically evoked brainstem potentials (AEBP) is examined by reference to the latency times. The symptom assessments entered in table 4 are given graphical markings for improvements, worsenings or equality of symptoms (table 25).

On examining table 25, it becomes clear that the improvements in symptoms refer to the anamnesis, the vestibulo-spinal system and in part to the acoustically evoked brainstem potentials. Optokinetic vision sequence control shows rather a worsening in symptoms. A summary of the assessments with regard to improvements, worsenings and equality of findings is made at the end of table 25. In this case each individual "+", each individual "-" and each individual "o" are added separately for the groups I - IV. In the analysis of the components of **Vertigoheel** with the examinations described, an exclusively positive or exclusively negative effect could be ascertained in no single substance.

One observes gradually that the strongest positive effect is produced by the constituent cocculus. Conium is in second place followed by petroleum. Ambergris develops the weakest positive effect. Negative effects are observed most frequently for petroleum and less frequently for ambergris, cocculus and conium. On comparison with the first **Vertigoheel** study, it must be assumed that the components in **Vertigoheel** develop an additional helping success because of synergistic effects when they are combined to a single medicine.

VI. On the action mechanism of Vertigoheel in the syndrome of the slowed down brainstem

The syndrome of the slowed down brainstem is characterized by objective reaction delays of brainstem control processes, such as prolonged caloric cumulation latency, prolonged perrotatory culmination latency and prolonged brainstem latencies of the significant waves of the acoustically evoked brainstem potentials (AEBP). A number of significant subjective complaints of the patients stand opposite these objective neurotometric findings.

These include: vertigo, nausea, staggering, tinnitus, hearing disturbances and a general drop in performance. The origin of this combination of complaints is seen in a degenerative retardation of metabolic processes in the brainstem.

Corresponding to the above described statistical analysis of the effectiveness of the individual components (table 25), the most important constituent of **Vertigoheel** is cocculus. The ingredient of cocculus is known as picrotoxin. Picrotoxin was earlier viewed as the most important antagonist against barbiturates. It stimulates broad sections of the central nervous system including the brainstem.

Table 25: *Emphasized findings on comparison of the neurotological examinations of the 4 special therapy groups on treatment with petroleum, ambergris, cocculus and conium—2nd* **Vertigoheel** *study*

Parameter	group I petroleum	group II ambergris	group III cocculus	group IV conium
Anamnesis:				
vertigo and nausea symptoms	+	++++	+++	++
vertigo triggering	+++	+	++++	++
tinnitus	+++	+	++++	++
headaches	-	-	°	++++
subjective self estimate	++++	++	+++	+
Vestibulo-ocular nystagmus:				
spontaneous nystagmus frequency	+	++	-	++++
caloric nystagmus frequency	++++	+++	+	++
caloric cumulation latency	++	++++	+++	++
caloric nystagmus amplitude	++	+	-	++++
Craniocorpography:				
stepping test + lateral deviations	+++	++	++++	+
standing test disturbances	+	++	++++	+++
Optokinetics:				
frequency kite vectors	----	-	---	--
time sequence kite vectors	---	-	--	----
Acoustically evoked brainstem potentials (AEBP):				
stimulation of the right ear	-	--	++++	++
stimulation of the left ear	---	--	+++	-
Summary of the assessments:				
+ =	24	22	33	29
- =	12	7	7	7
° =	-	-	1	-

Explanation of symbols:

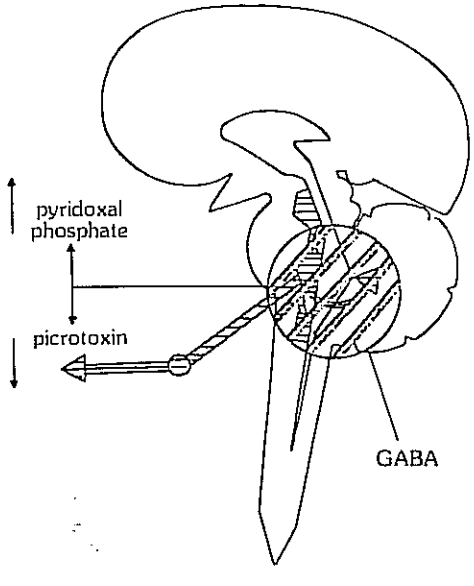
+ = expected improvements in symptoms
- = unexpected worsening of symptoms
° = unchanged symptoms

Gamma-aminobutyric acid (GABA) is an important inhibitory neurotransmitter, among other things also in the brainstem. Through gabaminergic loops, the cerebellum inhibits the vestibular nuclei, for instance. The inhibiting action of GABA can be increased, for instance, by the increased build up of gamma-aminobutyric acid by administering vitamin B_6 (pyridoxal phosphate) or similar medicines (fig. 14). Picrotoxin, on the other hand, is a typical antagonist of the inhibitory neurotransmitter GABA (fig. 14). Picrotoxin should develop its effect on a mechanism following receptor activation by blocking a chlorine ion channel opened by GABA. In this way a certain part of the inhibitions of the brainstem is reduced by restraining gabaminergic loops and the brainstem is increased in its activity on the other hand. This mechanism should be of extraordinary significance for the activation of the slowed down brainstem in the syndrome discussed here.

The next most effective constituent is coniin, or as an alkaloid also known as alpha-propyl-piperidine. It has an action both similar to nicotine and to curare. We know from the description of the poisoning on the death of Socrates that increasing paralysis of the spinal motor centres can occur in coniin poisoning. Paralysis of the motor centres in the medulla oblongata is particularly striking. Picrotoxin is frequently administered as an antidote. Cocculus and conium influence one another mutually in their special effect on the brainstem.

The aromatic compounds contained in ambergris and the cholesterol-like ambrain, as well as the active hydrocar-

Fig. 14: *Diagram of the influence of the neurotransmitter action of GABA in the brainstem. The active agent picrotoxin originating from the Anamirta cocculus is in a position to act against the inhibitory neurotransmitter GABA in its effect.*



bons in petroleum, should intensify the overall effect in a mutually potentising sense.

Vertigoheel contains in the administered form per tablet cocculus D 4, 210 mg; conium D 3, 30 mg; ambergris D 6, 30 mg; petroleum D 8, 30 mg.

Both the essential individual components of **Vertigoheel** and the total prepearation develop an improving effect towards vertigo and noises in the ear. It can be ascertained objectively that equilibrium regulation in the brainstem is normalized. This applies in particular for the vestibulo-ocular and vestibulo-spinal portion. Improvement in the stimulus conduction in the acoustic pathway system can also be observed.

Vertigoheel is thus suitable to a particular extent for treating brainstem degenerations with vertigo, nausea and tinnitus which are characterized by slowing down of the acoustic and vestibular brainstem regulating mechanisms. The syndrome of the slowed down brainstem is included in this to a particular extent.

References

(1) Berger, F., Handbuch der Drogenkunde, Vol. III, W. Mandrich-verlag, Wien, 1952.

(2) Claussen, C.-F., Der Schwindel und seine biologische behandlung mit Vertigoheel—Ergebnisse von klinischen experimentellen Untersuchungen, Biol. Med. 12, 6, 531-532 (1983).

(3) Claussen, C.-F., Bergmann, J., Bertora, G., Claussen, E., Clinical experimental testing and equilibriometric measurements to demonstrate the therapeutic effectiveness of a homeopathic remedy consisting of ambergris, cocculus, conium and petroleum in the diagnosis of vertigo and nausea, Drug Research 34 (II), 12, 1791-1798 (1984).

(4) Claussen, C.-F., Uber die Aufzeichnung und Auswertung ausgewahlter quantitativer Gleichgewichtsfunktionsprufungen, Habil. Schrift Berlin, 1970.

(5) Claussen, C.-F., Therapie bei Schwindel, Verhlg. der GNA, Vol. 4, Sammelband, Edition medicin & pharmacie, Hamburg, 1975.

(6) Claussen, C.-F., Uber den Schwindel, Verhlg. der GNA 1, 1-16 (1975)

(7) Claussen, C.-F., Luhmann M.v., Das Elektronystagmogramm und die neurootologische Kennliniendiagnostik, Edition medicin & pharmicie, Hamburg und Neu-Isenburg, 1976.

(8) Claussen, C.-F., Schwindel—Symptomatik, Diagnostik, Therapie, Ein Leitfaden, Edition medicin & pharmacie, Hamburg und Neu-Isenburg, 1981.

(9) Claussen, C.-F., Fort, E., Der Schwindelkranke, ein Patient vieler medizinischer Fachgebiete, Kopfklinium 1, 89-96 (1976).

(10) Claussen, C.-F., Luhmann, M.v., Aust, G., Erfahrungen mit einer neurootologischen Patientendatei, Ber. 18. Jahrestagg. d. Dtsch. Ges. f. Med. f. Dokumentation u. Statistik, pg. 149-152 (1976).

(11) Claussen, C.-F., Claussen, E., Objektive neurootologische Untersuchungen bei Vertigo und Tinnitus mittels Elektronystagmographie und akustisch evozierter Potentiale, Arch. f. Klin. exper. Ohr.-Nas.-Kehlk.-Heilk., currently in printing.

(12) Claussen, E., Claussen, C.-F., Der Schwindelpatient aus der Sicht der HNO-Facharztpraxis, Verhlg. d. GNA 6, 81-91 (1976).

(13) Claussen, C.-F., Presbyvertigo, Presbyataxie, Presbytinnitus, Springer-Verlag, Berlin, Heidelberg, New York, currently in printing.

(14) Dehnhardt, H.G., Das Symptom Schwindel bei inneren Erkrankungen, Verhlg. d. GNA 1, 363-374 (1975).

(15) Gessner, O., Gift- und Arzneipflanzen von Mitteleuropa, 3rd edition, Winther-Verlag, Heidelberg, 1974.

(16) Hoppe, H.A., Drogenkunde, Vol. 1, Verlag de Gruyter, Berlin, New York, 1975.

(17) Karsten, G., Weber, U., Stahl, E., Lehrbuch der Pharmakognosie, 9th edition, Gustav-Fischer-Verlag, Stuttgart, 1962.

(18) List, P.H., Horhammer, L., Hager's Handbuch der pharmazeutischen Praxis, vol. IV, Springer-Verlag, Berlin, Heidelberg, New York, 1973.

(19) Madaus, G., Lehrbuch der biologischen Heilmittel, vol. II, Verlag G. Olms, Hildesheim, New York, 1976.

(20) Mazger, J., Gesichtete homoopathische Arzneimittellehre, 4th edition, Haug-Verlag, Heidelberg, 1977.

(21) Rompp, H., Chemielexikon, 6th edition, Franck'sche Verlagsbuchandlung, Stuttgart, 1966.

(22) Rubin, W., Norris, C.H., Electronystagmography, Charles C. Thomas, Publishers, Springfield/Illinois, 1974.

(23) Spaich, W., Moderne Phytotherapie, Haug-Verlag, Heidelberg, 1977.

(24) Thomas, H., Handbuch der praktischen und wissenschaftlichen Pharmazie, Vol. IV, Verlag Urban & Schwarzenberg, Berlin, Wien, 1929.

(25) Wagner, H., Pharmazeutische Biologie, 2nd edition, Gustav-Fischer-Verlag, Stuttgart, New York, 1982.

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