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Subdividing Tinnitus into Bruits and Endogenous, Exogenous, and Other Forms

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Abstract: Tinnitus is an important complaint or disease or a combination, especially within our aging population. Neurootologists clinically deal with many different disorders of the human cranial senses, of which tinnitus is a very frequent type. With respect to neurootological clinical work, we delineate at least three groups of tinnitus. In *bruits*, patients complain of noise within the head (e.g., bubbling, hissing, pulsating); the bruit can be recorded as a physically existing sound in the human skull and is heard by patients. *Endogenous tinnitus* can be treated by external masking to suppress noise within the ears, whereas in *exogenous tinnitus*, patients seek to avoid any outside noise or sounds; they report that tinnitus decreases as soon as they go, for instance, into the cellar of a house or other soundproof place.

Key Words: audiometry; bruits; endogenous tinnitus; exogenous tinnitus; neurootology; sleep disorders; tinnitus

Tinnitus is a common complaint of patients in modern times. A 1984 epidemiological survey of the populations of Cardiff, Glasgow, Nottingham, and South Hampton reported that between 33.8 and 39% of affected persons answered positively to the general question, "Have you ever noticed noises in your head or ears?" [1]. The same study reported that moderate to severe annoyance of tinnitus is reported by 5.7–8.7% of the persons involved in this investigation.

According to studies of the U.S. American Tinnitus Association, approximately 36 million Americans suffer from tinnitus [2,3]. Currently, tinnitus is one of the most important symptoms in neurootology; others are vertigo, nausea, and hearing loss. A 1991 sample of 338 New Zealanders regularly experiencing tinnitus completed questionnaires posted to associations for people with tinnitus or hearing impairment. Nearly one-half of those sampled reported sometimes being depressed because of tinnitus. Those reporting depression and those reporting more severe problems as a consequence of their tinnitus saw more health care professionals and used more coping strategies. Most respondents did not remember exactly when they first noticed their tinnitus [4].

A 1993 questionnaire investigation of 1,091 patients at Bispebjerg Hospital, Copenhagen—"Tinnitus: Incidence and Handicap"—was conducted at a hearing center. A majority of patients (59%) claimed that they were troubled by tinnitus. Neither a greater degree of hearing loss nor a longer duration of tinnitus was shown to be associated with more severe tinnitus. Of patients with both subjective hearing loss and tinnitus, 23% stated that tinnitus was the greater problem, and 38% reported that their tinnitus and hearing loss were equally troublesome. Corresponding figures for patients with hearing impairment of such a degree that a hearing aid had been fitted were 9% and 41%, respectively. Such stress symptoms as headache, facial muscle tension, and sleep disturbances were correlated with tinnitus. Of patients with tinnitus, 83% were interested in obtaining treatment for their tinnitus [5].

The so-called Copenhagen Male Study reported on the results from a 10-year follow-up examination concerning hearing and factors known to cause hearing problems. The original sample consisted of 5,050 patients, and the present examination involved 3,387 (67%; median age, 63 years; range, 53–75 years). An increasing prevalence of 30–40% of hearing problems was

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demonstrated with increasing age. A prevalence of 17% of tinnitus of more than 5 minutes' duration was found; 3% indicated that their tinnitus was so annoying that it interfered with sleep, reading, or concentration. The prevalence of tinnitus increased up to the age of 70 and seemed to remain constant thereafter [5,6].

In Norway, 15% of the adult population has experienced shorter or longer periods of tinnitus. Three percent of these, in total some 7,000–10,000 persons, suffer from continuous tinnitus followed by symptoms that demonstrate handicap or occupational disability [7]. These observations have been similarly reported from many other countries by now.

GENERAL PHENOMENA OF TINNITUS

The term *tinnitus* has mixed meanings in that it is used to identify any noise without a human information function, which can mean both a normal and a diseased function of the human hearing. On one hand, tinnitus can also be regarded as a problem of acoustic resolution of the inner-ear microphone (i.e., the cochlear noise-to-signal ratio). In a well-dampened sound (proof) chamber, most normally hearing persons experience a sizzling sound in the ears, which is due to perceiving the molecular vibrations from the inner-ear fluids (as known in thermodynamics). Yet this underlying percept is masked in everyday life by regular environmental noise [8–10]. On the other hand, physicians are regularly confronted with tinnitus patients with problematic noises, which they describe as pulsating, humming, roaring, whistling, hissing, fullness of the ear, pressure, and pain in the ear.

Our ongoing study involves 823 tinnitus patients (77.52% male, 22.48% female; mean age, 50.87 years ± 8.68 years) from Bad Kissingen, Germany. All underwent clinical inpatient rehabilitation therapy of several weeks for a severe disabling tinnitus [11]. The subjective sensational qualities of their tinnitus are described in Table 1. All are still working. In these 823 patients, we have searched for descriptions of different time-intensity patterns of their tinnitus (Table 2). In this group with severe disabling tinnitus, we investigated the subjective background of discomfort in combination with other symptoms (as shown in Table 3). Additionally, the patients named the most irritating factors related to their tinnitus (Table 4).

Table 1. Subjective Classification of Ear Noises in 823 Tinnitus Patients

Complaint	Right Ear (%)	Left Ear (%)
Pulsating	1 (94)	1 (94)
Humming	7 (41)	6.93
Roaring	14.10	14.22
Whistling	50.67	51.76
Hissing	9.96	10.81
Pressure in the ear	6.32	5.83
Pain in the ear	14.10	14.22

Table 2. Subjective Classification of Different Time-Intensity Patterns of Tinnitus in 823 Tinnitus Patients

Time-Intensity Pattern	Percentage
Permanent	59.17
Intermittent	19.97
Fluctuating	43.26

Table 3. Classification of Subjective Background of Discomfort in 823 Tinnitus Patients

Subjective Complaint	Percentage
Headache	69.02
Migraine	4.13
Exhaustion	59.99
Lack of drive	42.16
Feeling of weakness	55.29
Forgetfulness	68.41
Disorientation	0.49
Daze	44.84
Tiredness	63.91
Insomnia	69.50

Table 4. Subjective Classification of the Most Irritating Factors Related to Tinnitus in 823 Tinnitus Patients

Factor	Percentage
All patients with specific additional factors	25.76
Difficulties in going to sleep	10.69
Difficulties in sleeping through	11.06
Depression	0.24
Abnormal sounds (also hallucinations)	2.67
Acute hearing loss	8.38

Sleep disturbances are a common and frequent complaint from tinnitus patients. Scientific studies have demonstrated decreased tolerance and increased discomfort accompanying tinnitus when insomnia and depression are associated with the disorder [8,12–14].

CLINICALLY DIFFERENT TYPES OF TINNITUS

Tinnitus is no longer an entity of a syndrome or a single disease. Owing to improvements in neurootometry, several different types of tinnitus can now be identified [9,15–18]. By means of modern audiometry, the frame for normal hearing can be clearly described objectively and quantitatively. Therefore, in any tinnitus case, a

thorough analysis of the hearing function and pathways needs to be performed, including threshold audiometry; audiometric tinnitus masking (if possible); acoustic dynamics between the measurable thresholds of hearing and acoustic discomfort; speech audiometry; otoacoustic emissions; acoustic brainstem evoked potentials; acoustic late evoked potentials, and the like. Likewise, also signs of pathology within the hearing pathways, between the ear and the human brain cortex, can now be measured [15,16,19–24].

Thus, we know from thorough neurootological studies that some 24% of disabling tinnitus has its source of irritation or dysfunction (or both) within the otoacoustic periphery (i.e., inner ear and cranial nerve VIII). Approximately 35% originate from the acoustic pathways within the brainstem, and nearly 41% originate within supratentorial structures or functions. These pathologies also should serve as basic information for deriving a systematic pharmacotherapy directed to the central nervous system focus of dysfunction. Only during the second half of the twentieth century did physicians develop the tools to differentiate objectively at least four different kinds of tinnitus (Fig. 1), which advance can provide physicians with a general orienting picture by a typical successive question-and-answer procedure.

Bruits

A typical medical question-and-answer procedure such as the following would provide a general orientation.

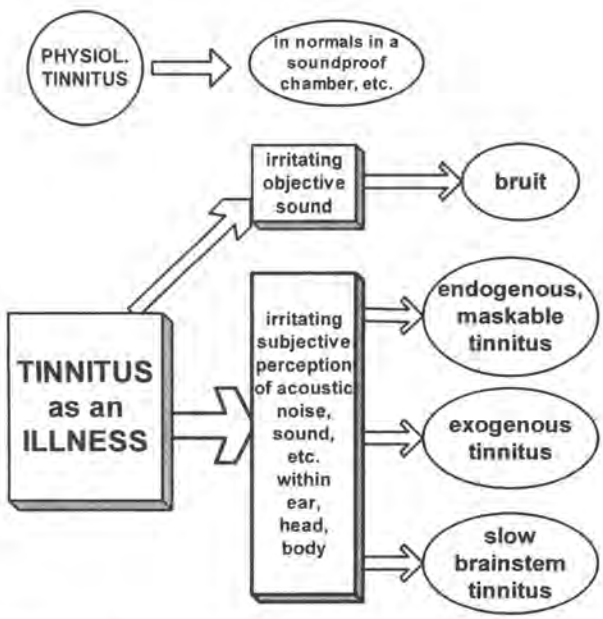

Figure 1. Categories of physiological and neurootological types of tinnitus.

Q: Have you been informed by somebody that he could hear a noise coming from your head?

A: Yes. His description listening from outside my head is similar to what I perceive.

By means of an auscultation through a stethoscope or a microphone, a genuine sound can then be derived objectively from a patient's skull. Patients frequently report a sound as bubbling, hissing, pulsating, and the like. The cause can be vascular in origin: abnormal curling of blood due to atheromas, vascular dissections, scars, compressions, high blood pressure amplitudes, and others.

Bruits also can originate from the middle ear and its connections toward the epipharynx: middle-ear inflammations with bubbling sounds of gas, whizzing middle-ear muscles, an open Eustachian tube, and the like. Cracking sounds, often misinterpreted as tinnitus, are reported from arthritic and other mandibular joint disorders. Additionally, sounds can be transferred from the cervical spine and its joints and from its vessels into the cranial structures, so that they become misinterpreted as tinnitus.

Endogenous Tinnitus

A typical medical question-and-answer routine can present a general orientation.

Q: Where is your feeling of well-being better: in a busy and noisy environment or in cave-like silence?

A: I much prefer a busy and noisy environment.

A patient with a maskable or endogenous tinnitus prefers covering up the tinnitus by external sounds. In using masking procedures, three zones of tinnitus can be distinguished easily according to audiometric measurements within the hearing field: low-tone tinnitus (at and below 750 Hz); middle-frequency tinnitus (1–2 kHz); and high-frequency tinnitus (>2–10 kHz or even 12 kHz).

Clinical pictures related to these three different audiometric types of endogenous tinnitus can be identified. Low-tone tinnitus is more frequently found in Ménière's disease and some other cochleoapical disorders, and middle-tone tinnitus more often is demonstrated in such diseases as otosclerosis. Most frequently, tinnitus is matched in the high-tone range and is related to noise trauma, whiplash, head and skull trauma, cardiovascular failures, stress, toxic events (including pharmaceuticals and nicotine or drug abuse), acoustic neuromas, and the like.

Also, several masking points may exist simultaneously. Dysfunctions of the inner ear contribute to the arousal of tinnitus, but tinnitus by itself depends on a

cortical process of the human brain. A sleeping patient does not suffer from any kind of tinnitus.

Since around 1985 [16,21,25–29], the Würzburg Neurootology Group of Claussen et al. has used vestibular evoked potentials (VestEP) and brain electrical activity mapping (BEAM) in major groups of patients suffering from an endogenous tinnitus. These researchers detected that such patients cortically respond in a typical, reproducible, and measurable manner: location of dwelling of the potentials around the upper gyrus of the temporal lobe (area Brodman 41); typical shortenings of the latencies of the evoked quantitative electroencephalographs (QEEGs); enlarged DC-shift of the evoked QEEGs; and typical cortical electrical burst expansion in three phases on the brain surface.

Since around 1990 [23,24,30], the New York Group of Shulman, Strashun, and Goldstein followed a neuroradiological path for deciphering the cortical modalities in tinnitus patients by using single-photon emission computed tomography (SPECT). In the temporal lobe of patients suffering from a maskable tinnitus, they discovered remarkably elevated metabolic processes.

Exogenous Tinnitus

Typical medical question-and-answer investigation can result in a general orientation here as well.

Q: Where is your feeling of well-being better: in a busy and noisy environment or in cave-like silence?

A: I much prefer a cave-like silence, as noise or a group of people speaking at the same time is most confusing. It provokes ringing and shrieking sounds within the ears.

Unlike those with endogenous tinnitus, patients suffering from exogenous tinnitus cannot benefit from masking noises in their surroundings. Some authors wrongly name this illness *hyperacusis*, but such patients do not hear better, as this term suggests. Seemingly better is the expression "syndrome of the hypersensitive ear" [31].

In exogenous tinnitus, pure-tone audiometry readings may be normal or exhibit regular deficits of the hearing threshold. However, no maskable tinnitus is present; yet, measuring the acoustic dynamics by adding the audiometrically recorded discomfort threshold demonstrates that the discomfort level, which usually lies between 1 and 8 kHz below 95 dB, rises below this level to values of 90–60 or even 50 dB. Persons being exposed to sound exceeding the level of their low discomfort threshold experience a loss of good understanding together with subjective pain and noise in the ears and accompanied by possible vegetative reactions.

Now hearing aids have been developed to such a level that they can adjust the incoming sounds by filtering, peak-clipping, cleaning of the sound signals, and other means so that they optimally fit into the remaining acoustic dynamics of the individually existing hearing field. Thus, hearing aids are the first choice in the toolbox for treating exogenous tinnitus. Some other methods for treating this type of tinnitus are physiotherapy, psychotherapy, stress reduction, and supportive pharmacotherapy [32,33].

Tinnitus in Slow Brainstem Syndrome

A generally orienting picture of tinnitus in slow brainstem syndrome can be elicited by a typical medical question-and-answer routine.

Q: How would you best describe your tinnitus?

A: With my growing age, I suffer from daze, disorientation, and a cloud of ringing and other sounds, which I cannot really localize in my ears or my head. But this flaw of noise disturbs me as much as my instability.

Among older patients, we regularly find those who complain about a hazy tinnitus in combination with vertigo, giddiness, and dizziness. They also report a reduced state of alertness and suffer from a connected statoacoustic problem.

Objectively, such patients exhibit an increase in the latencies of the experimentally provoked vestibular nystagmus and of the acoustically evoked brainstem potentials. This group of tinnitus patients is categorized under the label of "slow brainstem syndrome."

Especially in this group, we have noted in evaluating our therapeutic responses that a combination of *Cocculus indicus* (picrotoxin), *Conium* (coneine; hemlock), and Ambra and petrol oil (Vertigoheel) has a "tuning up" effect on the brainstem. As a result, the typical symptoms disappear [9,20].

Combined Endogenous and Exogenous Tinnitus

A combination of both types of subjective tinnitus (i.e., endogenous plus exogenous tinnitus) is also found among tinnitus patients [34]. Such patients report that their ringing, hissing, buzzing, humming, and other noises continue day and night but fluctuate. Especially the intensity of the noise can be increased radically when in a noisy environment, in a conversation with several participants, or in any other busy setting. Even though they show a maskable tinnitus, these patients report that therapeutic acoustic maskers do not reduce their suffering. Such patients need a thorough audiometric and neurootological workup.

This area of tinnitus disorders opens a frontier for treatment with pharmacotherapy, physiotherapy (including competitive kinesthetic interaction therapy), psychotherapy, therapy with instrumentations (e.g., hearing aids), electrotherapy, and other modalities.

NEUROOTOLOGICAL INVESTIGATIONS IN TINNITUS PATIENTS

In one of our Bad Kissingen retrospective studies, we examined 143 unselected tinnitus patients who came for a neurootological investigation. All were very cooperative. We tried to mask their tinnitus with a narrowband noise according to frequency and loudness intensity. However, of this total sample, only 69 patients (48.25%) were able to describe a noise by a similar sound profile in one or both ears masking their tinnitus. These cases then were classified into the category of endogenous tinnitus. In more than one-half of the sample (i.e., 51.74%), the tinnitus could not be masked.

These patients demonstrated that the personal disturbance due to their tinnitus can be decreased when exposed to silence or when using a comfortable hearing aid. This means that they are suffering from a so-called hypersensitive ear. These patients then were classified into the group of exogenous tinnitus.

With respect to the disease background, the cardiovascular disturbances (hypotension, hypertension, cardiac insufficiency) seemingly are more important than the metabolic diseases, such as diabetes mellitus, kidney disorders, or hyperlipidemia. The neurootological investigation included an entire network of functional tests in our neurootological office and laboratory at Bad Kissingen [8,9,34].

First, a thorough neurootological history is recorded by means of the neurootological anamnestic system Claussen (NOASC I). This scheme contains questions for general symptoms and specific vertigo and nausea symptoms; signs of tinnitus; hearing impairment; visual disturbances and other cranial nerve disturbances; a history of trauma to the head; orthopedic and neurological diseases; cardiovascular diseases; metabolic disorders (i.e., diabetes mellitus, nephropathia, etc.); and ear diseases and contains a setup of questions eliciting a self-categorization of success after treatment.

In the second step, patients are otologically inspected. The third step is a thorough audiometric investigation containing the measurements of the hearing threshold, the acoustic discomfort threshold, tinnitus masking, impedanciometry, and measurement of the stapedial muscle reflexes.

These tests are followed by objective measurements of the acoustic pathways by acoustic brainstem evoked potentials (ABEP), with 2,000 clicks at a sound level of approximately 60 dB above the hearing threshold, and acoustic late evoked potentials (ALEP), with 60-tone bursts of approximately 60 dB above the hearing threshold. With small-band noise, all patients are bilaterally examined for masking any tinnitus or tinnitus-like noise that they experience.

As the majority of the patients also complain of vertigo or nausea (or both), they undergo an extensive equilibriometric investigation with polygraphic electronystagmography and craniocorpography of the head and body movements. The electronystagmographically recorded tests contain vestibular ocular investigations with caloric stimulation and the evaluation according to the Claussen butterfly graph, the bilateral vestibular ocular per- and postrotatory test with the evaluation according to the Claussen rotatory intensity damping test (RIDT) scheme, and the retinoocular test with pendular ocular eye-tracking and monocular recording of the eye movements. The vestibular spinal recording of the craniocorpography is performed for the Romberg's standing test and for the Unterberger-Fukuda stepping test. Also, the equilibriometric findings form the basis for a referral to a neurootologist, who should complete a functional differential diagnosis and plan a lesion-oriented differential therapy.

At our Bad Kissingen neurootological research laboratory, we also use acoustically evoked responses (ABEP, ALEP) for objectively recording patient responses. Thus, we can systematically analyze the function at the hearing pathways from the ear throughout the posterior fossa by means of the ABEP. By means of our two-channel evoked response machine type ("BAD KISSINGEN"), we can easily gain both numerical and graphic data of the most varied tests on the same charts.

Finally, all the clinical data available are assembled in our central computer network ("CLAMEDEX"), which also acts as an expert system. Thus, we can quickly establish a differential diagnosis on the basis of our knowledge base from NODEC I–IV and other patient data analyses, followed by a systematically adapted differential therapy.

ENDOGENOUS VERSUS EXOGENOUS TINNITUS

For this study, we chose an unselected sample of 143 tinnitus patients who underwent a neurootological history recording and a series of neurootological hearing and equilibrium tests. All neurootological patients (see Table 1) underwent a thorough neurootological analysis. As shown in the Table 5, the sample was divided into two groups of patients with endogenous (i.e., maskable) and exogenous (i.e., unmaskable) tinnitus. The neurootological investigation followed the same procedure as was described earlier.

We systematically assembled and evaluated the data

Table 5. Basic Data of 143 Unselected Neurootological Tinnitus Patients

Sample	Number	Mean Age	± SD	Male	Female
Total group	143	47.9	14.9	79	64
Without maskable (exogenous) tinnitus	74	47.9	16.2	38	36
With maskable (endogenous) tinnitus	69	47.9	13.5	41	28

SD = standard deviation.

via the computer program Excel (Microsoft). The data we gained by this analysis are similar to those from an earlier study of 100 tinnitus patients, performed as a pilot study in 1984. Of our total sample of 143 patients, 74 (51.74%) complained of exogenous tinnitus (i.e., not maskable by means of narrow-band noise at any frequency compartment of the audiogram). At the time of the investigation, in 69 patients (48.25%), the tinnitus was maskable by means of audiometric methods (i.e., endogenous). Most of the following data, therefore, are represented comparatively for the total sample of tinnitus patients and for those patients with exogenous (unmaskable) and those with endogenous (audiometrically maskable) tinnitus.

The subjective general complaints for typical parameters (e.g., headache, drowsiness, and tiredness) demonstrate many more findings in patients with an exogenous tinnitus than in those with endogenous tinnitus (Table 6). For the signs of sleeplessness, anxiety, and partial depression, we find the opposite trend in the distribution, with more positive items among the patients with endogenous tinnitus. Vertigo and nausea symptoms were more frequently found in those with exogenous) tinnitus than among those with endogenous noise in

Table 6. General Subjective Complaints in 143 Neurootological Tinnitus Patients

Complaints	Percentage of All Patients (%)	Exogenous[a] Tinnitus (%)	Endogenous[b] Tinnitus (%)
Headache	58.0	68.9	46.4
Loss of efficiency	52.5	51.4	53.6
Fatigue	35.0	36.5	33.3
Weakness	30.1	31.1	29.0
Forgetfulness	36.4	36.5	36.3
Drowsiness	39.2	46.0	31.9
Tiredness	49.7	58.1	40.6
Sleeplessness	9.1	2.7	5.9
Anxiety	8.4	4.1	13.0
Depression	7.0	5.4	8.7

[a]Unmaskable (n = 74).
[b]Maskable (n = 69).
Note: Results according to the history scheme NOASC I.

Table 7. Vertigo and Nausea Symptoms in Unselected Neurootological Tinnitus Patients (percentages)

Complaints	Total Sample (n = 143)	Exogenous[a] Tinnitus (n = 74)	Endogenous[b] Tinnitus (n = 69)
All vertigo patients	79.0	85.1	72.5
Rocking	48.3	55.4	40.6
Lifting	7.7	9.5	5.8
Rotating	17.5	21.6	13.0
Falling tendency	25.2	31.1	18.8
Giddiness	44.8	54.1	34.8
Blackout	7.0	6.8	7.3
Instability	53.8	60.8	46.4
Claustrophobia	4.9	2.7	7.3
All nausea patients	50.3	60.8	39.2
Sweating	24.5	28.4	20.3
Palpitations	18.9	17.6	20.3
Malaise	34.3	41.9	26.1
Retching	4.9	6.8	2.9
Vomiting	4.2	6.8	1.5

[a]Unmaskable.
[b]Maskable.

one or the other ear (Table 7). The most frequently reported symptom was instability, followed by the rocking sensation and giddiness.

In our total sample, we found 2.7 vertigo complaints per patient. Among patients with the exogenous tinnitus, there were 2.8 complaints per patient, whereas those with the endogenous tinnitus accounted for 2.5 complaints per patient. The nausea symptoms were more equally distributed. In the total sample, we received 1.7 complaints per patient: 1.7 complaints per patient with exogenous tinnitus and 1.8 complaints in those with endogenous tinnitus. Clinically, the two samples demonstrate their differences, as the exogenous tinnitus patients exhibited more vertigo and nausea symptoms than did the endogenous tinnitus patients.

In recording patients' history, the investigator requested the patients to classify their noise according to pulsating, humming, whistling, or blissing, and with or without a fullness of the ear (Table 8). This table could

Table 8. Subjective Classification of Ear Noises in 143 Neurootological Tinnitus Patients (percentages)

Complaint	Total Sample (n = 143)	Exogenous[a] Tinnitus (n = 74)	Endogenous[b] Tinnitus (n = 69)
Pulsating	3.5	2.7	4.4
Humming	17.5	16.2	18.6
Whistling	30.8	27.0	34.8
Hissing	18.9	14.9	23.2
Fullness of the ear	16.9	25.7	7.3

[a]Unmaskable.
[b]Maskable.

easily lead to the conclusion that the most common type of tinnitus noise is the whistling, high-pitched sound. Astonishingly, the patients with exogenous tinnitus complained much more frequently about the fullness in the ear than did those with endogenous tinnitus.

Typical sound qualities of the tinnitus (humming, whistling, hissing) are found in 76.6% of the endogenous and in only 58.1% of the exogenous tinnitus patients.

The data concerning hearing deficits and status after ear operation and hearing prostheses (e.g., hearing aids) are shown in Table 9. There, the rates for hearing impairments are very similar for both the samples. The other subjective cranial nerve complaints (e.g., visual disturbances, trigeminal complaints, and facial nerve paralysis) are listed in Table 10. We also evaluated the disease background with respect to underlying disorders (e.g., cardiovascular diseases) as seen in Table 11.

The most important general diseases are the cardiovascular diseases. However, more than twice as many patients suffered from hypotension as from hypertension. Diabetes mellitus was more frequently found in patients with exogenous tinnitus, whereas hyperlipidemia was reported twice as often in patients with endogenous tinnitus.

The pure-tone hearing thresholds for air conduction showed only slightly worse results in patients with maskable tinnitus than among those with unmaskable ear noise (Table 12). Sixty-nine of the patients revealed a tinnitus that could be located or even masked by a narrow-band noise in either the right or the left ear. The mean frequency and loudness of the masking noise are reported in Table 13. Before performance of a masking test, 27 patients reported that their noise existed in the right ear, 46 that they had the tinnitus in the left ear, 57 complained of tinnitus in both ears, and 13 reported tinnitus diffusely distributed in the skull. Patients in

Table 9. Hearing-Related History in 143 Neurootological Tinnitus Patients (percentages)

Complaint	Total Sample (n = 143)	Exogenous[a] Tinnitus (n = 74)	Endogenous[b] Tinnitus (n = 69)
Hearing impairment	90.9	89.2	92.8
Deafness	2.1	1.4	2.9
Ear surgery	7.0	4.1	10.1
Hearing aid	3.5	5.4	1.5

[a]Unmaskable.
[b]Maskable.

Table 10. History of Other Subjective Cranial Nerve Disturbances in 143 Neurootological Tinnitus Patients (percentages)

Complaint	Total Sample (n = 143)	Exogenous[a] Tinnitus (n = 74)	Endogenous[b] Tinnitus (n = 69)
Visual disturbances	81.8	83.9	79.7
Trigeminal complaint	4.2	2.7	5.8
Facial nerve paralysis	2.1	2.7	2.0

[a]Unmaskable.
[b]Maskable.

Table 11. Disease Background of 143 Neurootological Tinnitus Patients (percentages)

Complaint	Total Sample (n = 143)	Exogenous[a] Tinnitus (n = 74)	Endogenous[b] Tinnitus (n = 69)
Cervical syndrome	29.4	29.7	29.0
Cardiovascular disease	49.7	52.7	46.4
Hypertension	11.9	12.2	11.6
Hypotension	25.9	24.3	27.5
Cardiac insufficiency	10.5	9.5	11.6
Diabetes mellitus	6.3	8.1	4.4
Nephropathia	2.8	1.4	4.4
Gastrointestinal disease	16.1	17.6	14.5
Viral infection	8.4	8.1	8.7
Hyperlipidemia	4.2	2.7	5.8

[a]Unmaskable.
[b]Maskable.

Table 12. Representative Mean Hearing Threshold in Decibels (Air Conduction) in 143 Neurootological Tinnitus Patients

Audiogram Test Frequency	Total Sample (n = 143)	Exogenous[a] Tinnitus (n = 74)	Endogenous[b] Tinnitus (n = 69)
Right ear			
500 cps	21.4 ± 18.6	20.1 ± 18.3	22.9 ± 18.4
1,000 cps	24.4 ± 18.8	23.5 ± 18.8	25.4 ± 18.9
4,000 cps	36.1 ± 25.3	35.0 ± 27.0	37.4 ± 23.4
Left ear			
500 cps	23.6 ± 17.0	22.4 ± 17.2	24.9 ± 16.9
1,000 cps	24.0 ± 15.6	23.2 ± 16.5	24.9 ± 14.9
4,000 cps	41.8 ± 25.5	38.5 ± 27.6	45.4 ± 22.7

[a]Unmaskable.
[b]Maskable.
Note: Data presented as mean plus or minus the standard deviation.

Table 13. Mean Frequency and Loudness of Maskable Tinnitus in 69 Neurootological Tinnitus Patients

Ear	Frequency (Hz)	Loudness (dB)
Right	3,403 ± 3,135	45.6 ± 21.4
Left	3,946 ± 3,302	51.1 ± 20.0

Note: Data presented as mean plus or minus the standard deviation.

the latter group could not relate the tinnitus to a particular ear.

The mean value of the masking frequency shows that it lies well above 1,000 Hz. However, this observation is relative, as the means come with a broad spread of the standard deviation. Most of the patients audiometrically demonstrated a high-tone tinnitus. By means of the neurootological investigation methods described earlier, 131 of our total sample of 143 patients underwent a differential equilibriometric investigation. Most frequently, central disequilibrium states or combined peripheral and central vestibular disorders were found (Table 14). The minimum ages of the patients in the various disequilibrium groups shows especially that several rather young patients complained of tinnitus. They had developed this disorder owing to a posttraumatic state.

The acoustic evoked potentials for the brainstem evoked and for the cortically evoked responses showed very little difference between the total sample and the patients with maskable or unmaskable tinnitus. However, the results were changed, with a significant differentiation between the groups concerning the interpeak latencies between waves I and V and waves III and V in patients with nonlocalizable vestibular lesions and those suffering from peripheral or combined peripheral and central vestibular disturbances. The acoustic late or cortically evoked potentials showed most variability in the absolute latencies of the contralateral acoustic pathways.

In pure-tone audiometry (see Table 12), patients with endogenous tinnitus exhibited a slightly deteriorated hearing threshold as compared to those with exogenous tinnitus. As at least the hearing receptor and the hearing nerve are bound into the common statoacoustic system, we also investigated the vestibulo-ocular and the vestibulospinal system by means of equilibriometric methods. On the basis of a long experience with equilibriometric tests, we classified 131 of 143 patients topodiagnostically. The most important groups, containing 93.9% of pathological findings of the equilibriometrically tested patients, are peripheral vestibular lesions (26.0%), combined peripheral and central lesions (32.1%), and central disequilibrium states (35.9%). This proportion indicates, with the typical acoustic pathway analysis due to evoked responses, that the majority of affected patients suffer from central neuro-otological degenerations.

Following the hypothesis that tinnitus may arise at the most varied parts of the hearing pathways between the receptor and the cortical end projection, our neuro-otological team could also prove cortical overactivity phenomena in the posterior temporal lobe by means of BEAM and VestEP [19,21,22,25,29,35]. Shulman et al. [23,24,30] demonstrated temporal lobe overactivity patterns by means of SPECT.

The tinnitus classification concept, including hypersensitive ears suffering from exogenous tinnitus, is important for the treatment design with loudness masking or avoidance of noise, both of which should to be selected according to the neurootometric findings.

CONCLUSIONS

Tinnitus presents with a endogenous disorder on one hand and an external sound-irritation-induced exogenous disorder on the other hand. Actually, much controversy or discrepancies concerning the nature, mechanisms, classification, management, or even definition of tinnitus exist among authors [2,3,7,13–15,17–19, 24,30,31,36–38]. Nevertheless, for some patients, tinnitus genuinely seems to be a problem that disturbs their daily activity, occupational ability, or even sleep.

Physicians have primary goals and responsibilities with respect to the management of tinnitus patients [2,8,9,11]. One method is exclusion by performing a complete neurootological examination of the disease, seeking to determine the cause of tinnitus. Another is to control (manage) tinnitus on the basis of exact topodiagnostics and classification. In both, modern neuro-otology should make an attempt to objectivize the subjective symptom of tinnitus. This special branch of neurootology also is called *tinnitology* [39].

Most of the audiological approaches for tinnitus studies are based on a psychometric principle; they require patients' cooperation and responsibility. Now,

Table 14. Equilibriometric Diagnosis Compared to Age Distribution in 131 Neurootological Tinnitus Patients

		Age		
Diagnosis	**No. (%)**	**Mean**	**Minimum**	**Maximum**
Normal	2 (1.5)	45	36	53
Lesions not to be localized	6 (4.6)	46	25	77
Peripheral vestibular lesion	34 (26.0)	46	12	86
Combined peripheral and central lesion	42 (32.1)	48	13	75
Central disequilibrium	47 (35.9)	50	17	84

clinical practice is confronting a need to develop a technology of such sensitivity as to detect gross or subtle changes. Tinnitology can differentiate between objective tinnitus (i.e., bruits) and subjective tinnitus involving the physiology of the systems being affected [31].

The term *tinnitus* does not describe a very concise entity of symptomatology. The term *bruit* stands for a physical objectively measurable noise created in the upper part of the body. *Tinnitus aurium* indicates a subjective experience of a noise that psychophysically seems to originate in the inner ears. This subjective noise can arise from any acoustic pathway disturbance. The latter, for instance, occurs most peripherally after a noise trauma to the ear, more centrally after a vertebrobasilar insufficiency afflicting the hearing pathways in the brainstem, or cortically as an aura of an epileptic seizure. Psychogenic causes for tinnitus also are discussed, although exogenous tinnitus depends on the dysregulation of the acoustic dynamics toward the incoming acoustic signals from the outer world.

Among our neurootological patients, we frequently find multisensory syndromes (e.g., with combinations of tinnitus, hearing impairment, vertigo, and nausea). One hundred forty-three randomly selected neurootological patients suffering from severe tinnitus underwent a complex neurosensory investigation, including neurootological history; classic audiometry; acoustic brainstem evoked potentials; acoustic cortically evoked potentials; visually evoked potentials; electronystagmography of spontaneous, caloric, rotatory, and optokinetic nystagmus; and craniocorpography of vestibular spine.

The statistical results demonstrate that tinnitus is bound to a multifactorial disease background. Topodiagnostic analysis of the statoacoustic data shows the existence of significantly more central than peripheral pathology [2,24,29–31,39].

As hearing is based on transmission of physical sounds from the surrounding world through the resonating space of the outer ear and the physical amplifier of the middle ear and as hearing finally is related to biological data transmission in the hearing pathways from the cochlea toward the temporal lobe of the human brain, our modeling of good hearing (i.e., understandable hearing) and bad hearing (i.e., nonunderstandable hearing, ear noise, or tinnitus) mainly is performed in dimensions of physics and modern data technology. However, the human spirit, with all the sensory capacities attached to it, does not live in a technically designed house nor in a computer but in a chemically constructed and biologically functioning body. Thus, many dysfunctions—even of the central nervous system—can also be provoked on a biochemical basis (e.g., through intoxications by solvents or through so-called untoward side effects of many kinds of drug therapy) [26,34].

Tinnitus and hypoacusis, complaints often expressed by neurootological patients, frequently are accompanied by vertigo and dizziness. Very frequently, these combined signs form the basis for a referral to a neurootologist, who then completes a functional differential diagnosis and plans a lesion-oriented differential therapy [2,3,8,9].

Combining sensorimotor tests with suprathreshold evoked responses of the acoustical type permits not only discrimination of peripheral inner-ear lesions of the cochlear and the vestibular type but differentiation of central pathology on the different levels within the map of the central statoacoustical system [8,15,16,18,19,22,33].

This, then, is a gross indication for an endogenous tinnitus. However, if tinnitus patients report that the opposite is the case—the tinnitus is provoked by environmental noise (traffic, partying, etc.) and that it improves in relative silence (e.g., at home, in a cellar), it should be named *exogenous tinnitus* [11,31]. Many patients maintain that the personal disturbance due to their tinnitus can be decreased when they are exposed to external noise (i.e., masking) [31].

For several decades, we have observed some attempts to classify the general entity known as *tinnitus* into subgroups. On our way to a better tinnitus classification, we take the following standpoint: At the least, the complaint of tinnitus classically has to be differentiated into bruits, tinnitus aurium, and tinnitus cranii sive cerebri (or also the "syndrome of the hypersensitive ear") [15,22,31].

The term *bruit* stands for a physically objectively measurable noise, created in the upper part of the body, that can be listened to by an external investigator through a stethoscope, for instance. *Tinnitus aurium* indicates a subjective experience of a noise, which seemingly originates in the inner ears of patients. However, this noise cannot be heard by an external investigator (e.g., by the use of a microphone or a stethoscope). A subjective noise that the patient cannot localize in one or both ears but is somewhere in the head, though still not objectively measurable, is called *tinnitus cranii sive cerebri*. The latter occurs also as an aura of an epileptic seizure. The classic literature [8,9] also discusses psychogenic causes for *tinnitus cranii sive cerebri*.

During the last decade, we have further developed the systematics of tinnitus classification. Besides the aforementioned bruits, we now differentiate between endogenous tinnitus and exogenous tinnitus and other special forms [22,24,31]. Among our tinnitus patients, we regularly find a group who report that their tinnitus subjectively can be localized either in the right or the left ear or in both ears. They further report that they feel relieved to a certain extent when they remain in a noisy environment. These patients (mostly clinically) also demonstrate that their tinnitus can be masked with audiometry using a narrow-band noise, which they then describe as equiv-

alent to their subjective noise according to frequency and loudness intensity. With respect to the special clinical picture, we name such tinnitus cases *endogenous tinnitus*. Patients with endogenous tinnitus now constitute approximately 48% of all the tinnitus cases we investigate.

Another group of our tinnitus patients states that personal disturbances due to their tinnitus can be decreased when they are exposed to maximum silence (e.g., in a cellar or when using a comfortable and shielding hearing aid). In some 52% of our tinnitus patients, we can prove that the tinnitus could not be masked. For those patients, the noise existed somewhere diffusely distributed inside their heads. When exposed to too much noise in their surroundings or at a party with many people talking at once, they feel more or less greatly disturbed by their tinnitus. Therefore, they try to escape from such external volume.

When audiometrically measuring the hearing threshold and the threshold of discomfort, we regularly find a shrinking of the distance between the two, which we describe as a reduction of the audiometric dynamics or hearing capacity. We call this type of tinnitus the *syndrome of the hypersensitive ear* [9,20,31].

Other special groups have been discovered in our sophisticated neurootometric laboratories, where we submit our patients to a network analysis of the statoacoustic pathways. Besides audiometry, we also apply vestibulometry. In a first example, we discovered that some of our tinnitus patients show normal maximum activities of the caloric responses; however, the nystagmus culmination latency was too greatly delayed. When in such patients with tinnitus we also performed ABEP, we found an increase in the latencies of wave V. We called this the *syndrome of the slow brainstem*. A significant number of these patients, mostly older patients, when undergoing pharmacological therapy with Conium and *Cocculus* together with Ambra and petrol oil (Vertigoheel), lost the undiscerned tinnitus in their heads. Usually, this syndrome is presented in combination with vertigo and giddiness, which also come be controlled with this therapy [2,9,20].

Generally speaking, no tinnitus occurs in a sleeping or unconscious patient. With respect to our most recent investigations, we know that the cortex of the human brain, especially the superior gyrus of the temporal lobe, plays a most important role for the release of tinnitus [2,3,9,16]. However, tinnitus can be triggered throughout the entire distance of the acoustic pathways from the cochlea in the inner ear, following cranial nerve VIII into the brainstem toward the acoustic nuclei, following further through the olivary nuclei, passing the inferior geniculate body running through the thalamus and the basal ganglia toward the cortical projections in the temporal lobe [2,8,9,15,16,22,24,31,32].

Because we have the possibility of functionally diagnosing disturbances at various levels of the statoacoustic pathways (e.g., by applying the caloric butterfly test and the vestibular stimulus response intensity comparison and by adding the perrotatory nystagmus characteristics to the characteristics of the Claussen butterfly chart), we can discriminate the height of the irritating focus between the inner ear, the eighth nerve, the brainstem, and the temporal lobe functionally.

Using the combination of ABEPs and acoustic cortically evoked potentials and the neurootometric functional localization scheme, we come to the conclusion that in approximately 24% of all tinnitus cases, the irritating focus is located in the acoustic periphery (i.e., inner ear or eighth nerve or both). In some 35%, the irritating focus leads to tinnitus within the central structures of the hearing pathways in the posterior fossa. For the remaining 41% of patients, we estimate that in objectively measurable functional irritations in the upper brain above the posterior fossa, psychogenic, iatrogenic, or pharmacological causes provide the main influence on the existence of tinnitus.

Until now, tinnitus has been understood to be a common complaint, as with headache, pain, fever, and the like. Clinicians should be aware of the fact that patients may have tried at least one treatment before entering an audiology clinic. Controlled studies are needed to avoid the spread of nonspecific and ineffective treatments in managing tinnitus.

Tinnitus cannot yet be cured routinely. Affected patients, however, need help to avoid countless ineffective treatments and considerable costs. Therefore, we have to split the general clinical diagnosis of tinnitus into subgroups for a better understanding of the underlying pathological mechanisms that may be curable by different approaches and methods.

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