MARJA ESTOLA-PARTANEN

Muscular Tension and Tinnitus

An Experimental Trial of Trigger Point Injections on Tinnitus

University of Tampere
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ACADEMIC DISSERTATION
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ACADEMIC DISSERTATION

To be presented, with the permission of
the Faculty of Medicine of the University of Tampere,
for public discussion in the main auditorium of Building K,
Medical School of the University of Tampere,
Teiskontie 35, Tampere, on December 8th, 2000, at 12 o'clock.

University of Tampere
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A. LIST OF ABBREVIATIONS

ABI = Auditory Brainstem Implant
ABR = Auditory Brainstem Response
ADP = Adenosine-Di-Phosphate
AICA = Anterior Inferior Cerebellar Artery
AMPA = α-Amino-3-Hydroxy-5-Methyl-4-Isoxazone-Proprionic Acid
ATP = Adenosine-Tri-Phosphate
AVCN = Antero-Ventral Cochlear Nucleus
BAEP = Brainstem Auditory Evoked Responses
BERA = Brainstem Evoked Response Audiometry
BIH = Benign Intracranial Hypertension
BSER = Brainstem Evoked Responses
CAP = Compoud Action Potentials
CBF = Cochlear Blood Flow
CGRP = Calcitonin Gene-Related Peptide
CEOAE = Click Evoked Otoacoustic Emissions
CMD = Craniomandibular Disorders
CN = Cochlear Nucleus
CNS = Central Nervous System
CNV = Contingent Negative Variation
CT = Computer Tomography
DCN = Dorsal Cochlear Nucleus
2DG = 2-Deoxyglucose
DPOAE = Distortion-Product Otoacoustic Emissions
EMG = Electromyography
ENG = Electroneuronography
ENT = Ear, Nose and Throat
GABA = Gamma Amino Butyric Acid
HL = Hearing Level
IC = Inferior Colliculus
IHC = Inner Hair Cells
LSO = Lateral Superior Olive
MGB = Medial Geniculate Body
MML = Minimum Masking Level
MOC = Medial Olive-Cochlear
MRI = Magnetic Resonance Imaging
MSN = Medullary Somatosensory Nuclei
MSO = Medial Superior Olive
MVEP = Myogenic Vestibular Evoked Potentials
NIHL = Noise-Induced Hearing Loss
NMDA = Non-N-Methyl-D-Aspartate
OAEs = Otoacoustic Emissions

OHC = Outer Hair Cells
PET = Position Emission Tomography
PST = Prolonged Spontaneous Tinnitus
PTM = Portable Tinnitus Masking equipment
PVCN = Posterioventral Cochlear Nucleus
RI = Residual Inhibition
SL = Sensation Level
SOAEs = Spontaneous Otoacoustic Emissions
SOC = Superior Olivary Complex
STSS = Subjective Tinnitus Severity Scale
TEOAE = Transiently Evoked Otoacoustic Emissions
T1 = Tinnitus 1 (Loudest tinnitus sound)
T2 = Tinnitus 2 (Second loudest tinnitus sound)
T3 = Tinnitus 3 (Third loudest tinnitus sound)
T4 = Tinnitus 4 (Fourth loudest tinnitus sound)
TMD = Temporomandibular Disorder
TMJ = Temporomandibular Joint
TNS = Transcutaneous Nerve Stimulation
TTS = Temporary Threshold Shift
VAS = Visual Analogy Scale

B. INTRODUCTION

Tinnitus is a perception of sound without an actual external acoustic stimulus. It is considered a symptom and not a disease itself.

The etiology of tinnitus has not been solved. Many different aspects considering the phenomenon are known but the mechanism leading to tinnitus is not understood and a valid animal model is lacking. The outer hair cells of cochlea are the prime candidates for tinnitus generation. The neurotransmitters of the auditory pathways, extralemniscal projections and spontaneous otoacoustic emissions (SOAEs) may be intimately connected with the generation of prolonged tinnitus. Tinnitus can also be produced by external causes like general diseases, drugs and above all by noise. However none of the presented factors is capable of explaining all tinnitus types or causing tinnitus to everyone.

Although various attempts and treatments have been tried to alleviate tinnitus no universal cure is available. It appears that several mechanisms leading to tinnitus generation probably exist and thus different ways of treating tinnitus might be justified. Drugs or operations are suitable only for the minority of tinnitus cases. The main effort of treatment is directed to coping with the situation. Self help groups gather to support the sufferers by sharing the experience with others. Professional experts can relieve the anxiety of the situation by proper counselling. The main effort is to relieve stress either physically (muscular) or psychologically.

For many years trigger point injection techniques have been accomplished to treat muscular pain. The reference pain zone from the trigger point can be situated far from the point itself, but is characteristic to that point every time. In a routine trigger point treatment for muscular otalgia, a patient suddenly mentioned that the injections influenced also her tinnitus and tinnitus vanished. In the present study 178 tinnitus patients were treated with lidocain trigger point injections. The results of the study are presented and reasons analyzed.

C. REVIEW OF LITERATURE

1. DEFINITION OF TINNITUS

In history several attempts have been made to classify tinnitus. Celsius in book De Medicina in 1st century AD classified it by the supposed etiology as did Gilbertus Anglicus in the 14th century. Duverney classified it in 1683 according to supposed place of generation in other words tinnitus in the ear and tinnitus in the brain. J. M. Itard classified tinnitus already in 1821 quite the same way than it is often classified today. He divided it into true tinnitus including vascular and tubal sounds and false tinnitus, which he considered could be the product of irritated auditory nerve, generated after a noise trauma or be symptomatic in hysterical and hypocondral personalities. The third type was fantastic tinnitus,which he considered to reflect instable personality and psychical diseases. (Stephens1987).

According to Stephens (1987) the English word tinnitus can be found in the Oxford English Dictionary 1963. He also mentioned that in the second edition of Blanchard´s Physicians´Dictionary the term Tinnitus aurium was defined as " a certain buzzing or tingling in the ears ". The stem word is the Latin tinnire, to ring. The term tinnitus is anyhow not limited to ringing sensations but includes all the false auditory sensations in spite of the character.

Dauman et al (1992) classified tinnitus into normal (or non-pathological) and pathological (or abnormal) tinnitus. Normal tinnitus occurs usually without hearing loss. It lasts less than five minutes and is perceived less than once a week. Normal tinnitus is usually monaural and tonal, generally single pitch high- frequency tone. Pathological tinnitus correlates often with hearing loss. Its duration is more than five minutes and it occurs more frequently than once a week or it is heard continuously.

Tinnitus can be temporal or permanent. Temporal tinnitus appears once and does not usually return. If it returns repeatedly it may be called intermittent. Permanent tinnitus is heard continuously (Dauman 1992).

Tinnitus can be classified by the supposed site of dysfunction. One of them is the middle ear. The other site of dysfunction is the auditory pathway medial to the middle ear and this type of tinnitus is called sensorineural. It could further be divided into peripheral, central or combination of both of them. In peripheral type the site of dysfunction is the cochlea but the sound is detected by central nervous system (CNS). In central tinnitus the cochlea is considered normal and the site of dysfunction is supposed to be the supratentorial brain structure or in the brainstem with some kind of abnormal neural activity. The third possibility is that both cochlea and CNS are involved in the formation of tinnitus (Dauman et al 1992). Zenner et al (1993) divided cochlear tinnitus into three types. Type I is cochlear-motor tinnitus and is usually acompanied by sensorineural hearing loss, positive recruitment and caused by pathological increase of the movement of outer hair cells (OHC). Type II is transduction tinnitus which the authors assumed might be caused by pathological change of the receptor potential of the inner hair cells (IHC) leading to a release of transmitters. Type

III is signal-transfer tinnitus which is caused by biochemical signalling cascades in auditory sensory cells.

Coles (1996) classified tinnitus to five main types; physiological, spontaneous otoacoustic emissions (SOAEs), temporary dysfunctional, pathological and pseudo-tinnitus. Physiological types contained hums (muscular or vascular) and snaps (mucular). Temporary dysfunctional tinnitus contained noise-induced, drug-induced and toxaemic tinnitus. Pseudo-tinnitus contained environmental and feigned tinnitus. The largest group was pathological tinnitus which was further divided into 1) extra-auditory tinnitus (containing muscular, respiratory and vascular subtypes), 2) conductive tinnitus , 3) sensorineural tinnitus (containing sensory, peripheral neural and central neural subtypes), 4) associated tinnitus (subtypes cervical and temporomandibular) and 4) psychological tinnitus (containing hallucinatory and imaginery subtypes).

In psychological classifications the emphasis is placed on the reactions that tinnitus creates in the patient. Tinnitus is called acceptable or unacceptable depending how much it is disturbing the patient (Dauman et al 1992). Several studies have classified tinnitus depending on the psychological reactions it causes to the individual (Lindberg et al 1984, Klockhoff et al 1967, Halford et al 1992, Goldstein et al 1996).

2. CHARACTERIZATION OF TINNITUS

2.1. Characterization by patient description

The tinnitus sound itself can be quite different in different patients. Stouffer et al (1990) asked 528 tinnitus patients to describe their tinnitus with the closest word. The most popular description was ringing, mentioned by 41.3 % of males and 32.8 % of females. Van den Abbeele et al (1992) studied 79 patients and 43 % of them described their tinnitus to be close to a whistle. Ringing was the most popular, 19 % of all, in a questional study by Tyler (1983) (table1). In a Finnish study by Kotti (1997) about 50 % of 175 tinnitus patients had high ringing tinnitus, next prevalent was whistling. A strange type of tinnitus called "explosive tinnitus" has been described by Teixido et al (1998). Tinnitus is characterized by "crashing" or "banging" noise occuring in, before or during sleep. 60 cases have been presented in the literature, the reason is unknown.

Table 1

DESCRIPTION OF TINNITUS

	Stouffer		Van den Abbeele	Tyler
	Males	Females		
Ringing	41.3%	32.8%		19 %
Buzzing	7.8%	15.3%		
Cricket	1.5%	9.8%		
Hissing	9.2%	6.0%	14 %	
Whistling	4.4%	3.4%	43 %	14 %
Humming	4.1%	6.8%	6 %	
Roaring	4.4%	4.7%		
Musical note	5.1%	3.0%		
Steam whistle	4.4%	3.4%		
Pulsing	2.0%	6.0%		
Rushing	2.0%	3.0%		13 %
Cracking	0.3%	2.6%		
Sizzing			6 %	
Other	<1%	<1%		
Several sounds			9 %	

2.2. Characterization with audiometer

The characterization of tinnitus can also be done with an audiometer by matching the tinnitus sound to the audiometric sounds and trying to find the closest replica of it. The three most popular audiometric ways to characterize tinnitus are pitch-matching, loudness matching and masking abilities of the tinnitus sound.

2.2.1. Pitch

When measuring the pitch, the frequency of a pure tone is adjusted to the pitch of tinnitus. If several tinnitus sounds occur, the most prominent of them is picked or several sounds are measured separately. Occasionally tinnitus is not a pure tone but more or less a narrowband noise, then it can be measured by the character of the most prominent offered pitch. Pitch-matching is sometimes difficult, because all the offered pitches can differ too much from the patient´s tinnitus. In pitch matching subjects should recognize the sound which seems to be like their tinnitus. If another sound, one octave above or below the first one is offered, the listener might find it to be identical with the first one. This is called octave confusion. Pitch-matching without comparing to the higher or lower octave might distort the results (Vernon 1987). Tyler (1992) reported problems with octave confusions and fluctuations in the pitch. 28 % of his patients had sudden changes and 21 % gradual changes in the pitch of tinnitus.

In Oregon 1033 people complaining about tinnitus were studied by Meikle et al (1992). Pitch was higher than 3000 Hz in most cases. 1500 Hz and lower was measured only from 8.2 % of the attendants, 1500- 3499 Hz from 16.9 %, 3500- 6499 Hz from 32.8 %, 6500- 8499 Hz from 23.8 % and higher than it from 18.2 %. Other studies also confirm the same observations, tinnitus is usually measured between 4000- 8000 Hz (Lutman 1993). However Trassera et al (1996) measured 53 tinnitus patients and recorded pitch levels in the middle tones with a peak around 3 kHz.

Wahlsröm et al (1996) asked the patients to describe tinnitus with their own words and then the pitch was measured. The most common word for 250 Hz tinnitus was rushing either in a narrow band noise or in a pure tone. In 4000 Hz pitch the description was a tone or hissing for a pure sound and whistling, whining or whelding for a narrow band noise. In 8000 Hz pitch the desriptions for a pure tone were rushing, beeping or ringing and for a narrow band noise rushing, ringing or beeping. Also non-tinnitus subjects were asked to describe both narrow band noises and pure tones given with the same three frequencies. Boat or fog-horn were the two most common descriptions for 250 Hz pure tone, rushing or workshop for narrow band noise. With a 4000 Hz pure tone sound beeping was the most common, with narrow band noise running water. If 8000 Hz was given as pure tone the most common comment was beeping but with a narrow band noise the top three were compressed air, waterfall and rain. A great overlapping seemed to exist considering the measurements and descriptions in both non-tinnitus listners and tinnitus patients. For instance the term wind was mentioned by 4 patients with a measured pitch 8000 Hz and 5 patients with measured pitch 250 Hz.

2.2.2. Loudness

Tinnitus loudness measurements with an audiometer can be done at the pitch frequency or at 1000 Hz frequency. Vernon (1987) used a starting tone of 1000 Hz and increased it gradually until the patient regarded it to be of identical loudness with tinnitus. The same procedure was then done with other frequencies including the pitch frequency. The sensation level (SL) of tinnitus, meaning the level of tinnitus above the hearing threshold level at the pitch frequency, was quite low, usually only 5-10 dB. The sensation level at 1000 Hz frequency was often found to be higher. In a study 79 % of the patients had tinnitus loudness of 6 dB or less using SL at the tinnitus frequency (Vernon 1987). In a survey of 1800 patients 51 % regarded their tinnitus to be 0-3 dB SL, 28 % matched it to 4-6 dB SL and 8 % to 7- 8 dB SL. A total of 87 % of the patients had their tinnitus loudness matched below 10 dB SL (Vernon et al 1984). Trassera et al (1996) investigated 53 tinnitus patients and mostly their tinnitus was measured between 5 dB and 10 dB.

Tinnitus can be also expressed with exact values recorded with an audiometer without comparing the matched tinnitus level at the pitch frequency to hearing theshold level, this is called tinnitus hearing level (HL). HL values are naturally greater than SL values. Tinnitus figures diverge also according to the ear used. Ishikawa et al (1992) compared measurement ears in unilateral tinnitus. When ipsilateral ear was used the mean value of tinnitus was 49 dB HL (range 2 to 93 dB), but in the contralateral ear the mean value of the same tinnitus was 40 dB HL (range 6 to 85 dB). Matsuhira et al (1996) studied the factors contributing to tinnitus loudness. The most important correlating factor was hearing threshold level in the ear with tinnitus (positive correlation), pitch matched frequency (tinnitus matched to 4000 Hz was sensed softer) and age (negative correlation).

2.2.3. Masking abilities

Minimum Masking Level (MML) is the smallest tone at the pitch frequency needed to mask tinnitus. First the pitch of tinnitus is determined and then the tone is gradually increased until the patient can no longer hear tinnitus. Masking can also be achieved with broadband noise. Tyler (1992) considered pure-tone masking to be frequency specific in normal-hearing listeners. If masking could be achieved with a pure tone by simply increasing the tone level of the masker, it was regarded as a sign of peripheral effect. When masking was frequency independent or tinnitus could not be masked at all tinnitus was considered to be of central origin.

Sometimes after a successful masking tinnitus disappears for a while when the masking sound is stopped. This is a phenomenon called Residual Inhibition (RI). Spaulding reported it in 1903 and Josephson in 1931 (Meikle 1984). Meikle et al (1984) tested 1800 people and 91 % of them had experienced some kind of RI.

2.3. Characterization with VAS-scale

Because tinnitus is mostly a subjective experience it can also be measured by the visual analogy scale (VAS). By using it the psychological effects can also be monitorized. The VAS-scale in pitch rating goes from 1 to 10, 1 meaning very low frequency and 10 extremely high. Stouffer et al (1990) used the VAS-scale in a survey and the average tinnitus pitch was 7.1 in a 1 to 10 VAS-scale. Also in a study by Bebear et al (1992) the great majority of individuals reported their tinnitus to be 4 or over it in the VAS-scale.

VAS has been widely used to express tinnitus loudness in question studies, because it gives possibilities to record more subjects than audiometric matchings. Stouffer et al (1990) asked 528 tinnitus patients to rate their tinnitus loudness in the VAS-scale from 1 to 10, 1 meaning soft and 10 very intense tinnitus and the average was 6.1 by male and 6.5 by female patients. More people considered their tinnitus to be loud rather than soft, 34 % reported values 8 or more and only 20 % 3 or less.

Lindberg et al (1992) asked 6 patients to measure the loudness of their tinnitus constantly with a portable tinnitus masking equipment (PTM). The device contained an analogue noise generator and a built-in clock and it was designed for different types of tinnitus matchings. The patients used the VAS-scale at the same time and the recordings of these two were compared. The correlations between VAS and PTM were significant ($p < 0.5$) in five subjects but one had recordings without significant covariation between the values.

2.4. Inconvenience caused by the tinnitus

Characterization of tinnitus can be done according to the patient´s attitude to tinnitus. Two tinnitus sounds with the same frequency and loudness can cause completely different psychological reactions in two different individuals. The questions of the psychological characters of tinnitus deal with the personal reaction and disability. Klockhoff et al (1967) published a subjective severity grading and it divides tinnitus into three grades. Grade 1 tinnitus is intermittent and might be occasionally annoying and disturb sleep. Grade 2 tinnitus is constant but does not bother very much at daytime. In quiet situations and at concentration it is felt annoying and may also prevent the patient from falling asleep. Grade 3 tinnitus is constant and the patient is aware of it constantly. Tinnitus might wake the individual up at night and it interferes with his ability to fall asleep. Halford et al (1992)

proposed Subjective Tinnitus Severity Scale (STSS), which comprises 16 items with answers yes/no. The questions are dealing with matters like how prominent, intrusive and distressing tinnitus is. By counting the answers the degree of the distress to the patient can be formed. Goldstein et al (1996) published a Tinnitus Stress Test in order to better understand all the subjective annoyance and depression caused by tinnitus. Lindberg et al (1984) devided tinnitus into three grades. I. Audible only in silent environment II. Audible at low environmental noise levels, masked by surrounding noise, does not disturb sleep but might make it difficult to fall asleep III. Audible at all environmental noise levels, makes it difficult to fall asleep, can disturb sleep, constantly dominating problem which can affect the patient's entire life.

2.5. Objective measurements of tinnitus

Several attempts have been made to measure tinnitus objectively but no proven method of measurement has been found. Objective detection of tinnitus has been studied by the evaluation of auditory-evoked magnetic fields, especially some objective methods have been based on the evaluation ratio of M100 and M200 waves (Hoke et al 1989). The authors noticed that in tinnitus patients the M200 was delayed and poorly developed or even missing. The amplitude ratio of M200 and M100 could distinguish tinnitus patients, they all had the ratio less than 0.5 and controls had a ratio above it. The method did not however separate the tinnitus patients from normal subjects in a work by Jacobson et al (1992) nor in the work of Colding-Jorgensen et al (1992). Kristeva et al (1992) found a weaker auditory magnetic field in tinnitus patients compared to the non-tinnitus group but only in the M200 component the difference was statistically significant. The method was greatly dependent on the recording position. Shiomi et al (1997) recorded an auditory-evoked magnetic field before and during tinnitus remission induced by intravenous lidocain. In tinnitus patients the N100 m peak became sharper during tinnitus remission but in the control patients no such change during lidocain infusion was experienced.

Jastreboff et al (1992) recorded auditory evoked brainstem potentials from normal hearing subjects with and without tinnitus and found a difference in the potentials recorded from tinnitus patients compared to normal subjects. More variability of the potentials was found if tinnitus was present. The main difference between the normal and tinnitus patients was observed around 7 ms which, according to the authors, reflected the fact that tinnitus is processed at the level of inferior colliculus or higher. Martin et al (1996) recorded spontaneous activity in the auditory nerve during neurosurgical operations and found spectral peak near 200 Hz in 12 of 14 subjects. Out of the 12 subjects 11 had tinnitus. 200 Hz peak could not be found with recording from pons, brainstem, cerebellum or muscle flap. Because a peak at 200 Hz frequency has been reported from cats and rabbits with asetosalicylate induced tinnitus and has not been reported in normal subjects, the authors considered that the 200 Hz peak might be an objective sign of tinnitus. The 200 Hz peak has not been reported in all types of tinnitus. For instance middle ear pathology might decrease its existence.

Mitchell et al (1996) noticed that distortion-product otoacoustic emissions (DPOAE) in tinnitus patients showed larger amplitudes than in controls below 1000 Hz or over 3000 Hz region, in mid-frequency region no difference could be detected. Attias et al (1996) reported a relationship between noise induced tinnitus and efferent neural auditory activity. 42 individuals with or without tinnitus and with or without noise induced hearing loss (NIHL) were studied with contralateral white noise stimulation and the effect on transient otoacoustic emissions (TOAEs) was recorded. For non-tinnitus controls, an increase of contralateral white noise intensity reduced TOAE amplitude regardless of the coexistence of

NIHL. Tinnitus patients responded with increased TOAE at lower stimulus intensity regardless of the existence of NIHL. The authors regarded it as a possible objective sign of tinnitus. Lockwood et al (1998) have noticed some differencies in the brain-mapping of tinnitus patients and controls with PET. Patients with tinnitus showed more widespread activation by the tones and they also had aberrant links between the limbic and auditory system.

3. EPIDEMIOLOGY OF TINNITUS

3.1. Prevalence of tinnitus in population studies

In Britain a large multi-purpose population study about hearing and its disorders was organized by Coles et al in MRC Institute of Hearing Research (Coles 1984). Over 23 000 letters were mailed to ordinary inhabitants of some cities and the answers were analyzed. 19 139 answers out of 23 000 were received and 767 subjects were interviewed by phone. Tinnitus was defined as spontaneous sound lasting more than five minutes and the prevalence rate of tinnitus was 15.5-16.8 % . Tinnitus severe enough to interfere sleep was experienced by 8 % of the population and extreme severe tinnitus limiting the ability to lead a normal life by 0.5 %. In the USA in a general population survey 32 % of the population had observed tinnitus and it was severe in 2 % (Stouffer et al 1990). In Sweden a random sample of 1 % of the inhabitants in the city of Gothenburg, age between 20 and 80 years, was questioned (Axelsson et al 1989). 66 % returned the letter and 14.2 % of the subjects suffered from tinnitus constantly or often. In 2.4 % tinnitus was so severe that it plagued all day. In British Columbia about 30 000 industrial workers working in noise of 85 dB or more for 8 hours, were asked about their tinnitus in a rutine health examination (Chung et al 1984). The prevalence of tinnitus was 6.6 % although it was expected to be a lot higher concidering to the circumstances.

3.2. Prevalence among children

The prevalence of tinnitus was 64% in partially hearing children, 12 to 18 years. Tinnitus was mostly intermittent, caused reluctance to wear hearing aid and problems in concentration (Graham 1981). The prevalence of tinnitus was 66 % in hearing-impaired and 29 % in deaf children, it was mostly intermittent (Graham et al 1987). Tinnitus varied from 6 % to 36 % in normal hearing children in different studies. In hearing-impaired children the prevalence varied from 24 % to 77 % (Stouffer et al 1992). 6-13 % of 140 normal-hearing and 24-29 % of 21 hearing-impaired children had tinnitus (Stouffer et al 1992). Gabriels (1996) investigated 628 patients with tinnitus, only 21 were under 18 years. Significant causes of tinnitus in children were ear infections and noise, especially loud music. Tinnitus caused disturbances in concentration and sleep and sensitivity to sound.

3.3. Prevalence among aged

In a study by Rosenhall et al (1991) 674 people, age 70 years, were questioned about their tinnitus. Half of the subjects were followed longitudinally at ages of 75 and 79 years. The prevalence of people with continious tinnitus in different age groups varied between 8-15 % , 20-42 % had occasional tinnitus. Rubinstein et al (1992) studied tinnitus prevalence among people over 70 years and found it to increase from 28 % in the 75 years old to 42 % in the 79 years old. Vernon et al (1996) studied the tinnitus character among the elderly (over 65 years of age). The pitch was lower, the loudness smaller, tinnitus easily maskable and residual

inhibition longer than in younger age groups. The prevalence was not studied but the duration of tinnitus was less than a year for over 20 % of the subjects.

3.4. Factors affecting the prevalence of tinnitus

As in children, hearing level seemed to be the most important co-factory explaining the prevalence of tinnitus in a study with 30000 workers. The age, shooting- or smoking history was studied and neither of them explained solely the prevalence of tinnitus, the hearing level de facto explained the outcome (Chung et al 1984). Tinnitus was more common with a co-existent hearing loss ($p<0.01$). When tinnitus occurred frequently, hearing was considered normal in 28 % of the cases and slightly impaired in 58 %, 12 % had marked hearing loss and 1 % was deaf. With constant tinnitus hearing was considered normal in 19% of the cases, slightly impaired in 55 %, markedly impaired in 19 % and 3 % were deaf (Axelsson et al 1989).

3.5. Site of tinnitus

Chung et al (1984) found no difference in the prevalence of tinnitus between the right and left ear. In many studies differences between the ears have existed, usually more tinnitus has been reported in the left ear. Stouffer et al (1990) subclassified the tinnitus into various locations in the head. The most frequent was left ear tinnitus, 21.4 % of the total, followed by both ears equally, 20.3 % and right ear tinnitus, 16.3 %. When all the subgroups were considered the left side dominated by 40.7 % compared to 34.6 % in the right side. In the study by Axelsson et al (1989) the site of tinnitus in 337 patients was more common in the left ear by females (62 %) and males (68 %). According to the authors it might reflect the hearing loss, which in some investigations seemed to be more common in the left ear. In a Japanese survey by Ishikawa et al (1992) with 80 patients the affected side was also more frequently the left (53.7 %) compared to the right (46.3 %). In the USA Meikle et al (1992) investigated 1033 patients in a tinnitus clinic. The generality sequence of the location of their tinnitus was left worse (42.3 %), right worse (34.6 %), equal in both sides (16.5 %) and the head (6.6 %).

Hazell (1981) considered that the predominance of the left-sided tinnitus might reflect the etiological factors relating to the dominance of one hemisphere. In a study of MRC Hearing Institute (1981) the possibility that the tinnitus side could be related to the side of handedness was considered, but no such correlation was observed. The fact that tinnitus is predominantly left-sided, has been considered to be a sign of physiological asymmetry of the lower brainstem in humans (Stouffer et al 1990). Right-sided hearing loss seemed to be associated more with tinnitus sensed in the head and left-sided hearing loss with tinnitus sensed in the left ear (Axelsson 1989).

3.6. Course of tinnitus

The course of tinnitus is variable and depends on the etiology of tinnitus. The severity of tinnitus often increases with time. In the study by Stouffer et al (1990) 60 % of the patients had experienced no changes in their tinnitus ever since it started, but in 34 % it had increased and in 7 % it had decreased. Prognosis was more favourable if the onset was acute, because in those instances only 10 % had an increase in loudness and 20 % had a decrease or tinnitus vanished. When the onset was gradual 20 % experienced an increase and 10 % a decrease or tinnitus vanished (Coles et al 1990).

3.7. Tinnitus related disorders

Hyperacusis, feeling of pressure or fullness of the ear, diplacusis and subjective hearing loss often accompany tinnitus. In hyperacusis ordinary sounds cause pain to the ear involved. Jastreboff et al (1993) considered hyperacusis to be a pretinnitus state. The supposed mechanism of it is increased central gain as a consequence of the increase of sensitivity of neurons at the subcortical level. Gordon (1997) described s.c.Hear (hyperactive ear) syndrome. He listed several other disorders than tinnitus into the disease complexas: audiosensitivity (sharp noise intolerance), hyperacusis, deafness, fullness of ear, dizziness, ataxia, pitch distortion, circadian variation, insomnia, depersonalization, neurasthenia, nausea and vomiting, fear and paranoia and finally susceptibility to many drugs and motion sickness.

4. POSSIBLE ETIOLOGIES OF TINNITUS

4.1. General diseases

Hesse et al (1996) noticed sleep disturbances in most of their tinnitus patients, mostly oxygen desaturation (29.1%) and sleep apnea (15.6%). Large amount of desaturation periods could be looked upon as a risk factor for chronic tinnitus. Derebery et al (1996) noticed that 40% of the patients coming to allergy testing complained about tinnitus. In 135 patients with Ménière syndrome and allergy, the treatment of allergy decreased also statistically the tinnitus severity. Kraft et al (1996) measured the fasting insuline levels and found hyperinsulinemia, with or without hyperglycemia, to be a major metabolic marker related to subjective idiopathic tinnitus. The authors found also early hypertension in tinnitus patients.

Depression and frustration was found in 36 % of the tinnitus patients and 35 % of them complained about stress or inability to relax (Tyler et al 1983). In a study by Stephens et al (1992) 14 % of the tinnitus patients were frustrated or depressed and 19 % irritated, stressed or had inability to relax. Meric et al (1996) investigated 281 tinnitus sufferers, half of them were found to have some psychic disorder. The two most common were hysteria and psychoastenia (26 % of each), followed by hypochondrias (21 %) and schizophrenia (20 %). Lewis et al (1992) described six case histories with tinnitus patients, five of them had committed suicide and one was killed by his son. Five of them were socially isolated and also five of them had visited a psychiatrist before entering the tinnitus clinic. Tinnitus was mostly in the left ear (4) and mostly of pulsatile character (4 cases out of 6). Lewis et al (1994) published a retrospective study of 28 tinnitus patients who had committed suicide mostly because of tinnitus. 10 individuals had a mental disease prior to the outbreak of tinnitus (2 schizophrenias, 5 major depressions, 2 anxiety states and one with hysterical fits). Tinnitus occured more often in the left ear than in the right ear (36% versus 16%) but mostly it was bilateral (40%). The character of tinnitus was mostly whistling (29%) followed by hissing (25%). Weiss (1984) mentioned the possibility of auditory hallucinations in alcoholic delirium, mostly they were transient but might become chronic.

4.2. Ear diseases

Axelsson (1992) refered 308 tinnitus patients with some kind of ear disease, 278 of them had sensorineural hearing loss. The most prominent group (117 individuals) was noise induced hearing loss (NIHL), meaning over one third of all the ear causes. Morbus Ménière was diagnosed from 11 % of the patients, sudden deafness from 5 %, presbyacusis from 5.5 % and acoustic trauma from 6 %. Conductive hearing loss was detected from nearly 10 % of the patients. The co-existence of hearing loss and tinnitus has been presented in other studies (Axelsson 1989, Stouffer 1990, Chung 1984). Dauman et al (1992) divided the ear diseases creating tinnitus into middle ear and sensorineural causes. The middle ear causes are disorders that produce sounds themselves like venous hums and arteriovenous fistulas, muscular problems as stapedius and palatal myoclonus, patulous Eustachian tube, temporomandibular and cervical disorders among others. Sensorineural tinnitus may be peripheral, central or a combination of them. In peripheral sensorineural tinnitus Dauman et al (1992) considered that cochlea or the eighth nerve are abnormal and they bring the normal central nervous system (CNS) to produce tinnitus. Vascular disorders, noise exposure, aging, drugs, otosclerosis affecting the cochlea and autoimmune disorders are possible candidates for such reactions. In the other sensorineural type Dauman et al (1992) considered the cochlea normal tinnitus originating from abnormal neural activity in the brain or brainstem. This might be a result from vascular disorders, tumours or degenerative processes of unknown reasons. Dauman et al (1992) considered that in many cases both the cochlea and the CNS might be involved in tinnitus generation, for instance in ototoxity, aging and noise exposure.

4.3. Possible role of craniomandibular disorders

Rubinstein et al (1992) studied 2659 individuals aged 70, 75 and 79 and found the prevalence of tinnitus to be from 28 % to 42 % except for the youngest age group where it was 19 %. Subjects with tinnitus had also significantly higher prevalence of craniomandibular disorders (CMD) (facial and cervical pain, ear pain, dysphagia, restriction of jaw movements and clicking sounds of the temporomandibular joint (TMJ)). Bruxism was also significantly more common in the tinnitus group. Chole et al (1992) found tinnitus to be significantly more prevalent among group of 338 patients with temporomandibular disorder (TMD) compared to 694 controls. Kempf et al (1993) examined the TMJ and dentognathological system of 138 patients with an inner ear disease, 13.8% of them had tinnitus. In the examination 79.7 % of them had pathological findings (44 % had TMJ disorder, 29 % parafunction of the occlusion and 35 % myopathy of the masticatory muscles). He recommended dentognathological examination for patients with inner ear dysfunctions of unknown etiology. Lusk et al (1985) published a case report of a trigeminal neuralgia patient who experienced a vibrating and roaring tinnitus while chewing. The problems started after repeated percutaneous radiofrequency destruction of the gasserian ganglion. The authors assumed that it could be explained by the ephaptic conduction of the nerves innervating the masticatory muscles and the tensor tympani muscle after trauma to the gasserian ganglion. Thus the tensor tympani muscle would contract in companion with the masticatory muscles causing tinnitus.

Bjorne (1991) found out in 39 patients with disabling idiopathic tinnitus that the lateral pterygoid muscle ipsilaterally to tinnitus was sore on palpation. He injected 1.8 ml of 2% lidocain into the muscle and tinnitus was relieved. The response varied from 20 % to 100 % but tinnitus reappeared as the anesthetic wore off. Tinnitus with fullness of ears, tinnitus

composed of two or more different sounds, tinnitus with gradual onset and hardly maskable tinnitus might be connected with TMD according to Vernon et al (1992).

4.4. Cervical disorders

Krausová et al (1968) treated patients with cervico-cranial disorders by manipulation and monitored the results with repeated audiological tests. The audiometric recordings were improved in 6 out of 23 patients. They considered that the results might be explained by the function of the deep neck reflexes. The typical clinical picture of cochlear disturbances in vertebro-basilar insufficiency was sensorineural hearing loss or complete unilateral deafness mostly combined with tinnitus (Decher 1975). Brügel et al reported in 1991 that hearing loss at low frequencies and tinnitus occured with pathological conditions of cervical spine. In several occasions tinnitus and hearing loss were reported to have started after the treatment in the cervical region and that the treatment had most likely triggered the disorders.

Hülse discovered in 1994 that some tinnitus cases might originate from the cervical disorders. He considered vertebrobasilar insufficiency as one cause for hearing disorders. Another cause was "vertebragenic hearing disorder" where the cervical disorders were accompanied by tinnitus, pressure in the ear, otalgia and hearing loss. He reviewed 259 patients with well-defined functional disorders of the upper cervical spine combined with symptoms of cervical vertigo. 15 % of them had subjective hearing disorders. Audiometric threshold shifts of 5-25 dB were observed in 40 %, mostly at low frequencies. The click-evoked otoacoustic responses were absent in spite of nearly normal hearing. The symptoms were reversible in some, and after chiropractic manipulation 5 of the patients had a measurable improvement in their hearing. The author reported that there was often a functional pathological finding in palpation in the neck ipsilateral to the hearing loss. Hülse considered that proprioseptive and nosiseptive afferents proceed from the cervical area C2 to nucleus cochlearis. A link from the cervical nerve C2 to nucleus cochlearis was reported by Pfaller et al (1988). Connections from the spinal dorsal column nuclei to the nucleus cochlearis and the connections from nucleus cuneatus to nucleus cochlearis has been demonstrated in cat by Itoh et al (1987). Connections from the lower cervical level dorsal ganglions to nucleus cuneatus had been proven to occur by Arvidsson et al (1990). Biesinger (1998) described several cases of tinnitus caused by cervical disorders, he considered the afferents from cervical vertebra to be the cause.

4.5. Drugs

Several drugs are known to cause tinnitus. Anticancer drugs, nitrogen mustard, cisplatin and methotrexate may produce tinnitus (Brown et al 1981). Aminoglycoside antibiotics kanamycin (and its congeners amicasin and tobramycin), gentamicin, neomycin and streptomycin (and its congener dihydrostreptomycin) can cause both hearing loss and tinnitus (Brown et al 1981). Vancomycin (a glycopeptide antibiotic), amphotericin B (a polyene antibiotic), erythromycin (a macrolide antibiotic) and chloroquine (4-hydroxyquinoline) are also possible causes of both hearing impairment and tinnitus. Sulphonamide, clindamycin, tetracyclines (doxycycline and minocycline) and metronidazole can cause solitary tinnitus (Brown et al 1981). Loop diuretics (ethacrynic acid, furosemide and bumetanide) can cause tinnitus and contemporary hearing loss, clopamide tinnitus without hearing loss (Brown et al 1981).

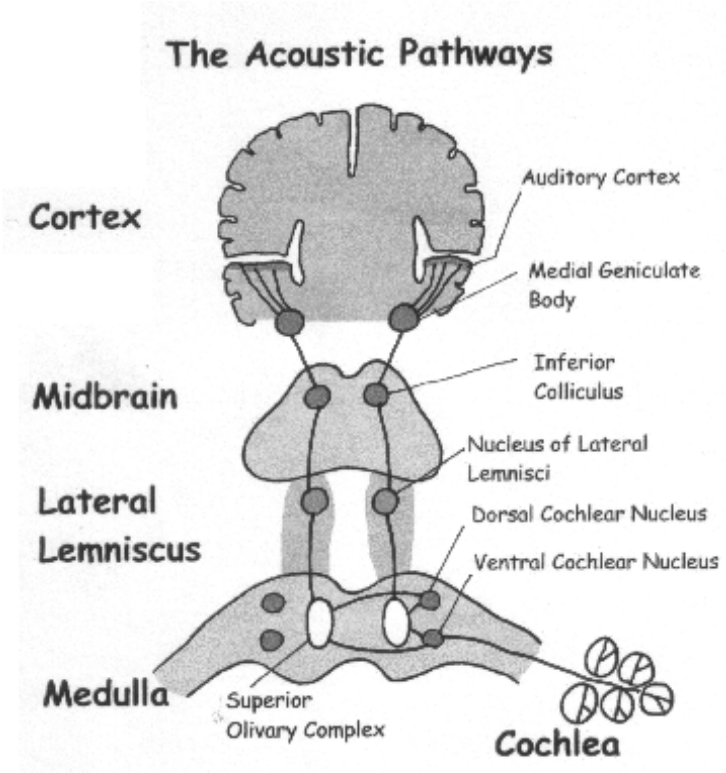
Salicylates especially acetosalicylic acid can cause tinnitus and hearing loss (Brown et al 1981). The coincidence with salicylates and tinnitus is so obvious that acetosalicylic acid is used as tinnitogenic agent for animal studies (Jastreboff 1992, Eggermont 1992, Kellerhals

1992). The serum salicylate level to produce tinnitus had to be over 19.6mg/100ml, average was 30.4mg/100ml, in a study by Mongan et al (1973). The tinnitus producing dose varied greatly (from 3600mg buffered asetosalicylic acid to 4800mg) in a group of 59 normal hearing people with rheumatoid arthritis. Other analgesics causing tinnitus with hearing loss are propionic acid derivatives (ibuprofen, naproxen and ketoprofen), pyrazolon derivatives (phenylbutazone and oxyphenbutazone), indole derivatives (indomethacin and sulindac) and fenamates (flufenamic acid and mefenamic acid) (Brown et al 1981). Narcotic analgesic propoxyphene can affect both hearing and tinnitus, two other narcotic analgesics, morphine and pentazocine cause tinnitus without hearing impairment (Brown et al 1981).

Cinchona alkaloids (quinine and quinine) might cause both tinnitus and hearing loss, as well as medroxyprogestine (oral contraceptive steroid) and propylthiouracil (antithyroid drug) (Brown et al 1981). Anticonvulsant carbamazepine has been tried in the treatment of tinnitus, but it can cause tinnitus itself (Brown et al 1981). Local anaesthetic lidocain can be used as anticonvulsant agent and it has anti-tinnitus effects but can cause tinnitus like two other local anaesthetics, mepivacaine and bupivacaine (Brown et al 1981). Drugs affecting biogenic amines can also produce tinnitus. The best known groups are antischizophrenic drugs (haloperidole), lithium, tricyclic antidepressants (imipramine, desipramine, doxepin, amitriptyline, nortriptyline, protriptyline, trimipramine), monoamine oxidase inhibitor antidepressants, H1 receptor blocking antihistamines (diphenhydramine, brompheniramine, cycline, promethazine and trimeprazine), drugs affecting the beta-adrenergic receptor (propranolol, practolol and pronetalol) and miscellaneous (levodopa) (Brown et al 1981).

Tinnitus without a hearing defect has been described after CNS stimulants (aminophylline and caffeine), vasodilators (verapamil), drugs affecting serum cholesterol (cholestyramine resin), steroids (prednisolone), organic solvents (methyl alcohol and bentzene), convulsant (penicillamine) and miscellaneous drugs (folic acid, phenformin and aminocaproic acid) (Brown et al 1981).

Fig. 1 The acoustic pathways



5. AUDITORY PATHWAYS AND TINNITUS

5.1. Normal functions of the auditory pathways

5.1.1. Anatomical considerations

Békésy described in 1953, based on cadaver studies, a model according to the movements of the basilar membrane, the so called travelling wave model . The basilar membrane gets a maximal response near the point of resonance. The amplitude of the upward and downward motion of the basilar membrane will reduce gradually towards the base and rapidly towards the apex creating an asymmethrical response (Wilson 1987). Johnstone suggested that this mechanism was not only passive one but the outer hair cells (OHC) assisted sharpening up the basilar membrane response (Johnstone 1992). Bohnke et al (1999) created a three-dimensional mechanical model of cochlea and their results fit with the early obseravations of Békésy (1928) about the waves in the inner ear.

The outer hair cells have stereocilias, which are embedded to the tectorial membrane. When the basilar membrane is displaced it bends the stereocilia against the tectorial membrane. The OHC are motile so inner hair cells (IHC) are activated through the tectorial membrane (Wilson 1987). On the top of the stereocilias are gates which make the exhange of K+ -ions possible. The flickering open and close of the gates makes electrical currents and influences for the gain processes of the acoustical energy (Johnstone 1992). OHC are cylindrical and change their axial stiffness as their membrane potiential is altered. Stiffness is decreased with depolarization and increased by hyperpolarization (He et al 1999). The efferent innervation of cochlea passes to both IHC and OHC, it reaches the IHCs via the spiral bundle and branches to several OHCs across the tunnel of Corti. The main efferent pathway to the cochlea goes through the crossed olivo-cochlear bundle (Wilson 1987).

The main auditory pathway is contralateral. The main pathway projects from the cochlea to the antero-ventral cochlear nucleus (AVCN), by trapezoid body to the contralateral superior olivary complex (SOC) and to lateral lemniscus and from there to cortex. Another pathway projects from cochlea via the posterioventral cochlear nucleus (PVCN) and dorsal cochlear nucleus (DCN) to the contralateral lateral lemniscus and the nucleus of the lateral lemniscus. The main nuclei upward are inferior colliculus (IC) and medial geniculate body (MGB). The ipsilateral pathway traverses AVCN, SOC, IC, MGB and terminates to the temporal cortex (Wilson 1987, Netter 1974). The cochlea has neural connections to the ipsilateral trigeminal ganglion which is the origin of basilar artery innervation. Thus the cochlea might have some importance in the CBF regulation by primary sensory innervation (Vass et al 1997).

In addition to the classical ascending auditory pathway the extralemniscal system also exists. The central nucleus of IC projects onto the MGB but the afferents of the external nucleus of IC project onto the association areas of the cortex. The primary auditory cortex and the contralateral IC are also connected to the dorsal cortex of IC, which in turn has projections onto the so called thalamo-cortical auditory system (Moller 1992). In addition the association areas of the cortex receive information from the somatosensory system and auditory information from the lateral lemniscus. The extralemniscal system does not mediate the sharp auditory sensations but more broad tuning sensations (Moller 1992). The central auditory system constructs a single sensation of the two separate informations coming from both inner ears. The integration starts in the CN, where the auditory information is refined by several control mechanisms through inhibition (Romand 1992).

5.1.2. Neurotransmitters in the auditory pathways

The main neurotransmitters of the non-auditory nerves in the cochlear nucleus (CN) are gamma amino butyric acid (GABA), glycine and acetylcholine (Romand 1992). The neurotransmitter in the auditory nerve is an excitatory amino acid , possibly glutamic acid. Catecholamine projections have been shown to exist in the CN, their main origin is the locus coeruleus. Serotoninergic fibres exist in the dorsal raphe nucleus of the CN. In addition enkephalin terminals and opiate receptors have been shown to exist in the CN. Other peptides which might affect the neurotransmission have also been found in the CN; somatostatin, substance P and vasopressin (Romand 1992).

In the superior olivary complex the main neurotransmitter is glutamate or a related excitatory amino acid. It acts through non-N-methyl-D-aspartate (NMDA) receptors. In the superior olive several other transmitters are also present and they may be involved in complex circuits. The principal cells of the lateral superior olive (LSO) and medial superior olive (MSO) receive indirect input from the contralateral ventral CN (Romand 1992).

NMDA- receptors have been found in the inferior colliculus (IC). From the superior olivary complex some glycinergic and some GABAergic connections project onto the IC. The dorsal nucleus of the lateral lemniscus sends GABAergic projections onto IC and GABA also seems to be the most important intrinsic neurotransmitter in IC. These GABAergic neurons probably act by sharping the output of IC neurons as their response to acoustical stimulation. The afferent projections from the IC might use an excitatory amino acid or other transmitters for instance asetylcholine as a neurotransmitter. They terminate to the medial geniculate body (MGB) , where NMDAL-(3H)glutamate-binding sites have also been found. Thus NMDA receptors might be part of the neurotransmission. In addition GABA, dopamine, serotonin, glycine, substance P-like, adrenergic cells have been found in the MGB (Romand 1992).

Afferent neurotransmissions from the MGB to the auditory cortex are supposedly mediated by an excitatory amino acid. The exact nature of the main afferent transmitter is not known. The most likely candidate seems to be glutamate. In the auditory cortex extrathalamic projections, which are cholinergic, noradrenergic, serotoninergic, and dopaminergic have been detected, in addition GABAergic cells and substance P terminals. Acetylcholine can influence the physiological functioning of cortical neurons and thus their means of processing of sensory information (Romand 1992).

5.2. Pathology in the auditory pathways and cochlea in tinnitus

5.2.1. The possible cochlear mechanisms

Kemp (1981) expressed the role of hair cells in tinnitus generation; abnormal spontaneous firing of hair cells could activate the associated auditory nerve. Bredberg et al (1972) studied normal and pathological cochleas with scanning electronmicroscopy and noticed partial decoupling of the pathological cilias of hair cells from the tectorial membrane. Tonndorf (1981) believed that a tight cilial coupling was of extreme importance in the normal function of the cochlea (Wilson et al 1981). Wilson speculated that a loose cilial coupling might rise the noise level by as much as 55 dB, which could be the basis of cochlear tinnitus (Wilson et al 1981). Evans et al (1981) listed the cochlear theories of tinnitus generation as follows: abnormal movements in the perilymp, hypersensitivity of the corda tympani, hair cell standing current flow increase after static loosening of cilias, uncoupling of the hair cell cilias from the tectorial membrane, abnormal coupling of the tectorial membrane to the hair cells, increase or decrease of the blood flow to the auditory system, rhythmic hyperactivity in the afferent- efferent auditory pathways resulting in a "reflex arc" formation.

Graham et al (1996) investigated the contralateral suppression of TEOAEs with broadband noise in normal individuals and tinnitus patients. The tinnitus patients had only unilateral tinnitus. In the 1-2 kHz region a significant difference was found between the tinnitus patients and the controls with 50 dB SL, in the tinnitus group less suppression of TEOAEs was found. This difference was detected only in the tinnitus ear and not in the non- tinnitus ear of the same patients. The authors concluded that this effect might point to the fact that efferent system might be contributing to the detection of tinnitus related activity that they assumed is originating in the cochlea.

Mitchell et al (1996) investigated distortion-product otoacustic emissions (DPOAEs) with tinnitus patients and controls and found them in half of the tinnitus group. The DPOAEs were found to be identical in the groups in the mid-frequency region (1000 Hz to 3000 Hz) but below 1000 Hz or over 3000 Hz DPOAEs produced by the tinnitus group had larger amplitudes than those produced by the controls. Also the DPOAE input-output functions at 1000 Hz were larger in the tinnitus patients than in the controls. These findigs suggest, according to the authors, a damage of OHC and perhaps detachment of the tectorial membrane without IHC or nerve damage.

5.2.2. The OHC- efferent innervation theories

On the top of the OHC stereocilias are gates, which make the exhange of K+- ions possible. Loud noises block the gates causing a temporal threshold shift. The same can be achieved by the use of ototoxic drugs which may be important in tinnitus generation (Johnstone 1992). Johnstone (1992) speculates that efferent system might also be involved in tinnitus generation. Medial system innervates the OHC and it might also send collaterals to the cochlear nucleus. If OHC function is upset by the loss of hair cells, efferent system is activating. Normally the activation of the efferent system increases hearing threshold and that might be compensated by the collaterals causing an increase of sensitivity in the cochlear nucleus. If the OHC gain is working improperly the threshold does not change, but the sensitivity of the cochlear nucleus still increases creating imbalanced output to the higher centers, which may be sensed as tinnitus. Also Veuillet et al (1992) considered that medial efferent system tries to sharp up the cochlear acoustical sensitivity in case of a high frequency hearing loss causing tinnitus.

Chéry-Croze et al (1993) investigated the effectiveness of the medial olivo-cochlear efferent system (MOC) by the use of otoacoustic emissions. An alteration of the function of MOC could be found in 19 of the 20 unilateral tinnitus patients and in 16 patients with bilateral tinnitus. When tinnitus was present an alteration of MOC functioning was always found in one ear at least and at the precise frequency of tinnitus. The authors consider this result as a lack of effectiveness of the efferent system.

5.2.3. The neurotransmitter imbalance

The generation of tinnitus may be caused by abnormal patterns of activity in the auditory nerve. A modification of the auditory nerve activity, for instance after noise induced hearing loss, increases the neural excitability of the IC (Romand 1992). The IC has a position as a rely station for the ascending and descending pathways, it is a station to connect the inputs from the nucleus of the lateral lemniscus, the superior olivary complex and the CN. If the balance between excitatory and inhibitory pathways is disrupted at the CN level, the activity may change at the IC level. Disturbances in the activity of the auditory neurons using the inhibitory neurotransmitters may lead to changes in the spontaneous activity (Romand 1992).

Glutamic acid is one of the neurotransmitters of the cochlea, it acts between the hair cells and afferent nerve fibres. It has also been detected in the cochlear nucleus, acting between the primary afferent nerve and cochlear nucleus (Murai 1992). Imbalance of input from the periphery might increase the release of glutamate. Some excitatory amino acids are known to destroy GABAergic and cholinergic cell bodies, glutamate might be able to do it as well. Glutamate might also operate via agonist-operated calcium channels leading to an excessive influx of calcium. Calcium causes damage to dendrites and to somatic regions rich of glutamate receptors, resulting in cell death (Romand 1992). Loss of neurons and the fiber connectivity in aging leads to changes in neuropeptide and transmitter levels. Because the neuropeptides and transmitters are often co-localized with one another, the degeneration of neurons affects also modulating and recognition mechanisms in several cortical areas. If there are local hypoxic ischemia attacks in the cortical regions it might lead to excessive release of excitatory amino acids like glutamate and aspartate. The calcium channels that are gated by excitatory amino acids, may then have an excess of calcium intake leading to cell death (Romand 1992).

Jastreboff et al (1992) studied the importance of calcium channels in tinnitus. They postulated that nimodipine acts by affecting cochlear potentials. It abolishes the negative component of the summating potential and that means that there must be L-type calcium channels in the cochlea OHC. Nimodipine might affect the slow motion of the OHC and the resting position of the tectorial membrane. Using nimodipine as a pre-treatment drug abolished the salicylate- or quinine-induced tinnitus in the guinea pigs (Jastreboff et al 1994).

5.2.4. Vascular compressions and central nervous system disorders

Tinnitus can result from vascular compression into the eighth nerve and it is relieved immediately after a decompression operation. Moller (1992) considered that kind of tinnitus usually benign with only slight inconvenience to the individual. Arenberg et al (1983) described patients with objective pulsatile tinnitus. The most common lesions were arteriovenous malformations near the temporal bone (these are branches of the external carotid artery to the transverse or sigmoid sinus). Brookes (1996) presented a study with 9 tinnitus patients with either air computed tomography or MRI proven vascular compression into the eighth nerve. Microvascular decompression was carried out to all of them and 7 patients experienced a marked improvement of their symptoms. Remley et al (1990) published a study of 107 patients with pulsatile tinnitus or vascular retrotympanic mass, 25 of them had objective tinnitus. The most common finding was a vascular tympanic membrane (35%), followed by temporal bone tumours (31%), acquired vascular lesions (25%), normal vascular variants (21%) and from the rest (20%) no lesion could be detected with computer tomography (CT), magnetic resonance imaging (MRI) or angiography.

Wiggs et al (1996) published an article with 2 pulsatile tinnitus cases caused by type I Arnold-Chiari malformation and 1 caused by congenital stenosis of the sylvian aqueduct. One case with Arnold-Chiari malformation was successfully treated with suboccipital decompression. Sismanis et al (1990) published a work of 31 cases of benign intracranial hypertension (BIH) with pulsatile-objective tinnitus, the presenting symptoms with the disorder were headaches and/or visual disturbances but occasionally no other than tinnitus. Light digital pressure over the ipsilateral internal jugular vein decreased or abolished pulsatile tinnitus in all patients. Three of the patients had disabling tinnitus and were performed a lumbarperitoneal shunt.

5.2.5. Extralemniscal theories

Moller (1992) recorded brainstem auditory evoked potentials (BAEP) and compound action potentials (CAP) from the exposed intracranial portion of the eighth nerve in patients undergoing microvascular compression operation. The results seemed to prove that intractable tinnitus is not generated in the auditory nerve or classical (lemniscal) ascending auditory pathway, but it might be produced in the extralemniscal part of the auditory system. The lemniscal system gives projections onto the primary auditory cortex but the extralemniscal system projects onto association areas of the cortex. Moller (1992) described patients who suffered from intractable tinnitus and hypersensitivity to sounds and experienced sound sensations while, for instance, rubbing their back with a towel. He considered that as an indication of neuronal "crosstalk" between the auditory and somatosensory areas in some cases. Moller investigated the effects of stimulation of a somatosensory nerve (Median nerve) on the sensation of tinnitus in patients with intractable tinnitus. While being stimulated 9 out of 18 patients experienced a change in their tinnitus, four times a distinct increase, once a weak increase and four times a decrease (two of them also a change in the character of tinnitus).

Jastreboff postulated (1996) that the theory of extralemniscal pathways of tinnitus was grounded by several reasons: 1) No such firing in the auditory nerve has been recorded which could be detected only from the tinnitus patients and not from those without tinnitus. 2) The latencies of peak V were slightly shorter in patients with tinnitus. 3) When tinnitus patients were stimulated with tactile stimuli they expressed changes in their tinnitus. This could only be mediated through the extralemniscal system or association cortex, a place of coordination of auditory and somatosensory information. He postulated that the subcortical extralemniscal areas seemed to be the most possible candidates for tinnitus, especially the external nucleus and the dorsal cortex of the IC.

5.2.6. Spontaneous otoacoustic emissions (SOAEs)

When a transient sound, usually a click-stimuli, is introduced into a sealed ear canal, a re-emission of it can be measured. The phenomen is called transiently evoked otoacoustic emission (TEOAE). If there is sufficient acoustical amplification in between the middle ear and cochlea, the incoming acoustical signal level builds up constantly resulting a continuous oscillation in the cochlea. The oscillation makes so called spontaneous otoacoustic emissions (SOAEs), which usually occur at the level 0- 20 dB SPL (Wilson 1987). Eight studies in two countries had screened SOAEs and tinnitus in 374 people, 187 of the subjects had normal hearing and 187 had sensorineural hearing loss; 17 % of the normal hearing subjects and 85 % of the hearing impaired subjects had tinnitus. SOAEs were detected from 40 % of the normal hearing and 26 % of the hearing impaired people. SOAEs and tinnitus together were found from 10 % of the subjects with normal hearing and 17 % of the people with hearing impairment. The tinnitus pitch matched to the SOAEs frequencies of the same subject in only 1 % of those with normal hearing and 0.6 % of the hearing impaired. The authors conclude that SOAEs might be the source of tinnitus but not very often and more frequently among normal hearing individuals (Norton 1990).

5.3. Animal studies

Tinnitus can be produced to animals by giving them large amounts of salicylates. Eggermont (1992) used 450 mg/ kg in 5 ml saline intra-peritoneally in cats in light anesthesia. The response of single neurons in the auditory cortex were recorded. The explanation for the action of salicylate in producing tinnitus is, according to the author, that the Ca^{2+} -activated K^{+} - currents are blocked or reduced by the action of salicylate. This current moderates the firing rate normally, but as it is abolished by salicylate, this leads to an increase of both spontaneous firing rate and the number of inter-spike-intervals between 5 and 75 ms. Because the Ca^{2+} -activated K^{+} -current is specific for the pyramidal cells in the central nervous system the author finds this result a suggestion of a central component in salicylate-induced tinnitus.

Jastreboff et al (1992) studied rats with salicylate-induced tinnitus. The authors used 350 mg / kg of sodium salicylate, which caused serum salicylate to be 60 mg / dl. The rats were taught to feel safe in background noise by giving an electrical shock whenever noise stopped. The nervousness of the rats was estimated by their habit of stopping to drink when frightened.The rats with tinnitus did not fear in silence anymore, because tinnitus gave them a false feeling of safety. By changing the background noise, the pitch and loudness of tinnitus in rats could be estimated. If tinnitus differed too much from the backgrond noise the rats could tell them apart. Tinnitus induced by sodium salicylate had pitch around or above 11 kHz and loudness around 73 dB SPL of 10 kHz tone. The animals with tinnitus seemed to have OHC damage and also some IHC damage in the cochlea. Salicylate causes a decrease

in serum calcium, thus salicylate-induced tinnitus seems to be a consequence from its effect on calcium homeostasis in the cochlea, especially in the perilymph (Jastreboff 1992). With a similar test schedule Jastreboff et al (1994) studied the effect of calcium on salicylate-induced tinnitus. Pre-treatment of the animals with calcium supplement caused a reduction of tinnitus. If the animals were pre-treated with nimodipine (a chalsium channel antagonist) no tinnitus-related behavior occured when they were given either salicylate or quinine.

Kellerhals et al (1992) used two different ways to create tinnitus, noise and ototoxic drugs and measured the weight loss in rats. Rats are very sensitive to stress and the weight loss was explained by the stress tinnitus produced in the animals.

6. TREATMENT OF TINNITUS

6.1. Examination and counselling

Hallam (1987) pondered whether tinnitus is a medical, psychological or psychiatric problem and came to the conclusion, that these aspects cannot be separated. Even tinnitus of apparently low intensity can cause extreme distress to some individuals. Counselling could, according to Hallam (1987), be sufficient to relieve the stress of tinnitus in most sufferers.

Hazell (1987) emphasized proper examination of the patient and information of the mechanism of tinnitus. In his study he divided the causes of tinnitus to be located in the ear or causes without a clear origin, systemic, causes. If tinnitus originated in the ear the treatable causes were wax, conductive hearing loss and some other conditions that could be treated for example Ménière syndrome. The systemic forms of tinnitus were caused for example by drugs, food allergy, TMJ disorders, hypertension, anaemia, dyslipidaemia, hyperthyroidism, diabetes, syphilis, hypoglycaemia (especially early morning) and cervical disorders. He considered that all the unilateral causes of tinnitus should be investigated with for instance BSER or radiography to exclude an early acoustic neuroma. After investigations he explained the purpose of the examination and gave the prognosis and treatment options for the patient. Especially he wanted to convince the patient that tinnitus did not mean cancer, imminent stroke or psychiatric disorder.

6.2. Drugs

6.2.1. Lidocain

Barany (1935) reported that administration of local anaesthetic (procain) intravenously could reduce, at least temporarily, tinnitus that was due to cochlear malfunction (Evans 1981). Lewy (1937) used lidocain intravenously for the tinnitus treatment and a relief from tinnitus was noticed while treating acute attacts of Meniérè disease in the 1950s (Goodey 1981). Melding et al (1978) treated 78 incurable tinnitus patients with intravenous administration of 1 % lidocain, maximum of 2 mg/ kg body weight. They reported a good response in 60 % and out of them a total relief in 63 %. The same observation was confirmed by Evans (1981), the effect was produced by 1.0- 1.5 mg/kg iv lidocain. Hilders et al (1992) did a controlled study giving intravenous lidocain with a infusion pump. The group consisted of nine patients with tinnitus that had lasted more than a year. The pharmacokinetic characteristics of lidocain were first calculated for each patient according to their plasma concentrations in intravenous administration of a test sample of lidocain. In the study a computer controlled infusion pump was used to maintain a steady level of at least 1.5 µg / ml of lidocain plasma concentration an hour. Five of the nine patients had a suppression of tinnitus into a non-

annoying level, slight suppression was reported by two, one had no response and one reported an increase of tinnitus. The results were rated on t VAS-scale. None of the patients reported alleviation from NaCl infusion alone. Hilders et al (1992) considered the therapeutic window of lidocain plasma level to suppress tinnitus into non-annoying level to be 0.6- 2.0 μg / ml.

The mechanism of lidocain on tinnitus is not known. It is a local anesthetic, used to control cardiac ventricular arrhytmias and it is also a central nervous system depressant (Murai 1992). Lidocain can prolong the neural refractory period and thus reduce the maximum firing rate of the nerve. Lidocain was found to be able to bind with melanin in the inner ear. Melanin might be involved in the neural transduction process of the cochlea (Murai 1992).

Transtympanal injections of 4 % lidocain and also intratympanal injections have been tried with good results (Podoshin 1992). Lidocain 4% has also been tried in Ménière disease with a 10.7 % success in demolishing tinnitus and 57.1 % in alleviating tinnitus. Podoshin et al (1992) did a survey of 52 patients with idiopathic tinnitus, they were given 2 % lidocain with 1cc saline through grommets in ears. The mean duration of tinnitus was eight years. The intratympanal installations were given five times a week. Nine of the patients had an improvement after the treatments, three of them had alleviations for more than a week. The first signs of improvement were found after the third injection, but the treatment caused severe vertigo to all patients and only nine patients completed the treatment.

6.2.2. Oral anticonvulsant drugs

Carbamazepine. Carbamazepine is an anticonvulsant and also used in the treatment of neuralgic pain. Goody (1981) searched 125 tinnitus patients with lidocain test and the responders were given oral carbamazepine. Out of the patients getting a complete relief from tinnitus with intravenous lidocain, 62 % obtained at least 60 % relief with carbamazepine. If lidocain failed to give any response, nothing was obtained with carbamazepine (Goodey 1981). In another study with carbamazepine some patients could benefit from carbamazepine even when they failed to obtain any proper relief with intravenous lidocain (Goodey 1987). Murai (1992) refered 3 studies and no difference between carbamazepine and placebo could be found in any of them. The dose of carbamazepine varied from 100 mg three times a day to 150 mg three times a day.

Other anticonvulsant drugs. Phenytoin is a anticonvulsant like carbamazepine but less effective in the tinnitus treatment and Goodey (1987) did not discover any effect with it on tinnitus. Vigabatrin is an anticonvulsant and it is supposed to act in tinnitus by potentiating the inhibitory action of GABA in the cochlear level (Beck et al 1992). Beck et al (1992) studied the effect of vigabatrin on severe drug resistant tinnitus. They had 16 patients, out of whom 7 responded to the drug, with a reduction of tinnitus more than 50 % in the VAS-scale. Coles (1996) reported that vigabatrin had no effect on tinnitus but provided considerable side-effects.

Barbiturates act in several sites of the central nervous system but the polysynaptic pathways and midbrain reticular formation are especially sensitive (Murai 1992). Amylobarbital has been reported to decrease the tinnitus intensity with a dose of 50 mg of sodium amylobarbitone in the morning and afternoon and 80 mg at night (Murai 1992). Goodey considered amylobarbitone even weaker than sodium valproate. It may also give trouble in a prolonged use because it is a habituating drug (Goodey 1981). In a double-blind cross-over study with sodium amylobarbitone no significant differences could be found in the pre- and posttreatment loudness values of 9 treated patients in a review by Murai et al (1992).

Phenobarbitone is a metabolic product of primidone. Phenobarbitone is supposed to provide some relief from tinnitus (Goodey 1987). Goodey considered primidone comparable to barbiturates but with more adverse effects (Goodey 1981). Sodium valproate used 200 mg three times a day and 400 mg at night provided a mild relief from tinnitus (Goodey 1981). The effects of the drug appeared in two weeks. The drug was well tolerated, drowsiness was the most important side-effect (Goodey 1981). Amino-oxyacetic acid is an inhibitor of the catabolism of GABA causing reduction of the endocochlear potential. The site of action on tinnitus is not known. The effect of it on tinnitus was investigated with a dose of 50 to 70 mg four times a day for a week. In the active drug group 3 out of 10 patients and in the placebo group 2 out of 10 patients got some relief from the drug (Murai 1992).

Lamotrigine is a new anti-epileptic drug with sodium channel blocking abilities and it also inhibits glutamate release. Davies et al (1996) have tested the drug in 40 tinnitus patients without epilepsy and with severe tinnitus over 6 months. At the time of publication all patients had not completed the study, but only a limited effect on tinnitus seemed to exist.

6.2.3. Psychopharmacas

Tricyclic antidepressants. The action of antidepressants on tinnitus is not known. They may act only by changing the patient´s attitude to tinnitus making the situation more tolerable. Nortriptyline is mostly used in tinnitus suppression (Goodey 1981). Trimipramine 150 mg four times a day for six weeks created complete improvement in 1 out of 19 patients and partial improvement in 8. No change was reported by 3 and 7 patients had worsening of tinnitus. In the same study 8 patients had partial improvement with placebo (Murai 1992).

Benzodiazepines. Benzodiazepines are tranquillizers and used in tinnitus treatment. They act as anxiolytics, hypnotics and skeletal muscle relaxants. Their actual site and mode of action is not known, but their effect may be mediated through GABA receptors. Benzodiazepines may inhibit both monosynaptic and polysynaptic reflexes either by acting as inhibitory neuronal transmitters or by blocking excitatory synaptic transmission (Murai 1992). According to Goodey (1987) bentzodiazepines are not effective against tinnitus. Johnson et al (1992) came to another conclusion with alprazolam in a double blind study. The dose was raised from 0.5 mg to 1.0 mg after the first week and up to 1.5 mg after four weeks, the dosage was kept for four weeks. A relief was reported in 13 out of 17 patients in alprazolam group and 1 out of 19 in the placebo group. They considered the side effects of the drug minimal. According to Coles (1993) alprazolam is highly addictive and should not be used for this purpose. In a study of the characteristics of several benzodiazepines (diazepam, flurazepam, oxazepam and clonazepam) in tinnitus suppression with the placebo group given antihistamins, oxazepam and clonazepam had success rates of greater than 50 % (Murai 1992).

6.2.4. Other drugs

Betahistine. Betahistine is a specific treatment of Ménière disease (Goodey 1987, Pharmaca Phennica 1997). It has also been tried for tinnitus relief but in unselected cases no help has been found (Goodey 1987). Jauhiainen et al (1993) considered that it might be useful if tinnitus was related to the Ménière disease. According to Coles (1996) betahistine may improve the arterial blood supply to the cochlea and it might help if tinnitus was associated with arterial deficiency. He regarded 16mg t.d.s. as the minimum dosage and 3 months as the minimum duration.

Calcium channel blockers. As calcium homeostasis seems to be involved in tinnitus formation the interest in calcium channel blocking agents has also increased. Flunarizine seemed to affect the tinnitus patients who had dizziness at the same time (Murai 1992). Jastreboff et al (1996) studied two calcium channel blockers, nifedipine and nimodipine in the tinnitus treatment. They used male rats with salicylate-induced tinnitus and investigated their behavior after drug injections. Nimodipine seemed to be more potent, the dose of nifedipine had to be 8.66 times that of nimodipine, but both seemed to influence tinnitus in rats.They explain the effect by 1) a decrease of the calcium influx into the OHC and other cochlear cells 2) a decrease of the calcium influx into the neurons of the auditory pathway and 3) an increase of blood circulation into the cochlea or to the brain. Coles (1996) has tried nimodipine without success in the tinnitus treatment in humans but he mentioned that some positive results by another group had been obtained.

Neurotransmitter antagonists. Glutamic acid diethylester (GDEE) is a glutamic acid antagonist. Both glutamic acid and GDEE have been tried in the tinnitus treatment, both were given intravenously, the total amount being from 50 mg to 100 mg. The treatment was effective for several patients even for months (Murai 1992).

Caroverine is a potent α-amino-3-hydroxy-5-methyl-4-isoxazone-proprionic acid (AMPA) receptor antagonist and in higher doses also a non-competitive N-methyl-d-aspartate (NMDA) antagonist. AMPA and NMDA are receptors mediating the glutaminergic neurotransmission. Glutamate is a neurotransmitter between IHC and their afferents. The activity of these receptors might be disturbed in tinnitus. Caroverine was tried intravenously for 30 patients with assumed cochlear-synaptic pathology and associated tinnitus. In the genuine group 63.3 % responded immediately and in the control group (with 30 individuals) none. Some of the responders had tinnitus reduction for over a week, and improvements in the hearing level up to 10 dB were also measured in 7 individuals (Denk et al 1997).

Taurine. Taurine is a compound that is distributed in many tissues for example in heart, brain and muscle and it is binded to the cell membranes. The organ of Corti contains taurine and taurine seemes to be necessary for good hearing in cats (Harding 1992). Between the scala media and scala tympani there is a substantial calcium gradient and taurine may be associated with these calcium channels. Harding et al (1992) used 1g / day taurine supplement in 18 patients, 2 of them had a great improvement and 2 had a slight improvement. The authors considered it worth a try, because it has no side-effects, it is economically affordable and it is already widely distributed in the foods.

Streptomycin. Streptomycin is an aminoglycoside antibiotic, which is able to block receptor currents after being applied to the apical tips of ciliated vestibular hair cells. There is immediately a selective destruction of the type I hair cells of the crista ampullaris, sometimes it is followed with type II hair cells (Murai 1992). A tinnitus reduction in five out of eight Ménière patients has been achieved in one study using streptomycin in controlling Ménière syndromes (Murai 1992).

Ginkgo biloba. Ginkgo biloba is a plant extract which has been used alone and with other treatments for tinnitus. Von Wedel et al (1996) tried it alone and in combination with Low-Power-laser. In the final results there were no differences between the groups. The authors consider that adding laser did not improve the results, which were not promising. Zhou et al (1996) found however that EGb 761 (an extract of Ginkgo Biloba) attenuated salicylate-induced tinnitus in a dose-dependent nonlinear manner in rats suggesting at least some effectiviness of the drug on some types of tinnitus. The tinnitus behavioral expression of the animals was eliminated by a dose of 50 mg/kg/day.

Histamine. Clemis et al (1996) tried the effect of histamine on tinnitus, the drug was given either in subcutaneous or sublingual forms. The patients were mostly vertigo patients or patients with sudden sensorineural hearing loss, 21 individuals suffered from tinnitus as the main or only problem. In three quarters of the histamine treated patients in the sudden sensorineural hearing loss group tinnitus was resolved or improved. In the tinnitus group (altogether 34 people) 17 patients reported a substantial improvement, 6 some improvement and 6 no change in tinnitus.

Diuretics. The use of diuretics in the tinnitus treatment is based to the assumption that tinnitus is caused by the imbalance of fluids in the cochlea leading to endolymphatic hydrops. Another explanation was presented by Kemp (1981): if tinnitus is suspected to be SOAE it may be treated with furosemide or ethacrynic acid which are loop diuretics and shown to diminish the cochlear echo responses. The treatment has been tried on monkeys, but the loop diuretics were found rather to suppress than exacerbate the cochlear mechanical tinnitus. Baskill et al (1996) tried furosemide in tinnitus produced by SOAEs. The drug reduces the endocochlear potential and causes a reduction in firing rate of the cochlear nerve. None of the 6 patients got any relief of the treatment, the maximum dose was 80 mg/day.

Other drugs. The aim to improve the cochlear blood flow has led to the use of vasodilators. The most important vasodilators are nicotinic acid and papaverine (Clemis 1996). Alcohol has been reported to help in some tinnitus cases. Stephens et al (1996) concluded that alcohol seemed to have no effect in 60% of the cases, helps in 20 % of the cases and seemed to worsen tinnitus in 20 % of the cases. Cyclandelate is a vasodilating agent and acts directly on the smooth muscle of blood vessels. In a study by Hester et al (1995) it was tried on tinnitus. Only less than a half of the treatment group (12/29 individuals) completed the treatment because of side-effects. The mean tinnitus loudness score after three months treatment period was significantly lower in the treatment group than in the control group, the pretreatment values did not differ. The study was however not conclusive.

Asetosalicylic acid is known to produce tinnitus (Evans 1981). It is also used in tinnitus treatment if the tinnitus is a SOAE sound, which is quite seldom the case. Baskill et al (1996) mentioned one successful suppression of tinnitus by asetosalicylic acid.

Hyperbaric oxygen has been used for hearing disorders and tinnitus. Almeling et al (1996) treated 178 patients with acute tinnitus in a chamber-treatment. The reduction of tinnitus loudness for more than 50% was found in 19.9 % -29.3 % of the patients depending on the length of the tinnitus history before the treatment. Carbogen has also been used against tinnitus (Pringle 1996).

Baclofen is a gamma-aminobutyric acid analog selective for the GABA B receptor. It is used for the treatment of increased muscle tone and spasticity and also for the treatment of trigeminus neuralgia. In animal studies it causes a suppression of both the cochlear nucleus and the inferior colliculus. Westerberg et al (1996) used baclofen for tinnitus during a three weeks course. The subjective and objective evaluation failed to indicate any differences between the active drug and the placebo.

6.3. Masking

6.3.1. Hearing aids

Tinnitus might be masked with amplification of the everyday sounds with a hearing aid. The use of hearing aids against tinnitus in otosclerosis has been tried already in 1947 by Salzman et al. They compared hearing aids to a fenestration operation and supposed that when a lot of outside noise is entered into the cochlea the result is a kind of masking (Salzman et al 1947). Vernon found hearing aids to be beneficial in bilateral slowly sweeping high tone hearing loss but not with the steeply sloping audiogram (Vernon 1977). Melin et al (1987) studied the effect of hearing aids on tinnitus with 39 patients and got no benefit from it against tinnitus in VAS. In the interview a significant decrease was reported by the patients (13/ 39) who used the aid more than 2 hours daily. The researchers explained the discrepancy with the expectations of the people, who tend to exaggerate the help given by a long treatment. Authors also considered that VAS might be too insensitive to these kind of treatments. High-frequency hearing aids were recommended by Miller (1981) for subjects who have normal hearing at frequencies 250- 1000 Hz and a drop at 2000 Hz to the level of 40 dB or more.

6.3.2. Maskers

Masking means relieving the distress of tinnitus with the help of a more acceptable, masking sound. Actually in a broad sense masking is also produced by natural surrounding sounds for instance waves by the seaside. Masking is commonly achieved by a white noise generator with filters and volume control (Hazell 1981). Vernon et al (1981) considered that masking is effective in 60- 80 % of cases but maskers must be properly fitted to achieve this effect. Hazell et al (1981) emphasized the importance of instruction and counselling. Only 12 % of patients got continuous relief with maskers given out in an office visit but the acceptance increased to 81 % with counselling and a follow up in the hospital. The success rate of maskers in general has been lower, Roeser et al (1980) reported a benefit in 26 % of the patients with maskers. The effectiviness of maskers seemed to differ from 10 % to 80 % (Roeser et al 1980).

6.3.3. Comparing hearing aids to maskers

Several reports about the usability of maskers and hearing aids in tinnitus treatments have been published. Roeser et al (1980) compared hearing aids and maskers with a conclusion that hearing aids were more effective. Melin et al (1987) rewieved other comparisons : Kiessling (1980) considered hearing aids more effective, Coles (1984) found maskers better and Mehlum et al (1984) found hearing aids and maskers equally effective.

In a longitudinal study on 204 tinnitus patients Sheldrake et al (1992) found hearing aids and maskers to be equally effective on tinnitus. During the study time 37 % of the masker users and 5 % of the hearing aid users had stopped to wear the device. Of all the patients they studied 57 % received maskers, 41 % hearing aids and 2 % combination instruments (containing both a hearing aid and a masker). The maximal habituation to tinnitus seemed to happen between 2 and 8 years.

Goldstein et al (1992) listed the total amount of devices used in their clinic for tinnitus masking purpose during 1977- 1990. The total amount of instruments diminished from 197 in 1977- 1980 to 45 in 1987- 1990. 97 maskers were purchased in the first three years, in the last three years only 12. The same tendency was shown in the purchase of tinnitus

instruments (containing a masker and an amplifier in the same instrument), the decrease was from 39 to 6 and even hearing aids used for tinnitus purpose diminished from 37 to 13 in the same years, the overall success rate was 85 % with right fitted devices. This was achieved by a careful patient selection.

6.3.4. Cochlear implants

The cochlear implant creates a sensation of sound by stimulating the auditory nerve with an electrical current (Berliner et al 1987). In 1973 one patient receiving a cochlear implant reported a disappearance of tinnitus while wearing the device (Berliner et al 1987). In a questional survey 10 patients out of 12 reported a positive effect on tinnitus while wearing a cochlear implant (Berliner et al 1987). Bredberg et al (1992) published a study of 18 tinnitus patients wearing a cochlear implant, 50 % of them reported an improvement of tinnitus while using the cochlear implant. Zwolan et al (1992) had 28 tinnitus patients wearing a cochlear implant, 54 % of them reported that using the implant decreased the loudness of their tinnitus and 71 % thought it was helpful.

6.4.Treatment by stimulation

According to Soussi et al (1994) electrical stimulations have been tried in tinnitus treatment by Volta already in 1800, Duchenne de Bouloigne tried electrodes in the external canal in 1855 and Polizer tried direct and alternating currents on many diseases of the ear including tinnitus in 1883. Positive polarity has been noticed to suppress tinnitus but negative polarity to produce a sensation of sound. Negative DC current may also damage the cochlea in guinea pigs (Steenerson et al 1996).

6.4.1. Transcutaneous nerve stimulation (TNS)

The effect of low-frequency (2 Hz) TNS is based on its ability to induce a wide-spread, non-segmental and prolonged increased microcirculation due to sympatico-inhibition in vascular beds (Kaada et al 1989). Kaada et al (1989) treated 29 tinnitus patients by using TNS stimulator, 9 patients reported a reduction of their tinnitus. The improvement was more common with low-frequency (125- 500 Hz) tinnitus. 7 patients reported also an improvement of hearing mostly in the low-frequency band. In another study with 20 Meniérè patients Scott et al (1992) could not find any beneficial effects on their tinnitus but self-recordings in the TNS group expressed increased ability to hear.

6.4.2. Other electrical stimulations

Transcutaneous electrical stimulation around the ear. By the use of a low amperage, low frequency variable square wave applied to, on and around the auricle, Engelberg et al (1985) were able to reduce tinnitus in six of the ten subjects participating in their survey. They used 13 cutaneous sites selected by their low resistance readings with the help of a pulse generator with a sensing mode (Alpha Stim 2000). Those sites were empirically selected for their abilty to reduce tinnitus after several trials. The stimulation was given there by the same pulse generator using modified square DC biphasic pulse with a frequency of 0.5, 1.0 or 2.0 Hz for 24- 120 seconds. In 2 subjects with bilateral tinnitus one ear responded but the other did not. In the second experiment Engelberg et al (1985) had 10 treatment patients (17 ears) and 10 control patients (15 ears), the latter were given false stimulation. In the placebo group 1/ 15 ears improved and in the treatment group 15/ 17 ears improved (meaning 9/ 10 of the treatment group patients). Steenerson et al (1996) tried electric stimulation of the auricle for tinnitus. They found that the skin resistance of the auricle in the tinnitus patients was

increased but decreased during the treatment. In unilateral tinnitus cases only the side with tinnitus had increased skin resistance in the auricle. Out of 246 patients 53 % experienced a reduction of their tinnitus. Transcutaneous electrostimulation was found effective on noise-induced temporary threshold shift (TTS) in voluntary patients. Electrostimulation reduced TTS in the majority of cases and the reduction was statistically significant. The used points for electrodes were the pretragal point, the point beneath the zygomandibular arch of cheek, the point over the styloid foramen and the paramedian point in the nape of neck, which have also been used in tinnitus treatments (Tachibana et al 1992).

Electrical promontory and round window stimulation. Aran et al (1981) tried both promontory and round window electrical stimulation on their patients. Promontory stimulation was achieved by a sine-wave generator with constant-current square waves. 83 patients were treated and 43 % of them experienced a diminution of tinnitus, a complete disappearance in 25 %. Okusa et al (1993) tried electrical stimulation with transtympanic needle electrone on promontory to relieve tinnitus in 62 sensorineural deaf patients. The frequency varied from 50 Hz to 400 Hz and the maximum intensity was less than 100 uA. In 46 of 68 ears a reduction of tinnitus was achieved and the method seemed to be the most effective in noise induced hearing loss. Residual inhibition was accomplished in twenty patients lasting from hours up to one week. Ward et al (1992) compared the effect of promontory and external ear canal electrical stimulation; 64 patients were given promontory stimulation, 79 external canal stimulation and 60 patients were treated with both of them. The promontory stimulation was effective in 48- 50 % of cases and the external canal stimulation in 28- 30 % of the cases and the difference was statistically significant.

Aran et al (1981) used a ball electrode in the round window stimulation. Eleven patients with a round window electrode were investigated during stimulations and in 6 of them tinnitus vanished. The stimulus was inaudible for some of the patients. The promontory and round window stimulation were compared in 12 patients. The promontory stimulation was ineffective in all, but five subjects experienced a complete suppression of tinnitus with the round window stimulation. Hazell et al (1993) tried direct round window stimulation with 4 Hz to 2 kHz electrical current. It induced an auditory sensation in all subjects. In seven out of nine subjects a total suppression of tinnitus was achieved, but in no case without a sensation of hearing.

Electrical brainstem stimulation. Soussi et al (1994) tried the auditory brainstem implant (ABI) in 18 bilaterally deafened patients. The electrical brainstem stimulation was achieved by an electrode placed on the dorsal cochlear nucleus of neurofibromatosis-2 patient after translabyrinthine tumor removal. The device was placed at the entrance to the fourth ventricle over the cochlear nucleus complex. The frequency of the output was between 250 Hz and 4000 Hz. Only ten of the patients could use the implant, seven subjects used the implant daily and six of them reported a considerable reduction in the tinnitus intensity.

6.5. Relaxation

Several relaxation techniques have been used to overcome the distress caused by tinnitus. Stress is a well-known aggravating factor of tinnitus and relaxation regardless the way it has been produced has been reported to help (Gabriels 1996). Many of the different treatment modalities are based on the relief from tinnitus caused by muscular relaxation (Hallam 1987, Hazell 1987).

6.6. Acupuncture

Acupuncture is a Chinese treatment used traditionally also for tinnitus suppression. Hansen et al (1982) studied in a double blind controlled trial the effectiveness of the traditional Chinese acupuncture on tinnitus. No difference between the actual treatment and placebo acupuncture among 17 patients with chronic unilateral tinnitus could be found. Marks et al (1984) did also a double-blind cross-over controlled treatment trial on tinnitus with 14 patients. Five patients got an improvement from the genuine treatment and one from the placebo treatment, the effect was not statistically significant. The authors considered that acupuncture may be benefical for some tinnitus patients. Nilsson et al (1992) used acupuncture to treat 56 patients with continuous and severe tinnitus. In three patients the suppression of tinnitus lasted for at least ten days and 12 patients had intensity reductions for hours/days. On the contrary the statistical analysis of the whole group failed to show any significant improvement. The authors stated that acupuncture may be useful in the tinnitus treatment in selected cases. Gu et al (1992) used acupuncture for 193 patients with tinnitus. 55 % of the patients improved, among them 31 % had a significant relief, 18 % some and 6 % an occasional relief. One of their patients had a tinnitus free period of 18 months after the treatment period. Most of the patients (72 %) responded before the fourth treatment and the effect became stronger along the continuation of the treatment.

6.7. Biofeedback

Biofeedback methods are used to teach an individual to exert conscious control over those areas of body outside normal conscious mind. House (1978) tried the biofeedback method with 16 tinnitus patients in 3 months' program, with 12 one hour settings. More than a half seemed to benefit from biofeedback training, and the ability to cope with tinnitus improved. Landis et al (1992) tried biofeedback for chronic tinnitus patients in a 5 months' program with a trained therapist for 90 minutes weekly and in addition independently 30 minutes daily. The patients were taught to use various, individually most suitable relaxation techniques to control the stress with the help of the sympathetic nervous system feedback markers. Only seven subjects were able to complete the whole program. Audiological measurements did not confirm any reduction of tinnitus but the training seemed to affect the patients´ability to cope with the situation. The same result with biofeedback was achieved by Lindberg et al (1987) who reported no reduction of tinnitus in tinnitus ratings in 72 % of the subjects but 65 % of them informed having a subjective improvement.

Grossan (1976) used EMG feedback with 51 patients. He had a device with sensors placed on the frontalis muscle and earphones coming from it. In case of muscular tension a click was heard through the earphones. The patients got aware of muscular tension by hearing the click. The patients were taught the technique in six 20 minutes' visits. Four patients had a measurable improvement of tinnitus, 40 felt a subjectively significant improvement. The author concluded that the best candidates for this kind of treatment were people with the anxiety type of muscle tension related to tinnitus. Ogata et al (1993) used measurements in the frontalis muscle with connections to monitor for the patients to see and hear their muscle contractions. Seven patients with therapy resistant intractable tinnitus were treated once or twice a week with biofeedback sessions lasting 18 minutes, and depending on the individual 2 to 16 sessions were given. Three patients improved, one patient had a reduction of tinnitus during the sessions but not otherwise and three patients did not benefit.

6.8. Hypnosis

Marks et al (1984) tried hypnotherapy for therapy-resistantchronic tinnitus. A group of 14 patients were treated, one of them showed a marked improvement in VAS and 5 had some improvement. Hallam (1987) tried hypnosis and relaxation with two tinnitus patients. One of them was given a post-hypnotic suggestion that whenever the patient was aware of tinnitus it changed to a pleasant music. Tinnitus was relieved in both patients. In another experiment the combination of relaxation and hypnotherapy was used to change tinnitus to a more pleasant sound. The patients were also taught to turn tinnitus down with a control knob in their imagination. After the treatment tinnitus was better accepted by the patients than before the treatment. Attias et al (1993) compared self-hypnosis, masking and “attentiveness to the patient´s complaints” in the treatment of tinnitus. Self-hypnosis reduced the tinnitus severity significantly, attentiveness partly but masking did not have any significant effect.

6.9. Treatment of the external ear canal

In a few instances tinnitus can be caused by wax or hair in the ear canal. The sound is mostly rasping or a friction noise or the underlying tinnitus is rended more audible by the blockage of ear canal (Wilson 1987). Vernon (1977) lists wax against eardrum to the correctable tinnitus causes. The removal of the cause might remove tinnitus but not always (Douek 1981). Coles (1996) on the contrary stated that many patients claim that their tinnitus has started immediately after an ear syringe and he considered the co-insidence too frequent to be just due to chance.

6.10. Surgery

Surgery has relatively little to offer for tinnitus treatment in general. However, some conditions can be effectively treated with surgery.

6.10.1. Middle ear surgery

Hazell (1987) investigated 472 patients with tinnitus and only 6 % had the diagnosis otitis media and 4 % otosclerosis. Thus the possibility that middle ear operation can resolve tinnitus is limited to very few people as it is a rare cause of tinnitus. Parisier et al (1984) investigated the occurence of tinnitus in patients with chronic mastoiditis and cholesteatoma. Pre-operative tinnitus occured in 34% of 82 patients and in 10 cases (12%) it improved after the operation. In addition in 10 cases (12%) pre-operatively tinnitus-free people noticed tinnitus after surgery. Thus the amount of people with tinnitus increased post-operatively.

The effects of stapes surgery in 190 cases of otosclerosis were investigated, 78.9 % of the patients had pre-operative tinnitus. When air/ bone closure was better than 15 dB in all frequencies and the stapedectomy was performed, tinnitus disappeared in 31 % of the patients, decreased in 33 %, unchanged in 23 % and increased in 3 %. In an another study only low and medium tone tinnitus disappeared after stapedectomy. Thus tinnitus between 250 Hz to 1000 Hz might be caused by ossicular impedance changes or by high pressure of the labyrinthine fluids (Hazell 1987).

If the Eustachian tube remains open all the time individual might hear the sound of his own respiration in the ear combined often with a feeling of fullness and blocking of the ear and generation of reverberant effects in the ear by one’s own voice. Symptoms often disappear by bending the head down between the knees (Hazell 1990). Different techniques have been

tried to narrow the lumen, for instance 20 % of silver nitrate application to the Eustachian tube via the nasopharynx. Also Teflon injection has been used into the Eustachian cushions, with this technique 19 out of 26 patients had a complete disappearance of the problem. The insertion of the tensor velipalatini muscle, opener of the Eustachian tube, has been also been transposed with an improvement of tinnitus in three patients (Hazell 1987).

Musculus stapedius and tensor tympani may cause sounds as a response to environmental loud sounds or by touching the skin in front of the ear involved or, for instance, blinking an eye. The synkinetic contraction of the tensor tympani and masseted has been reported to cause tinnitus. Two patients with tinnitus caused by stapedius muscle were helped by the section of the muscle. Also four patients with sounds in the ear elicited by loud tones and in one patient also by touching the skin of the cheek became symptom-free after the stapedius and tensor tympani tenotomy (Hazell 1990).

6.10.2. Inner ear surgery

At the begining of Ménière disease tinnitus may be prevalent only during a vertigo attack. As the disease progresses tinnitus may become constant and be low-pitched. With the endolymphatic sac operation about one third of patients with Ménière disease experienced a disappearance of tinnitus, another third reported a reduced level of the tinnitus intensity and one third had their preoperative intensity one year after the operation (Hazell 1990). In Ménière disease the destruction of labyrinth is performed in order to control the vertigo. The results of ablative surgery varied greatly, in a review provided by Hazell (1990) tinnitus could be abolished in 1-67%, improved in 6-61%, stayed in the preoperative value in 5-44% and aggravated in 0-60% of cases.

The sectioning of the eighth cranial nerve is an alternative operation. Reports on nerve sectioning are not in agreement. House et al (1981) presented 68 patients with a mostly vestibular nerve section but sometimes also the cochlear nerve had been sectioned. Only one of the operated patients had a complete disappearance of tinnitus, 43 % had their tinnitus diminished but in 27 % tinnitus did not change and it was aggravated in 28 %. Better results were obtained with a translabyrinthine section of both the superior and inferior vestibular nerves and the cochlear nerve. Out of 93 patients with tinnitus, tinnitus was ceased in 67% of the patients, the situation had improved in 28 % and only 5 % of the patients got no relief (Pulec 1984).

In 27 cases of post-stapedectomy perilymph fistula, 10 of the subjects had a very severe roaring type tinnitus. In spite of the closing operation tinnitus did not improve very much especially if pre-operative tinnitus had been severe (Hazell 1987).

House et al (1981) presented 500 vestibular schwannoma patients, 470 of them had vestibular schwannoma removed through the translabyrinth approach. 414 patients of the 500 had tinnitus preoperatively. Postoperatively 40 % reported that their tinnitus was better, 10 % stated that it did not change in the operation and 50 % had an aggravation of the tinnitus.

6.10.3. Surgery for vascular abnoralities and central nervous system disorders

Vascular abnormalities can be divided into arteriovenous shunts, arterio-arterial shunts, venous hums and other arterial bruits (Hazell 1987). Pulse synchronous tinnitus is sometimes audible by the stethoscope. It is often aggravated by turning the head to the opposite direction and reduced by turning the head to the affected side. Light pressure on the

internal jugular vein abolishes the sound (Hazell 1987). In these cases the ligation of the jugular vein has been suggested.

Arteriovenous malformations are commonly found in branches of occipital artery and transverse sinus. There may be several interconnections between these vascular structures. Branches of the internal carotic artery and vertebral artery may become fistulous. Sometimes fistulas are found in the middle fossa between the posterior branches of the middle meningeal artery and greater petrosal sinus. A connection between the internal carotic artery and the cavernous sinus is almost always a result from a severe head trauma (Hazell 1987). By embolizations with gelatin sponge to the feeding vessels in severe pulsatile tinnitus 75 % of the patients obtained a lasting satisfactory result (Hazell 1987). Wiggs et al (1996) presented 2 pulsatile tinnitus patients with Arnold-Chiari malformation, one patient was performed a suboccipital decompression and her symptoms subsided.

Vascular loops can also be detected in the internal auditory canal. The anterior inferior cerebellar artery (AICA) might enter to the internal auditory canal producing compression into the eighth nerve and patients with vascular loop in the internal auditory canal close to the eighth nerve have been found. With vascular decompression of the eighth nerve two thirds of them became better and the rest remainded in the preoperative level or their tinnitus worsened (Hazell 1987). Brookes (1996) published nine cases of vascular decompression surgery in tinnitus patients with proven vascular loop in contact with the cochlear nerve. Four of the patients were operated by the retrolabyrinthine and five with the retrosigmoid approach, the latter prefered by the author. The tinnitus was pulsatile in three patients and all the patients claimed their tinnitus to be extremely severe. All the previous therapy had been unsatisfactory. Tinnitus was completely abolished in three patients and significantly improved to a sensation level of under 10 dB in four patients. One patient had a reduction to a sensation level of 15 dB and in 2 cases there was no change to the preoperative values.

7. TRIGGER POINTS

7.1. The character of trigger points

Travell et al (1983) define trigger point as " a focus of hyperirritability in a tissue that, when compressed, is locally tender and, if sufficiently hypersensitive gives rise to referred pain and tenderness and sometimes to referred autonomic phenomena and distortion of proprioception". The authors divided the types into myofascial, cutaneous, fascial, ligamentous and periosteal trigger points. Han et al (1997) list the associated problems that combine with the trigger point and pain as follows: stiffness, weakness, increased fatigability and decreased range of motion.

The pain from the trigger points is referred in specific patterns characteristic to that specific point in the muscle. The more sensitive the trigger point, the more intense is the referred pain. The referred pain does not follow a segmental model, but the trigger point and the referred pain area are usually within the same dermatome. Trigger points can be latent or active, latent meaning points that can be manually detected but do not elicit any referred pain or autonomic phenomena. The active ones elicit pain or autonomic phenomena with typical reference zones. The activity of the trigger points varies even on an hour-to-hour basis and the latent trigger points can be converted to active ones with acute overload, overwork fatigue, chilling, direct trauma, visceral disease, emotional stress or they may be activated by other active trigger points (Travell et al 1983).

The trigger points are a source of pain not only locally but refer influence to a distance from the site of injection (Travell et al 1983). Myofascial trigger points may refer vascular, secretory and pilomotoric autonomic changes (Travell et al 1983). They may also produce skeletar motor, visual, vestibular and space-perceptual disturbances. Vasomotor autonomic effects are common with trigger points. For instance active trigger points in the shoulder area produce skin temperature changes. Active trigger points in the sternal division of the sternocleidomastoid muscle produce also a discharge of tears and reddening of the conjunctiva. Secretary autonomic changes produced by active trigger points are for instance coryza, lacrimation and variations in the sweat pattern. Active trigger points in the clavicular division of the sternocleidomastoid muscle may cause imbalance and disorientation of the body in space or postural dizziness. Sternocleidomastoid trigger points cause also visual disturbances like blurring of vision and intermittent double vision without pupillary changes (Travell et al 1983).

The termographic pattern of the trigger points was evaluated by Diakow (1992). The thermographic evaluation was performed to patients with neck and low back injuries and the author considered the method to be a useful tool to distinguish the latent and active trigger points in trigger point characterization. Kruse et al (1992) noticed asymmetric thermal patterns in all trigger points in sensory referral area and also distal to the referral area before compression. During compression of the trigger point the temperature of the thermal referral area showed a reduction in temperature from precompression levels. If similar asymptomatic area was compressed no significant temperature changes were observed.

Referred pain from the neck and shoulder area is more common than from the buttocks, knees and hips. The muscles that harbor most trigger points are sternocleidomastoid muscle, trapezius, masseter, temporalis, pterygoid, levator scapulae and splenius capitis and cervicis. In the head and neck region tinnitus, tension headache, temporomandibular joint pain, eye symptoms and torticollis might be caused by myofascial pain syndrome (Han et al 1997).

The theory that myofascial pain is originating primary from the trigger points has also been questioned. Quintner et al (1994) proposed that trigger points do not exist at all. The deep aching pain and autonomic phenomena combined with them could be explained by referred pain and hyperalgesia in the nearby compressed nerve.

7.2. The prevalence of trigger points

Latent trigger points were found in the shoulder muscles from 54 % of women and 45 % men in one study among 200 asymptomatic 17- 35 year old subjects. One or more tender areas were detected from 49.5 % of the individuals, but these points gave a referring pain reaction only in 12.5 % of the subjects. Of the total of 250 tender areas noted, 84.7 % occured in just four muscles: the trapezius, the levator scapulae, the infraspinatus and the scalenes. Women seemed to be more susceptible than men to develop trigger points. Among 739 college students significantly more women than men had headaches and problems of temporo-mandibular joint area. Twice as many women as men had tenderness in their lateral pterygoid muscle (Travell et al 1983). Myofascial pain syndrome was found in 30% of women aged 20-40 years, 6% of them needed treatment. Pain increased in the second week of menstrual cycle suggesting hormonal influence. Shoulder girdle and lower back had more trigger points than the rest of the body (Han et al 1997).

7.3. The pathogenesis of trigger points

The trigger point is supposed to develop after an acute injury or after repeated microtrauma capable of causing disruption of the sarcoplasmic reticulum at the same point. Prolonged poor posture, lack of exercise, poor nutritional habits especially vitamin deficiency might also predispose to the trigger point formation as might parafunctional oral habits like teeth grinding, sleep and joint disturbances. Traumas might cause release of calcium ions in the presence of ATP and this activates actin-myosin contractile mechanism leading to the taut muscle band formation. As the contractile activity of the local muscle fibres increase, it leads to the accumulation of serotonin, kinins, histamine and prostaglandins. The local muscle activity increase leads also to the activation of the muscle nociceptors (group III and IV nerve endings), which may be responsible for the local and referred pain (Han et al 1997).

7.4. Fibromyalgia and myofascial pain syndrome

Fibromyalgia syndrome and myofascial pain syndrome express themselves with tender or trigger points in the muscles. Both diseases are more common among women. They might in fact be different stages of the same disease but Bennett (1991) considered them to be different entities. In myofascial pain syndrome the problems are limited to muscles in a certain area with trigger points and referred pain zones and no tender points could be found in other areas of the body. In fibromyalgia syndrome the tender points are detected in all four quadrants of the body. In addition there are fatigue of the muscles and Raynaud´s phenomen in fibromyalgia syndrome but not in myofascial pain syndrome. Sleeping disorders are noticed in fibromyalgia, namely disturbances in stage IV sleep character, the alpha rhythm (7.5- 11 Hz) is superimposed by a slower delta rhythm (0.5- 2 Hz) in the slow wave sleep. The tendency for this kind of sleeping disorders has also been detected in healthy relatives of fibromyalgia patients in six families and it might be hereditary. Thus the tendency to develop fibromyalgia might be genetic (Bennett 1991).

Borg-Stein et al (1996) characterized fibromyalgia as a condition where tender points occur in a widespread manner. They also considered that the two phenomena can coexist leading to overlapping of syndromes.

7.5. The laboratory findings of the trigger points

7.5.1. Blood tests

Routine blood tests cannot be used to separate the people with either myofascular pain syndrome or fibromyalgia from healthy individuals (Travell et al 1983). A significant increase in blood myoglobin was found in 21 of 26 patients who were receiving massage for their myofascial pain syndrome. A positive correlation was found between the amount of myoglobulinemia and muscle tension and pain. The five patients who did not benefit from the massage did not get myoglobulinemia either (Bennett 1991).

7.5.2. Biopsies

In myofascial trigger point the biopsy has revealed disruption of myofibrillar structure, increased amounts of ground substance, mucopolysaccharides, multifocal loss of selected oxidated enzymes and occasional "ragged red" fibres suggestive of mitochondrial damage. However, these changes have often been found in healthy individuals as well (Bennett

1991). The light microscopy findings of muscular biopsies from the left trapezius of 12 fibromyalgia patients consisted of scattered hyalinized fibers, occasional split fibers, and an increase of central nuclei in two biopsies. No inflammatory changes were found but some fibers looked "moth-eaten", that kind of appearance was detected from type I fibers in 42 % of the patients and some kind of athrophy in type II fibers was noticed in 58 % of the patients (Bennett 1991). The accumulation of water and fat, mucopolysaccharides, platelets and degranulation mast cells have also been found in fibrositic nodules. Platelets and mast cells are capable of releasing serotonin and histamine. They are substances capable of stimulating peripheral nerve endings and the excess of serotonin or histamin may lead to a hyperirritable state (Han et al 1997).

A decreased level of high energy phosphates (ATP, ADP and phosphorylcreatinine) in muscular biopsies at the trigger points have also been found. Muscle biopsies from the trapezius muscles of fibromyalgia patients were showing a lack of high energy phosphates compared to muscular biopsies from normal subjects or muscular biopsies from the tibialis anterior muscle of the same patients. Low lewels of high-energy phosphates in flexor forearm muscle were also found from patients with fibromyalgia (Bennett 1991).

7.5.3. Electromyographic studies

The nodules in trigger point areas were originally thought to be a muscle spasm but they have proven to be electrically silent. Bursts of motor activity have been reported while stimulating the trigger points (Bennett 1991). Muscles with trigger points have been shown to express significantly higher motor unit action potentials upon snapping than normal muscles (Benett 1991). In some studies two types of spontaneous activity in the trigger points could be found: a low amplitude constant background activity and an intermittent higher amplitude activity. Curare abolished all voluntary activity but could not diminish the spontaneous trigger point activity. Phentolamine, a selective sympathetic blocking agent blocking the action of noradrenaline, was able to abolish the spontaneous activity of trigger points, but not the voluntary activity. These findings suggest that trigger points may have connections to the sympathetic nervous system (Gerwin 1994).

EMG made for 10 individuals with myofascial syndrome involving the shoulder girdle, showed a shorter endurance on the more painful side in all patients. In addition the endurance times were significantly reduced compared to healthy volunteers. The endurance time on the more painful side was directly related to EMG fatigue meaning that myofascial pain syndrome has decreased the working capacity of the shoulder muscles (Bennett 1991). Carlson et al (1993) used a single trigger point injection with 2% lidocaine solution (without adrenaline). After an injection of lidocain into the trapezius muscle a significant ($p<0.001$) reduction of pain in the ipsilateral masseter muscle and a significant ($p<0.03$) EMG activity reduction in the same muscle were observed. The finding suggests that trigger points can act as sources of deep pain by causing heterotopic sensory and motor changes in distant anatomic regions.

McNulty et al (1994) evaluated trigger points with needle EMG in a stressful situation. The needles were put to a trigger point in the trapezius and also in another nontender point in the same muscle and the measurents were done during psychological stress caused by mental arithmetic exercises. The trigger points showed a significant increase in their ENG activity during stress but no such response was shown by the nontender points. During nonstressful situations no difference was found in the ENG activity regardless of the place.

7.6. The supposed neural connections of the trigger points

The pain in the trigger points might be mediated by A-delta III nociceptive fibres responding to thermal and high-threshold mechanical stimulus or thin unmyelinated group IV C fibres, which respond to thermal, mechanical and chemical stimulation. They are nociceptive fibres, situated in a close relationship to blood vessels in the skin and in the muscle. Free nociceptive nerve endings have not been found in muscle fibrils. The free nerve endings of nociceptive nerves are sensitive to substances like bradykinin, 5-hydroxytryptamine and histamine, which are naturally occuring pain-producing substances. Trauma or repetitive strain to the muscle may release these substances leading to myofascial pain syndromes. Opioids have an analgesic effect when injected locally in areas with periphery inflammation of the muscle. On the contrary adrenaline sensitizes the damaged muscle to pain but does not affect normal muscles, suggesting some connections with the myofascial pain and the sympatethic nervous system (Gerwin 1994).

The group III A-delta and IV C fibres terminate in the superficial dorsal horn. The dorsal horn neurones, receiving input from the skeletal muscle, have 2 receptive fields, one from the muscle and the other from the skin that might be distant from the muscle. The input from those two fields is then converging to the same neurone, which can explain some cutaneous representations described with the deep muscular pain. In a condition when descending inhibitory impulses are blocked or facilitory ones increased, a state of hyperalgesia can occur. This has been produced with a reversible cold block of the spinal cord and it affected more the neurones recieving input from deep tissues than those with only cutaneous representations. Mild weakness of muscles with trigger points can be explained by the reflex inhibition of the anterior horn cell function as a result of painful sensory input (Gerdwin 1994).

7.7. Treatment of trigger points

The treatment of trigger points and the associated pain and restricted motion can be achieved by several methods. Pharmacological agents, massage, ultrasound, vapocoolant treatment, exercise or electrical stimulation or injections may be used. The injection techniques have been performed with acupuncture needles as dry needling or with injection of isotonic saline, procaine, lidocain, bupivacaine or mepivacaine. In several study designs the response seemed to be connected to needling itself and not so much to the medicine used. However the effect of saline or local anesthetic was more effective than dry needling or placebo. Injection seemed to be most effective when the site of maximal tenderness was located and the exact place injected. The pain relieving effect of local anesthetic lasted longer than the actual duration of the medicine should last (Han et al 1997). Hong (1994) preferred lidocain 0.5% for dry needling because in their study 42% of the patients with lidocain injection developed postinjection soreness compared to 100% of those with dry needling although significant improvement to the pain and range of motion was experienced in both groops. Murphy (1997) used trigger point injection in temporomandibular disorder with success.

Han et al (1997) listed several theories to explain the effect of injection techniques. The mechanical disruption of muscle fibres and nerve endings might be caused by injection. The mechanical disruption of muscle fibres might also increase intracellular potassium leading to depolarization of nerve fibres. In one theory injection might interrupt the positive feedback mechanism perpetuating pain. Local injection might dilute the local nociceptive agents causing pain. The vasodilatory effect of local anesthetics might also increase the removal of metabolities. The release of endogenous opioid, depletion of substance P, kinins and

histamine from afferent nerve fibres, aberrant reflex circuit disruption and sympathetic tone alteration are all possible but unproven explanations for the prolonged effect of local anesthetic injections in the pain treatment (Han et al 1997). Some of the trigger points combine with acupuncture maps; 71 % overall correspondence was reported by letting a 3 cm difference of distance to be between the two (Travell et al 1983). The difference between acupuncture and trigger point injections is the following: the local anestethics used in the trigger point injection treatment and s.c. dry needling (only needles and no anesthetic used) in the acupuncture and the individuality of points in the trigger point injections. In acupuncture the places of the needles are strictly determined by the acupuncture maps made for every disorder. In trigger point injections the injection places are decided individually without a fixed map according to the distribution of the hardest nearby places (Han et al 1997).

The factors associated with a failure of trigger point injection in pain treatment were analyzed by Hopwood et al (1994) and several factors could be found. Constant pain, pain associated with unemployment, no relief with analgesic medication, prolonged duration of pain and change in social activity were all found to affect the outcome of the treatment. Some of the reasons were connected to each other reflecting the multidimensional character of the sensing of pain. According to the authors the individuality of the reactions to the treatment might be explained with the complex character of pain.

7.8. Tinnitus and trigger points

Eriksson et al (1996) investigated trigger points in jaw and neck regions by tinnitus patients and controls. 104 people were examined (58 with tinnitus and 46 controls) and the muscles concerned were rectus capitis posterior minor and major, obliquus capitis superior and inferior, sternocleidomastoid, masseter and medial pterygoid. The tinnitus patients were found to have significantly more trigger points in the relevant musculature of the jaw and neck region compared to controls. However no clear-cut relationship between the presence of one or several trigger points and the incidence of tinnitus was found. 17 patients were offered massage and streching therapy and 10 of them had experienced some relief from tinnitus, the effect was noticed after the third treatment. All patients were also given a placebo ultra-sonic treatment, 2 of the patients benefited from the placebo treatment. The difference between the genuine and placebo treatment was statistically significant.

D. THE AIM OF THE STUDY

1. To evaluate the tinnitus character in nonselected primary care ENT patients.

2. To study the character of the tinnitus type which might be triggered by cervical muscular tension.

3. To test the effects of cervical muscle trigger point injection technique with lidocain on nonselected, voluntary primary care tinnitus patients.

4. To evaluate audiometrically treatment effects on tinnitus instantly after each treatment, after the series of treatments and long time treatment effects six months after the treatment period.

5. To compare the responders and non-responders to find out the possible candidates who could receive maximal benefit from the treatment.

E. MATERIAL AND METHODS

1.THE TIME OF THE RESEARCH AND THE SELECTION OF THE GROUP

The research was done in Imatra during 1991-1993, the last treatments started in 1993 and were completed in January 1994. During 1991-1993 all people searching medical help for their tinnitus were asked if they wanted to participate in a tinnitus survey. The nonwilling patients were treated in traditional ways with reassurance and sometimes with betahistine.

2. THE PARTICIPANTS OF THE STUDY

During the study period 228 attendants participated in the study and 10 of them approved to wait 6 months before the onset of treatment. Those 10 had their tinnitus measured once a month or at least three times in the pretreatment period. They were used as controls of the measurement technique and its reliability. The same tinnitus loudness than in the first visit could be measured from them every time during their monthly visits. The treated patients were given at least one treatment session and it was repeated at least once. 21 people did not complete the treatment series and they were all excluded from the study. After the treatment period all people were asked to come to an interview 6-24 months after the last treatment. During that time 2 patients had died and 3 were considered mentally unstable and one was suicidal and was in the treatment period in local psychiatric unit. 22 did not come to the interview for various reasons. Totally 178 people were included in the treatment group.

The selection for the treatment and control groups were decided by the patients. The control group consisted of 39 people with tinnitus, who were not treated. They were measured and examined like the treatment group and asked the same questions. No placebo treatments were accomplished because, with former experiments with pain treatment, needling of false points might aggravate the symptoms. NaCl injections are also used in pain treatment and they seem to act nearly as well as lidocain, so NaCl was not considered a proper placebo.

3. THE CHARACTER OF THE GROUPS

3.1.Age

The treatment group consisted of 178 people (104 female and 74 male). The youngest was 18 years and oldest 84 years, the mean age was 60.4 years.The control group consisted of 39 people with tinnitus (21 female and 18 male).The youngest was 30 years and oldest 77 years, the mean age was 58.9 years. The mean age of the whole group (treated and control group together) was 60 years. No significant difference was found comparing the age of patients between the groups using the Chi-Square test, p=0.16.

3.2. Diseases

3.2.1. General diseases and operations

Diseases that could affect the patients' well-being and the amount of tinnitus were asked. The listed general diseases were heart insufficiency, coronary artery disease, hypertonia, diabetes, epilepsy and cerebral arterial insufficiency. The listed operations were hand operations, shoulder operations, hip operations, leg operations, neck opertions (thyroidea included), head operations (tonsillectomy and other intraoral operations included) and back operations. No statistical differences concerning general diseases or operations between the treated group and the controls were found.

3.2.2. Degenerative and traumatic disorders

Degenerative diseases were reported by patients especially in the back (84 subjects) and neck (80 subjects). Fifty one patients reported degenerations in the knee, ankle or feet, 36 patients in the shoulder, 31 patients in the elbow, wrist or hand and 28 patients in the hip. 143 fractures or distorsions were reported, most of them in limbs, (66 subjects), followed by fractures or distortions in the skull (26 subjects), in other parts of the body (18 subjects), in facial bones (13 subjects), in lumbar or thoracal spine (14 subjects) and in cervical spine (11 subjects). A significant difference existed in the degenerative disorder in the cervical area, more degenerative disorders occured in the treated group, ($p < 0.01$). Similarly more degenerative disorders of the shoulder area were discovered in the treated group ($p < 0.01$). Both disorders were mostly bilateral.

3.2.3 Ear Diseases

29 subjects had had over 5 otitis media attacks. Chronic otitis media was reported by 4 subjects and 9 fenestroplastias had been accomplished. No statistical differences existed between the groups in infectious diseases or operations. Other diseases affecting hearing were reported by altogether 114 subjects, most common being a noise induced hearing loss (31 cases). All diseases affecting hearing were mostly bilateral (83 cases), but the unilateral cases were more numerous in the left ear (22 cases) than in the right ear (11 cases). The treated and control groups did not statistically differ with regard to ear diseases.

3.3 Working history

53 subjects had worked over 21 years in noisy circumstances, 26 subjects 11 to 20 years, 27 subjects 4 to 10 years and 12 subjects less than 3 years. The type of noise was mostly undefined (41 cases). The secondly common was industrial noise in a paper factory (38 cases), followed by industrial noise in metal industry (14 cases), other industrial noise (11 cases), lumber industry (2 cases), machine workshop noise (3 cases) and transportation (2 cases). No statistical difference existed between the treated groups and control patients concerning the working history.

3.4 General medication and tinnitus treatments

No statistical differences were observed between the groups in the use of drugs against other diseases than tinnitus.

Treatments to control tinnitus were questioned. A statistically significant difference existed between the treated and control groups, with regard to vasodilators, more in the control group ($p<0.01$), psychopharmacas tried but discontinued more in the control group ($p<0.05$), antihistamins more in the control group ($p<0.05$) and also more other medication ($p<0.01$) in the control group. Control group had tried more physiotherapy ($p<0.001$) and more analgesics($p<0.001$) in tinnitus treatment. No difference between the groups was found concerning the use of acupuncture or maskers.

3.5. Status

Table 2

STATUS

EAR STATUS				
Tympanic membrane	**Treated N/%**		**Control N/%**	
Colour	***Right ear***	***Left ear***	***Right ear***	***Left ear***
Transparent	103 / 58%	106 / 60%	29 / 74%	29 / 74%
Grey	67 / 38%	66 / 37%	10 / 26%	10 / 26%
Dark	3 / 2%	1 / 1%	0 / 0%	0 / 0%
Plaques	3 / 2%	3 /2%	0 / 0%	0 / 0%
Undefined	2 / 1%	2 / 1%	0 / 0%	0 / 0%
Movement				
Free	140 / 79%	141 / 79%	34 / 87%	35 / 90%
Stiff	34 / 19%	34 / 19%	4 / 10%	3 / 8%
Immobile	2 / 1%	1 / 1%	1 / 3%	1 / 3%
Undefined	2 / 1%	2 /1%	0 / 0%	0 / 0%
Position				
Normal	164 / 92%	164 / 92%	36 / 92%	34 / 87%
Retraction	8 / 5%	8 / 5%	1 / 3%	2 / 5%
Adhesive	2 / 1%	2 / 1%	1 / 3%	0 / 0%
Perforation	0 / 0%	1 / 1%	1 / 3%	0 / 0%
Pseudomembrane	3 / 2%	3 / 2%	0 / 0%	2 / 5%
Undefined	1 / 1%	1 / 1%	0 / 0%	0 / 0%

No statistical difference between the groups was found concerning the ear status.

4. THE EXAMINATION SCHEDULE

4.1. Medical examination

The otolaryngological examination was conventional. During the examination the muscle tone in the cervical and upper thoracic area was palpated with hands and classified. Both sides were palpated separately. The side with more trigger points in the muscles was classified having more tension. The selected trigger points must have felt harder than its surroundings and usually at least a little painful according to the searching instructions (Travell et al 1983).

The pure tone audiogram was taken by a trained audiology technician (Interacoustics Basic Diagnostic Audiometer Model AD 12 USA). Both the air conduction and the bone conduction were taken. The audiology technician did also all the tinnitus measurements. 2 trained interviewers carried out the questioning in the beginning of the treatment and once

again after the series were completed. The questions were asked in separate sessions and the treatment team was not present.

The patients were questioned about the level of tinnitus after every treatment by the treatment team. When they attended the subsequent treatment the possible change of tinnitus level during the interval was asked by the treatment team. The number of treatments varied depending on the treatment response. The non-responding group was adviced to discontinue the treatment after the second needling.

4.2. Tinnitus measurements

4.2.1. The measurement technique

The matching of tinnitus was first done by determining the pitch of tinnitus. It was done by presenting different tones by audiometer based on the audiogram and the subject's description of tinnitus. The "homing-in" of the final pitch was achieved by narrowing the scale. The loudness of the tinnitus was matched by using the closest pitch and matching the loudness of tinnitus in the frequency of the patient´s tinnitus. When the patient reported the same loudness twice, during the forking up and down the volume, it was taken as the level of tinnitus. When tinnitus was a noise, not a tone, masking narrow band noises were used in the matching. Tinnitus was always measured from the affected ear. In case of equal tinnitus in both ears the ear more convenient to the patient was used. Otherwise the ear with louder tinnitus was used or both sides were measured separately. Masking tests for tinnitus were not included in the searching plan because masking, by residual inhibition, might have affected the results.

4.2.2. The number of tinnitus measurements

The matching of tinnitus was performed in most cases at least before every treatment but it was tried to be accomplished also half an hour after the treatment every time. The VAS was asked before the treatment series but only the matching of tinnitus and the patient´s description of tinnitus were used in the follow-up. At least the most prominent sound was tried to be matched but in a case with another tinnitus sound efforts were made to measure it as well. Sometimes the other tinnitus sounds could not be measured because of their character or because they were intermittent. Because the amount of them was small the measurements of the 2 most prominent tinnitus sounds / place were used during the survey.

4.2.3. The classification of the measurements

The tinnitus measurements were classified according to the tinnitus sounds, not according to the subjects with tinnitus. The sites of tinnitus were: right ear, left ear and head. If two sounds were situated in the same place (for instance in the same ear), they were recorded tinnitus number 1 (*T1*) and tinnitus number 2 (*T2*), tinnitus 1 being louder. If the subject had bilateral tinnitus, the cases were classified as *T1* right and *T1* left even if tinnitus was identical in both ears. Thus one patient might at the most have 6 recordings, right ear tinnitus (right *T1* and *T2*), left ear tinnitus (left *T1* and *T2*)and head tinnitus (head *T1* and *T2*). The term head tinnitus included all the tinnitus sounds sensed in other place than ear, for instance in the occipital area or outside the skull. In that way the amount of happenings to tinnitus sounds is larger than the amount of people in the survey and larger than the amount of ears because one subject might be registered to three different "tinnitus *T1* cases".

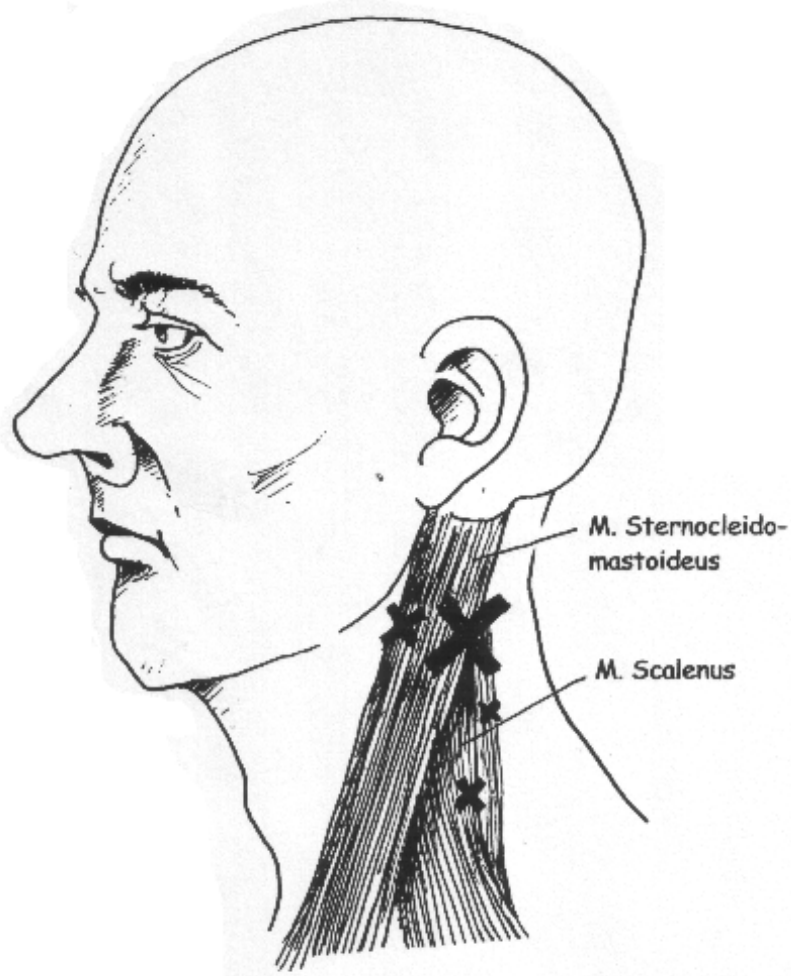


Fig. 2 The most used injection points, side view

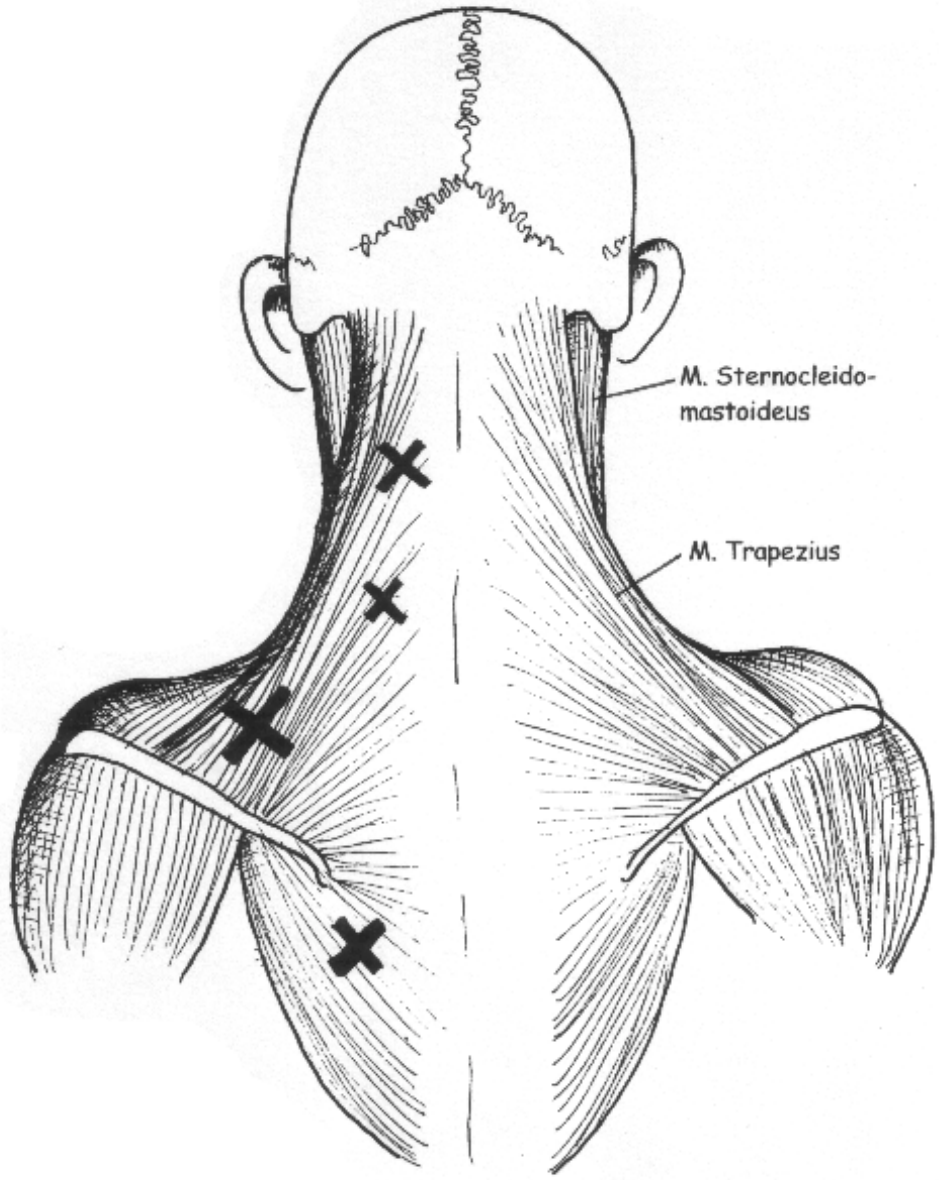


Fig. 3 The most used injection points, back view

5. THE TREATMENT SCHEDULE

5.1. The undertaking of the treatment

The treatment was accomplished with lidocain 1% solution (Lidocain, Orion). It was injected into the selected trigger points in the cervical and upper thoracic region. In case of several trigger points all of them were intended to be treated beginning with the worst one (the biggest and most painful) and proceeding to the weakest one. If the patient felt dizzy or could not tolerate the injections otherwise, the treatment was stopped. In those cases less points were used in one setting and more treatment settings were used. The contralateral trigger points were treated if they were prominent and the patient tolerated the treatment without side-effects. In every point about 0.1-1.0 ml of lidocain was injected. Depending on the number of detectable trigger points in the patient, during a session 1 to 16 points were treated (0.1 to 7.0 ml lidocain / subject per session). In the case of unilateral tinnitus the injections with lidocain solution were started from the ipsilateral side. Mostly injected muscles were sternocleidomastoideus, trapezius, levator scapulae, scalenus medius, splenius capitis, semispinalis capitis, supraspinatus, serratus anterior and long neck muscles. Before the injections Fluori-Methane solution (Gebauer Company, Cleveland, Ohio) was used to reduce the muscular tension in the muscles harboring most trigger points. The solution is used for treatment of myofascial pain, muscle spasms and restricted muscle motion. The mechanism is explained by the fact that a sudden drop of skin temperature might produce a temporary anesthesia by blocking the spinal stretch reflex and thus the sensation of pain in higher centers. The allowed decreased pain sensation enables stronger passive stretching than without the cooling (Han et al 1997).

Only 5 patients were treated more than 5 times (2.9 % of all the cases). The tinnitus matching was taken every time before the treatment and also half an hour after the treatment. The patients were also asked about tinnitus during the interval between the treatment settings and two weeks after the latest treatment. The mean number of treatments was 3.2, the number varied between 1 and 51. The number of patients in the presented 5 treatments are listed in table 3.

Table 3

NUMBER OF TREATMENTS

Number of treatments	1	2	3	4	5	6 or more
Number of patients	3	100	32	19	12	5

The treatment made some patients dizzy or light-headed, but others could stand a lot of it, so the amount of lidocain tolerated by the patient was used. In the first treatment 2 ml or less were used by 90.7 % of all the patients and the highest amount was 5 ml (table 4). The number of trigger points is presented in table 5.

Table 4

AMOUNT OF LIDOCAIN DURING TREATMENTS
(number of patients having treated with different amounts of lidocain)

	TREATMENT SESSIONS				
LIDOCAIN	**I**	**II**	**III**	**IV**	**V**
Less than 1ml	19	13	10	6	4
1-1.9ml	34	13	5	2	2
2-4ml	118	129	48	29	15
Over 4ml	1	15	9	1	1
Mean amount	1.8ml	2.8ml	2.9ml	2.3ml	2.4ml

Table 5

THE NUMBER OF INJECTED TRIGGER POINTS
(The number of patients/ treatment)

	TREATMENT SESSIONS				
POINTS	**I**	**II**	**III**	**IV**	**V**
0-1	19	8	8	4	3
2-5	69	40	14	12	2
6-9	56	74	32	11	13
10 or over	9	35	10	8	1

MEAN NUMBER OF INJECTED POINTS

	TREATMENT SESSIONS				
MEAN	**I**	**II**	**III**	**IV**	**V**
NUMBER	4.9	7	6.3	6.3	6

5.2. The series of treatments

Retreatment after the first setting was mostly performed 5 to 10 days from the first treatment. After the second treatment the schedule was stopped with the non-responders. Our earlier experiences seemed to prove that either tinnitus responds after two sessions or it will not respond at all. If tinnitus seemed to respond after two sessions, the treatment was continued first weekly and then once or twice a month as long as it was considered benefical. With complete disappearance of tinnitus, patients were informed to contact the clinic in case of relapsing tinnitus. In those cases the retreatment was tried to be accomplished as soon as possible. The treatment was also stopped when tinnitus became softer and after several sessions did not improve more. When treatments were stopped patients were informed to contact the clinic in case of aggravation and patients were retreated every time. The intervals differed quite much from each other according to the results obtained in the previous treatment. The long treatment intervals were due to cases of disappearing and reappearing tinnitus. The second treatment was undertaken when tinnitus reappeared and those cases interfere also the mean duration of treatment intervals.

Table 6

THE INTERVAL BETWEEN TREATMENTS
(number of patients with variation in the interval between treatments)

INTERVAL	TREATMENT INTERVAL (number of patients)			
Days	I-II	II-III	III-IV	IV-V
4-7	68	12	10	1
8-14	78	25	16	12
15-40	17	17	5	5
Over 40	8	21	13	6

MEAN INTERVALS BETWEEN TREATMENTS (DAYS)

MEAN DURATION	TREATMENT INTERVALS			
	I-II	II-III	III-IV	IV-V
Days	19.2	62.6	63.7	44.7

6. THE STATISTICS

Chi-square analysis was used in the comparision between the treatment and control groups and the responders and non-responders. The Student´s t-test was used for paired data. In searching the outcome variables logistic regression was used with chi-square analysis.

F. RESULTS

1. TINNITUS HISTORY

1.1. Duration of tinnitus

The duration of tinnitus varied in the treated and control groups. The duration of tinnitus by the first visit is listed in table 7. No statistical differences were found between the groups concerning the duration of tinnitus.

Table 7

DURATION OF TINNITUS
(Number of cases)

Duration	Treated	Controls
1day	1	
2-7 days	3	2
8-30days	23	5
1-3months	21	5
4-6months	19	5
7-12months	24	2
1-3years	28	8
4-10years	25	8
11or more years	20	3
Undefined	14	1

1.2. The site of tinnitus and the number of sounds

Tinnitus was recorded by the number of ears or places with tinnitus. The number of tinnitus sounds are presented in table 8 according to the place they were heard. Tinnitus sounds observed outside ears were classified “head”. The number of tinnitus sounds tended to be more prominent in the left ear than in the right ear in both treatment and control groups. In the whole group (treated and controls together) the number of tinnitus sounds in the left ear was statistically more prominent ($p<0.001$) than in the right ear. The treated and control groups did not statistically differ in the laterality and number of tinnitus sounds.

Table 8

THE NUMBER OF TINNITUS SOUNDS/ PLACE (NUMBER OF CASES)

NUMBER OF SOUNDS	RIGHT EAR		LEFT EAR		HEAD	
	Treated	Control	Treated	Control	Treated	Control
1 sound	100	23	121	28	10	3
2 sounds	15	3	25	2	1	0
3 sounds	6	0	4	1	2	0
4 or more	1	0	2	0	0	0
No sounds	55	12	26	6	163	36
Unknown	1	1	0	2	2	0

1.3. Description of tinnitus sounds

In the treatment group the most common description for tinnitus was a ringing sound, the T1 sound in the right ear was ringing in 51 subjects, in the left ear in 58 subjects and in the head in 4 subjects. Among T2 sounds ringing was the closest description in 3 right ears and 3 left ears. Second in order was chirping sound like a grasshopper in both ears.

In the control group ringing was the most common description of tinnitus, in the right ear in 10 subjects and in the left ear in 11 subjects. In head tinnitus whoosing was the most prominent, 2 out of three subjects had that kind of tinnitus. The list with all the descriptions of T1 and T2 are in table 9. No statistical differences existed between the treatment and control group in the description of tinnitus.

Table 9

THE DESCRIPTION OF TINNITUS BY WORDS (NUMBER OF CASES)

T1-SOUNDS

	TREATED SUBJECTS			CONTROL SUBJECTS		
	Right ear	**Left ear**	**Head**	**Right ear**	**Left ear**	**Head**
Ringing	51	58	4	10	11	0
Chirping	27	36	3	6	5	0
Whistling	22	24	4	2	5	2
Humming	10	15	2	2	3	0
Pulse	2	4	0	0	1	0
Rustling	0	0	0	0	0	0
Morse-like	0	1	0	0	0	0
Snapping	0	1	0	1	0	0
Hissing	0	0	0	0	0	0
Other	9	11	1	3	4	0
Undefined	0	0	1	1	2	1

T2-SOUNDS

	TREATED SUBJECTS			CONTROL SUBJECTS		
	Right ear	**Left ear**	**Head**	**Right ear**	**Left ear**	**Head**
Ringing	3	3	0	0	1	0
Chirping	1	5	0	1	1	0
Whistling	4	6	0	1	0	0
Humming	2	5	1	0	0	0
Pulse	0	0	0	0	0	0
Rustling	2	2	0	0	0	0
Morse-like	1	2	0	0	0	0
Snapping	2	1	0	0	0	0
Hissing	0	0	1	0	0	0
Other	6	7	0	0	1	0
Undefined	0	1	1	0	0	0

A difference between female and male attendants were discovered concerning descriptions of *T1* sounds. The most common *T1* description in the males was ringing in both ears but among the females the distribution of sounds was more even and chirping was the most prevalent characterization. In the whole group (treated and controls together) the difference between the description of *T1* sound according to different gender was statistically highly significant for both right ($p< 0.001$) and left ears ($p< 0.001$) but no differences in the head *T1* sounds were found. The difference between the female and male gender was statistically highly significant also in the treatment group *T1* sounds in right ($p< 0.001$) and left ears ($p< 0.001$) but not in the head *T1* sounds. No differences in the control group existed concerning to gender and description of the tinnitus sounds. No differences between the treatment group and the control group were found according to the description even when the both sexes were examined separately (table 10).

Table 10

THE DESCRIPTION OF TINNITUS COMPARED TO GENDER
(TREATED AND CONTROL PATIENTS)

	RIGHT EAR				**LEFT EAR**			
	MALE		FEMALE		MALE		FEMALE	
	N	**%**	**N**	**%**	**N**	**%**	**N**	**%**
Ringing	43	65,2	18	22,5	48	59,3	21	21
Chirping	12	18,2	21	26,3	14	17,3	27	27
Whoosing	9	13,6	15	18,8	13	16	16	16
Humming			12	15			18	18
Jumping			2	2,5	1	1,2	4	4
Snapping	1	1,5					1	1
Morse-like					1	1,2		
Rusting								
Other	1	1,5	11	13,8	4	4,9	11	11
Undefined			1	1,3			2	2
TOTAL	66		80		81		100	

1.4. The tinnitus sounds listed according to loudness

The distribution of tinnitus sounds between the ears showed left side preponderance. Their number in the left side was higher and so was their loudness. In the treatment group the loudest sound was 89 cases in the left ear, 59 cases in the right ear and in 24 cases equally loud in both ears or was sensed in the head, 6 cases were unclear. The second loudest sound was 47 cases in the left ear, 35 cases in the right ear and 10 cases it was bilateral or sensed in the head. The third sound was heard 7 cases in the right ear, 4 cases in the left ear and 3 cases bilaterally or in the head. The fourth sounds were more prominent in the left ear, 3 cases in the left ear, 2 cases in the right ear and 2 bilateral cases.

In the control group the tinnitus sounds distributed more evenly. The loudest tinnitus sound was 16 cases in the right ear, 15 cases in the left ear and 8 cases sensed either in the head or equally in both ears. The second loudest sound was 4 cases in the right ear, 11 cases in the left ear and 5 cases bilateral or sensed in the head. Third tinnitus sound occured only in 3 cases, 1 in the left ear, 1 bilateral case and in 1 case the place was unclear. The fourth sound was reported to exist in only 1 case but the site was unclear.

No statistically significant differences were found between the groups in the distribution of tinnitus sounds based on their loudness.

1.5. The behavior of tinnitus

1.5.1. Natural changes

Loudness fluctuations. In the whole group (treated and controls together) the loudness of tinnitus fluctuated on an everyday basis in 37.9% of right *T1* cases, in 43.7% of left *T1* cases and in 47.4% of head *T1* cases.

In the treatment group among subjects with one solitary *T1* tinnitus / site, 76 right *T1* cases, 80 left *T1* cases and 7 head sensed *T1* cases no loudness fluctuations existed. Loudness fluctuations were reported by 42 right *T1* cases, 64 left *T1* cases and 8 head *T1* cases. In some subjects 2 steady sounds (*T1* and *T2*) occurred / site, 2 cases in right ear and in 2 cases in the left.

In the control group with one solitary *T1* tinnitus / site in 14 right *T1* cases, 19 left *T1* cases and 2 head *T1* cases no fluctuations existed. Loudness fluctuations occured in 10 right *T1* cases, in 10 left *T1* cases and in 1 head *T1*. In 1 right ear 2 non-fluctuating tinnitus sounds (*T1* and *T2*) were reported and in 1 left ear 2 fluctuating sounds.

No statistically significant differences in the fluctuation of the loudness of tinnitus between the groups were found.

Spontaneous temporary disappearances In the both groups occasional spontaneous temporary disappearances of *T1* occured. In the whole study group 54.1% of right *T1*, 52.6% of the left *T1* and 37.6% of the head *T1* disappeared at least for minutes. *T2* sounds disappeared at least for minutes in 69.6% of right *T2* cases, in 63.6% of left *T2* cases and in 20% of head *T2* cases. Tinnitus was not heard all the time in 43.9% of right *T1* cases, in 44.2% of left *T1* cases and in 35.3% of head *T1* cases. Some of the disappearances might be due to external masking but *T1* disappearances for days was reported by 31.5% of right *T1* cases, 19.6% of left *T1* cases and 31.3% of head *T1* cases. Tinnitus disappearance was more common with second sounds, 46.7% of right *T2* cases and 57.7% of left *T2* cases had disappeared for days, but no head *T2* disappearances for days had occurred. No statistical difference could be found between the groups in the spontaneous temporary disappearances of the tinnitus sounds.

1.5.2. The influence of head position

The influence of head position changes on the behaviour of tinnitus was questioned. The effect of turning head to the left and right, to up and down and sideways ear to the shoulder in both sides on the fluctuation of tinnitus was asked. Both aggravation and improvement were reported. T1 was hearable only in a certain head position in three ears. The number and character of changes are listed in table 11.

No statistical differences were found according to the influence of head position changes on tinnitus fluctuations between the groups.

Table 11

THE EFFECT OF HEAD MOVEMENTS ON TINNITUS
(The number of cases reporting fluctuations of T1 or T2)

	TREATED SUBJECTS			CONTROL SUBJECTS		
TURNING	**Right ear**	**Left ear**	**Head**	**Right ear**	**Left ear**	**Head**
Head right, ear to						
Shoulder						
No effect	114	136	14	20	26	2
Increase	4	5	0	2	1	0
Decrease	1	1	0	0	1	0
Head left, ear to						
Shoulder						
No effect	111	136	12	19	27	2
Increase	6	7	0	1	1	0
Decrease	2	2	0	0	1	0
Head in front						
No effect	115	137	12	19	24	2
Increase	5	7	0	1	1	0
Decrease	1	1	0	0	2	0
Head back						
No effect	116	141	14	20	26	2
Increase	4	4	0	0	0	0
Decrease	0	0	0	0	1	0
Head sideways						
to right						
No effect	116	137	12	21	27	2
Increase	5	4	0	0	0	0
Decrease	1	2	0	1	1	0
Head sideways						
to left						
No effect	112	136	14	21	27	2
Increase	7	3	0	0	0	0
Decrease	1	2	0	0	0	0

1.5.3. The influence of stress

Physical stress affected the loudness of tinnitus (either *T1* or *T2* or both) in nearly half of the cases (44% in right *T1* and 43% in left *T1* in the treatment group and 48% in right *T1* and 45% in left *T1* in the control group). Psychological stress affected tinnitus (*T1*, *T2* or both) in fewer cases (27% in right *T1* and 21% in left *T1* in the treatment group and 28% in right *T1* and 27% in left *T1* in the control group). Staying awake affected more than one third (37% in right *T1* and 34% in left *T1* in the treatment group and 32% in right *T1* and 30% in left *T1* in the control group). The effects are listed in table 12.

No statistical differences between the groups were found concerning the influence of physical or psychological stress or staying awake on tinnitus loudness .

1.5.4. The temperature changes

About one tenth of cases had some fluctuations of tinnitus (*T1, T2* or both) with outside temperature changes or changes with weather type (14% in right *T1* and 14% in left *T1* in the treatment group and 4% in right *T1* and 3% in left *T1* in the control group). The effects are listed in table 12. No statistically significant differencies concerning the influence of temperature changes were found between the groups.

Sauna affected a little more than 15% of cases causing fluctuations of tinnitus (*T1, T2* or both) (19% in right *T1* and 17% in left *T1* in the treatment group and 16% in right *T1* and 19% in left *T1* in the control group). The effects of sauna are presented in table 12. No statistical differences between the groups concerning the influence of sauna were encountered.

Table 12

THE EFFECT OF PHYSICAL AND PSYCHOLOGICAL PHENOMENA ON TINNITUS LOUDNESS
(The number of cases reporting fluctuations of T1 or T2)

	TREATMENT SUBJECTS			CONTROL SUBJECTS		
	Right ear	**Left ear**	**Head**	**Right ear**	**Left ear**	**Head**
PHYSICAL STRESS						
No effect	62	79	10	13	16	2
Increases	49	57	2	12	14	0
Decreases	6	8	1	0	0	0
PSYCHOLOGICAL						
STRESS						
No effect	80	107	6	16	17	1
Increases	33	37	4	7	9	1
Decreases	0	0	1	0	0	0
SAUNA						
No effect	96	119	9	16	19	3
Increases	11	16	1	3	5	0
Decreases	12	10	1	0	0	0
TEMPERATURE						
CHANGES						
No effect	98	123	6	18	23	3
Increases	11	13	3	1	1	0
Decreases	6	2	1	0	0	0
STAYING AWAKE						
No effect	71	90	6	13	16	3
Increases	46	55	5	8	9	0
Decreases	0	0	0	0	0	0

1.6. Drugs and tinnitus

The subjects were questioned about the effect of drugs on tinnitus (*T1*, *T2* or both). In the whole group (both treated and controls) antihypertensive medication caused an increase of tinnitus in 1 case and a decrease in 2 cases. Vasodilative agents decreased tinnitus in 2 cases. Betahistidine caused decrease of tinnitus in 17 cases and increase in 3 cases. Antidepressants caused a decrease of tinnitus in 9 cases. With sleeping pills the reduction of tinnitus happened in 6 cases, aggravation in 2 cases. Alcohol decreased tinnitus in 31 cases and increased it in 16 cases. Taking coffein caused 3 decreases of tinnitus and 15 increases of tinnitus.

No statistical differences between the treated and control groups were found concerning the influence of analgesics, antihypertensive medication, vasodilative agents, betahistidine, antidepressants, sleeping pills, alcohol and coffee, tea or cola-drinks on tinnitus.

1.7.The potential etiology of tinnitus

The patients were asked their own opinion about the factors that might have affected their tinnitus.

1.7.1. Ear disorders

In the treatment group otitis episode was a cause of tinnitus in 1 case, in 10 cases otitis was considered a possible cause in tinnitus generation. Ear operation was a cause of tinnitus in 1 case and a possible cause in 1 case. Noise trauma was reported to be a cause of 15 tinnitus cases and a possible cause in 20 cases. Other reasons for hearing loss were a cause of tinnitus in 2 cases and a possible cause in 1 case. Explosive injury induced hearing loss was reported as a cause of tinnitus in 5 cases.

In the control group an otitis media was considered a possible cause of tinnitus in 1 case. Noise trauma was reported as a cause for the outbreak of tinnitus in 2 cases and as a possible cause for tinnitus in 5 cases. Other reasons for hearing loss were a cause in 1 case.

No statistical differences were found concerning patients´opinions of otological causes of tinnitus between the treated and control groups.

1.7.2. General disorders

In the treatment group a general disease (arthritis reumatoides, epilepsy, diabetes, respiratory illness etc) was considered to be a possible cause of tinnitus in 1 case, hypertonia in 1 case, an accident in 7 cases, medication in 2 cases, other than ear operations in 1 case and stress in 2 cases.

In the control group no cases of tinnitus occured by a general disease, by other than ear operations, hypertonia or medication. An accident as a cause of tinnitus was reported in 1 case and stress in 1 case.

No statistical differencies were found between the treated and control groups concerning the patient´s opinion of tinnitus generated by general disorders.

1.7.3. Local disorders

In the treatment group the muscular tension in the neck area was considered a possible cause of tinnitus in 4 cases, dental malocclusion in 1 case. In the control group the muscular tension in the neck area was considered a possible cause of tinnitus in 2 cases.

No statistical difference was found between the treated and control groups according to the patient´s opinion of tinnitus generated by local disorders.

2. TINNITUS RELATED ANNOYANCE

2.1. Subjective hypacusis

Subjects were asked about their subjective hearing with a questionnaire. In the treatment group no hearing difficulty was reported in the right ear in 43 % of cases and in the left ear in 41% of cases. A severe or very severe hearing problem occured in the right ear in 11 % of cases and in the left ear in 14% of cases.

In the control group subjective normal hearing was experienced in the right ear in 39 % of cases in the left ear in 39 % of cases. A severe or very severe hearing difficulty was reported in little over 40 % of cases in both ears.

Significantly more subjects experienced slight hearing problems in the right ear in the treated group when compared to the control group. In the control group more severe hearing problems were reported, the difference between the groups was statistically highly significant ($p < 0.001$). The similar kind of difference could be detected also in the left ear, more subjects experienced slight hearing problems in the treated group and more severe hearing problems in the control group, the difference between the groups was statistically highly significant ($p < 0.001$).

2.2. Headache

The headaches situated mostly all over the head and not in the side. In the treatment group 96 patients considered their headache to be situated all over the head, in 10 cases it was mostly in the right side and in 11 cases mostly in the left side, the rest, in 54 cases no headaches existed. In the control group 19 patients considered similarly their headache to be situated mostly all over the head, in 3 cases it was mostly in the right side and in 3 cases in the left side, in 13 cases no headaches existed. No statistically significant difference was found considering the headache between the groups.

2.3. Vertigo

In the treated group 2 subjects had constant vertigo. Vertigo attacks occured several times a month in 29 subjects, a few times a year in 60 subjects and in 12 subjects less frequently than once a year. In the control group 1 subject had constant vertigo. Vertigo attacks occurred several times a month in 5 subjects, in 16 subjects a few times a year and in 3 subjects less frequently than once a year. No statistically significant difference was found considering about the vertigo between the groups.

2.4. Sensations of the ear (pressure and fullness)

In the treated group pressure in the ear occured in the right ear in 30 cases and in the left ear in 34 cases, in 1 case it was present all the time, others were intermittent. Fullness of the ear occured in the right ear in 44 cases and in the left ear in 52 cases but constantly in only 2 left ears, others were intermittent.

In the control group pressure occured in 4 cases in the right ear and in 5 cases in the left ear but constantly only in 1 case. Fullness of the ear had been experienced in 10 cases in the right ear and in 13 cases in the left ear, constantly in the right ear in 1 case and in the left ear in 1 case.

A statistically significant difference was found between the groups concerning pressure in the right ear, being more frequent in the treatment group ($p<0.005$). The difference was not significant in the left ear. No statistical difference existed with fullness of the ears between the groups.

2.5. Distortions of hearing

In the treated group diplacusis had been experienced in the right ear in 30 cases and in the left ear in 27 cases (constantly in the right ear in 1 case and in the left ear in 1 case). Hyperacusis was reported in the right ear in 65 cases and in the left ear in 62 cases (constantly in the right ear in 2 cases and in the left ear in 1 case). Hearing distortion was reported in the right ear in 23 cases and in the left ear in 26 cases (constantly in one case in the left ear).

In the control group diplacusis was reported in the right ear in 9 cases and in the left ear in 7 cases (constantly in 1 case in the right ear). Hyperacusis was experienced in the right ear in 22 cases and in the left ear in 20 cases (constantly in 1 case in the right ear and 1 in the left ear). Hearing distortion was reported in 9 cases in the right ear and in 9 cases in the left ear (constantly in the right ear in 1 case and in the left ear in 1 case).

No statistical difference was found concerning distortions of hearing or hyperacusis between the groups.

3.THE MUSCULAR STATUS

3.1. The neck muscles

In the treated group muscular tension was stronger in the right side of the neck than in the left side of the neck in 28 % of the subjects; stronger in the left side of the neck than in the right side of the neck in 33 % of the subjects and muscular tension was equal in both sides in 30 % of the subjects.

In the control group muscular tension was stronger in the left side of the neck than in the right side of the neck in 23 % of the subjects; stronger tension in the right side of the neck than in the left side of the neck in 8 % of the subjects and in 28 % of the subjects equal muscular tension in both sides of the neck was found.

No statistical difference was found in the sidedness of the muscular tension in the neck between the groups.

3.2. The shoulder muscles

In the treated group in 26 % of the subjects the muscular tension was stronger in the right shoulder area than in the left shoulder area, in 20 % of the subjects the muscular tension was stronger in the left shoulder area than in the right shoulder area and in 36 % of the subjects equal muscular tension in both sides occured.

In the control group in 26 % of the subjects the muscular tension was stronger in the left shoulder area than in the right shoulder area, in 10 % of the subjects the muscular tension was stronger in the right shoulder area than in the left shoulder area and equal tension in both sides was detected in 26 % of the subjects.

No statistical difference was found in the muscular status of the shoulder area between the groups.

3.3. The scapular muscles

In the treated group muscular tension around the scapula was stronger in the right side than in the left side in 15 % of the subjects, stronger in the left side than in the right side in 12 % of the subjects and sides were considered equal in 13 % of the subjects.

In the control group muscular tension around the scapula was stronger in the left side than in the right side in 13 % of the subjects and stronger in the right side than in the left side in 5 % of the subjects, equally tensive in both sides were 5 % of the group.

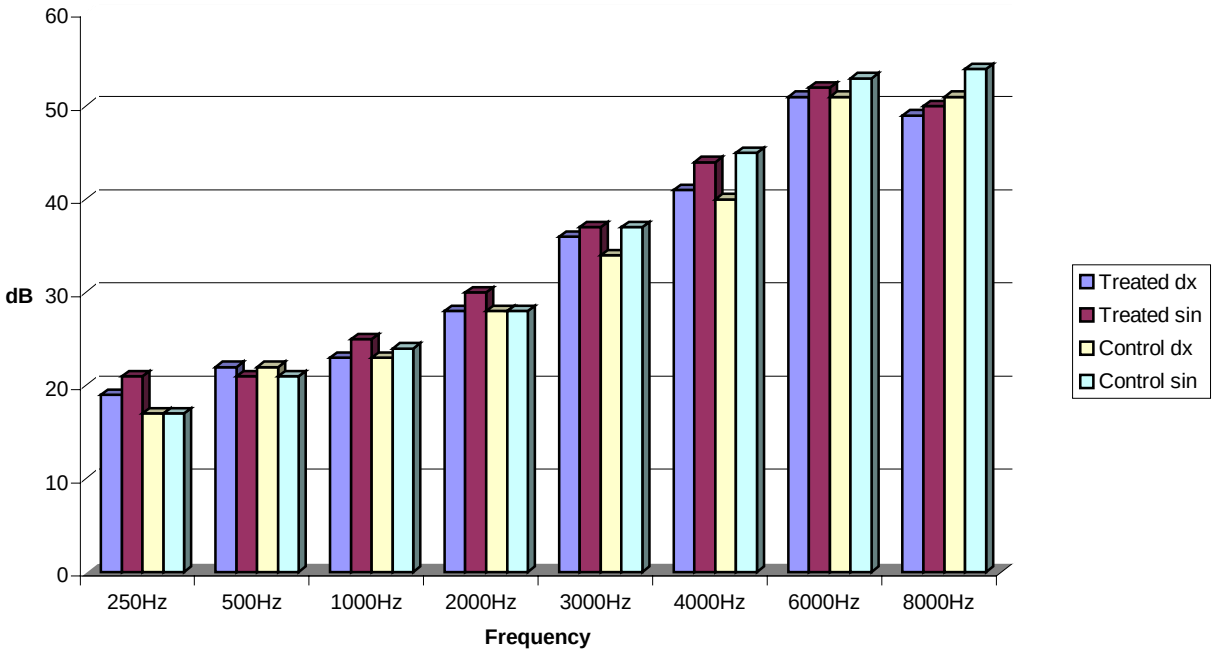
No statistically significant difference was found concerning the muscular tension in the scapular area between the groups.

4. HEARING MEASUREMENTS

The audiometric measurements were done to everyone. The results of audiological examinations are presented in fig.4.

No statistically significant differences between the treated and control groups were found concerning hearing.

Fig.4 MEAN HEARING (dB) IN TREATED AND CONTROLS



5. TINNITUS MEASUREMENTS

5.1. Audiometrically

Tinnitus was matched with the help of an audiometer. In the treated group the matching was impossible in right *T1* in 3 % of the cases and in left *T1* in 3 % of the cases. In addition it was not done in right *T1* in 25 % of the cases and in left *T1* in 25 % of the cases. Most of these unmatched cases were having a more prominent tinnitus on the other side or the tinnitus was heard bilaterally and it was measured only in one side. Finding the right sound in matching of *T1* was considered easy by the patient in 38 % of the cases right *T1* and in 50% the left *T1*. In 1% of the cases with right ear tinnitus and in 6 % of the cases with left ear tinnitus two sounds existed and both *T1* and *T2* were considered easy to match by the patient. In 29 % of the cases no tinnitus in the right side and in 14% no tinnitus in the left side existed. The recordings are presented in table 13 where they are in absolute values.

In the control group the matching was impossible with right *T1* in 5 % of the cases. Matching was considered easy by the patient in right *T1* in 37 % of the cases and in left *T1* in 74% of the cases. Matching was considered easy by the patient with both *T1* and *T2* in the right ear in 5 % of the cases. Measurement was not done in 16 % of right *T1* and 15% of left *T1*. In 41 % of the cases no right-sided tinnitus and 10% no left sided tinnitus existed. The tinnitus matchings of the control group are presented in table 13 in absolute values.

No statistical difference was found in the audiometric matchings of tinnitus frequency, volume or the easiness of the measurements between the groups.

Table 13a

MEASURED TINNITUS IN TREATED AND CONTROLS (*T1*)

MEASURED VOLUME OF TINNITUS

MEASURED TINNITUS (dB)	NUMBER OF CASES TREATED		CONTROLS	
	Right ear	Left ear	Right ear	Left ear
5dB	2		1	
10dB	5	4		7
15dB	3	7	2	2
20dB	5	8	2	4
25dB	6	3	1	1
30dB	8	5		1
35dB	5	5	1	1
40dB	6	10		2
45dB	3	11	1	1
50dB	3	8	1	
55dB	4	7	2	
60dB	1	3		2
65dB	2	2	2	1
70dB	7	6	1	3
75dB	3	4		1
80dB	4	6		
85dB	1	4	2	
90dB			1	
95dB	1			
100dB	1			
TOTAL	70	93	17	26

Table 13b

MEASURED TINNITUS IN TREATED AND CONTROLS (*T1*)

MEASURED FREQUENCY OF TINNITUS

MEASURED TINNITUS (Hz)	NUMBER OF CASES			
	TREATED		CONTROLS	
	Right ear	Left ear	Right ear	Left ear
250Hz	6	11	1	5
500Hz	7	11	1	5
1000Hz	4	8	1	1
2000Hz		5		2
3000Hz	8	9	3	2
4000Hz	12	15		2
6000Hz	15	15	5	3
8000Hz	17	18	7	5
TOTAL	69	92	18	25

Table 13c

MEASURED TINNITUS IN TREATED AND CONTROLS (T1)

	TREATED		CONTROLS	
TINNITUS	Right ear	Left ear	Right ear	Left ear
Mean volume (dB)	43	46	47	33
Mean frequency (Hz)	4450	3774	5375	3230
Median volume (dB)	40	45	50	23
Median frequency (Hz)	4000	4000	6000	2000

5.2. VAS-scale

The patients were asked to estimate the loudness of all their tinnitus sounds in a VAS scale from 0 to 10; 0 meaning no tinnitus and 10 meaning extremely loud tinnitus. The loudest tinnitus sensed by the subject was always *T1* in that site. The second loudest tinnitus was a tinnitus sound classified softer than the loudest tinnitus by the subject. The second tinnitus was mostly *T1* sensed in another site but it could also be *T2* in the same site than the loudest tinnitus. The third and fourth tinnitus were softer tinnitus sounds either in the same site than previous sounds or sensed in another site. The mean loudness of the loudest sensed tinnitus of the subject in VAS was 5.5 in the treated group and 5.2 for the control group. The mean VAS of the second loudest sound was 3.8 in the treatment group and 4.0 in the control group. The mean VAS of the third loudest sound was 3.4 in the treatment group and 3.5 in the control group. The VAS of the fourth loudest sound was 3.0 in the treatment group and 6 in the control group. The VAS scaling for all 4 tinnitus sounds are presented in table 14. No difference was found in the VAS scales between the groups.

Table 14

TINNITUS LOUDNESS OF ALL FOUR TINNITUS SOUNDS WITH VAS
(Number of cases)

	LOUDEST		SECOND		THIRD		FOURTH	
VAS	**Treated**	**Controls**	**Treated**	**Controls**	**Treated**	**Controls**	**Treated**	**Controls**
1	1		9	1	1		1	
2		2	13	3	3		2	
3	14	4	18	4	3	1	1	
4	37	6	27	4	5	1	2	
5	41	14	15	6	3		1	
6	28	2	8	2				1
7	39	5	3					
8	16	5	2	1				
9	1							
10	1							

6.THE EFFECT ON TINNITUS DURING THE TREATMENT PERIOD

6.1. The subjective effects

6.1.1. The subjective effect of the treatment on the feeling of tinnitus

The treated subjects had to estimate the treatment effect after every treatment. After the first treatment tinnitus improved in right *T1* in 50 % of the 109 cases, in left *T1* in 49 % of the 136 cases and in head *T1* in 33 % of the 15 cases. After the second treatment tinnitus improved in right *T1* in 43 % of the 106 cases, in left *T1* in 48 % of the 128 cases and in head *T1* in 27 % of the 15 cases.

After the third treatment tinnitus improved in right *T1* in 61 % of the 46 cases, in left *T1* in 49 % of the 57 cases and in head *T1* in 36 % of the 11 cases. In the fourth treatment tinnitus improved in right *T1* in 54 % of the 24 cases, in left *T1* in 60 % of the 30 cases and in head *T1* in 38 % of the 8 cases. After the fifth treatment tinnitus improved in right *T1* in 60 % of

the 15 cases, in left *T1*in 47 % of the 15 cases but none of the 3 cases with head *T1* considered any improvement.

6*.1.2. The effect of the treatment on the character of tinnitus*

In the first treatment *T1* sound was sensed “softer in character” after treatment in 26 % of the cases with right *T1*, in 31 % of the cases with left *T1* and in 20 % of the cases with head *T1*; however in 3 % of the cases with right *T1* and in 2 % of the cases with left *T1* a brighter tinnitus sound was experienced. A few solitary changes in the character of tinnitus *T2* sounds occured also, mostly softening.

In the second treatment softening occured in 24 % of the cases with right *T1*, in 25 % of the cases with left *T1* and in 13 % of the cases head *T1*. The tinnitus brightened in 2 % of the cases with right *T1* and in 6 % of the cases with left *T1*. The changes in the tinnitus character are presented in fig.5 and 6.

Fig.5 CHANGE OF TINNITUS CHARACTER (T1) RIGHT EAR

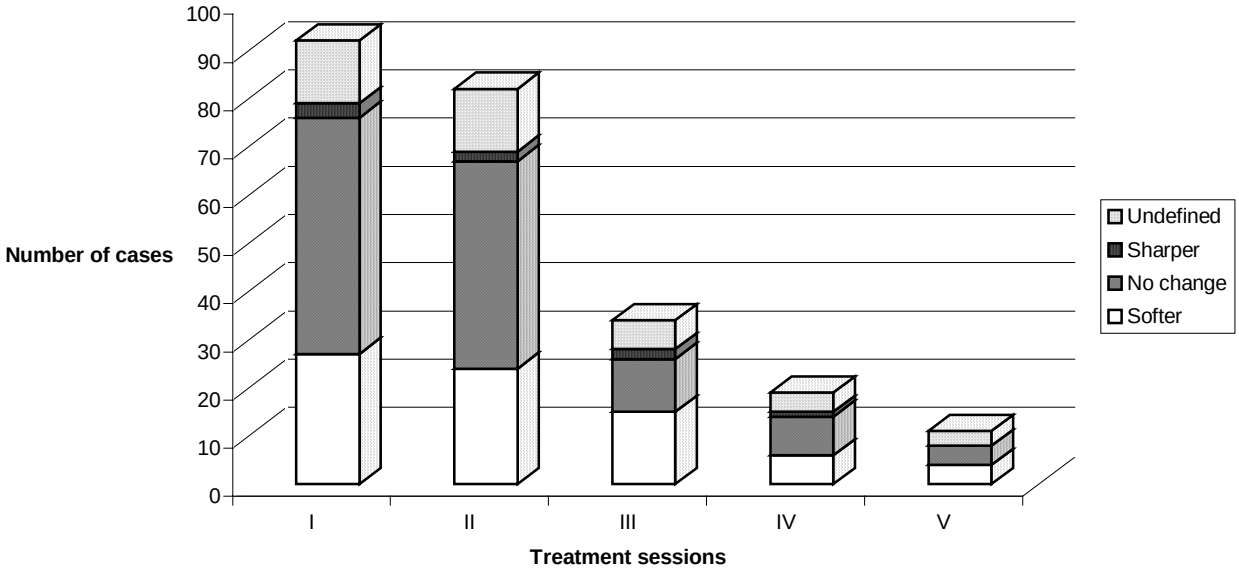
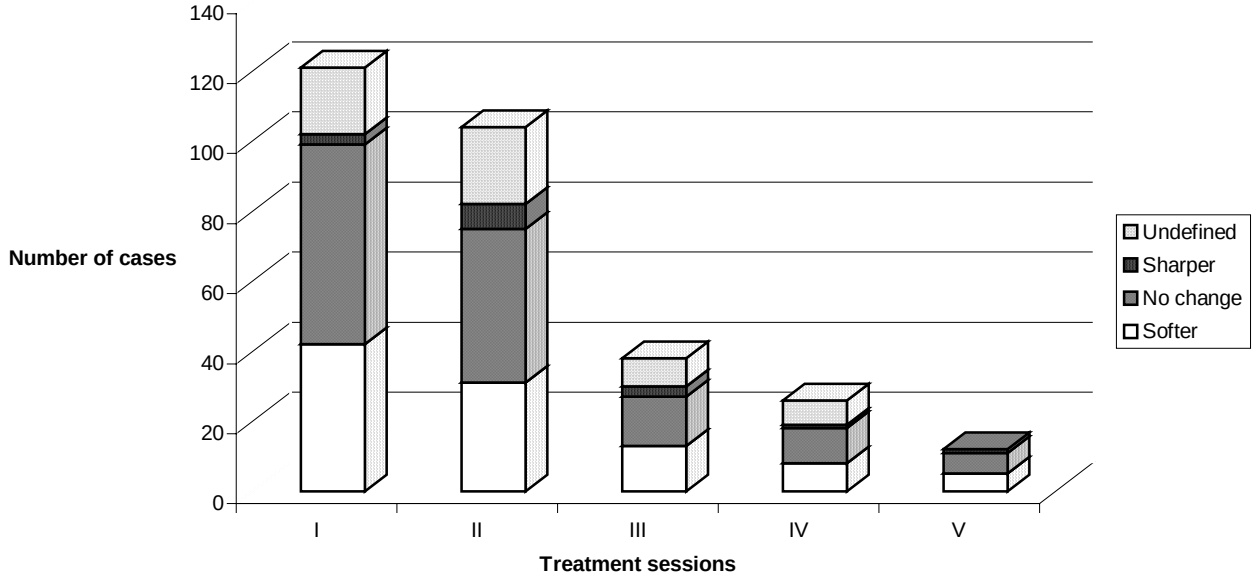


Fig.6 CHANGE OF TINNITUS CHARACTER (T1) LEFT EAR



6.1.3. The effect on the site of tinnitus

The site where tinnitus was sensed changed several times during the sessions. 10 times tinnitus sensed in the left ear (*T1*) moved to be heard in the head. In 4 cases right *T1* moved to to be sensed in the head. Left *T1* moved in 3 cases to be heard in the right ear and 2 right *T1* cases changed to be heard in the left ear. Head *T1* moved in 3 cases from the head to be sensed in the left ear. In 1 case tinnitus sensed outside the head changed from an undefined outside tinnitus sound to be heard in the right ear. In 1 case outside tinnitus moved to be sensed in the head. In 3 cases tinnitus moved to be sensed outside the head, in 1 case it was originally right *T1*, in 1 case left *T1* and in 1 case head *T1*.

6.2. The objective measurements of tinnitus during the treatment period

6.2.1. The changes in loudness

The number of loudness changes. The effect of the treatment was also measured by matching tinnitus. In the first treatment tinnitus disappeared or diminshed at least 10 dB from the pre-treatment value during the 30 minutes after the treatment with right *T1* in 36 % of the cases, with left *T1* in 36 % of the cases and with head *T1* in 33 % of the cases. The loudness changes from the treated and measured group during the treatment periods are presented in percents in fig.7 and 8.

Because the treatment effect was not complete within half an hour, patients were also asked to report the effects after that. Between the first and second treatment tinnitus disappeared or diminshed with right *T1* in 51 % of the cases with left *T1* in 44 % of the cases and with head *T1* in 43 % of the cases. The post-treatment effects during the treatment periods are presented in percents in fig. 9 and 10.

In the second treatment *T1* disappeared or diminshed at least 10 dB in 33 % of the cases with right *T1*, in 44 % of the cases with left *T1* but in no cases with head *T1*. After the measurements, tinnitus disappeared or dimished within two weeks after the second treatment

in 46 % of the cases with right *T1*, in 44 % of the cases with left *T1* and in 50 % of the cases with head *T1*.

In the third treatment tinnitus disappeared or diminshed at least 10 dB with right *T1* in 28 % of the cases, with left *T1* in 18 % of the cases and in no cases with head *T1*. During the post-treatment time after the third treatment *T1* sounds diminshed or disappeared in 38 % of the cases with right *T1*, in 41 % of the cases with left *T1* and in 40 % of head *T1*.

In the fourth treatment tinnitus disappeared or diminshed at least 10 dB with right *T1* in 24 % of the cases, with left *T1* in 35 % of the cases and with head *T1* in no cases. After the fourth treatment tinnitus diminshed or disappeared in 40 % of the cases with right *T1*, in 39 % of the cases with left *T1* and in 43 % of the cases with head *T1*. In the fifth treatment tinnitus disappeared or diminished at least 10 dB in 39 % of the cases with right *T1* and 14 % of the cases with left *T1*. After the fifth treatment tinnitus disappeared or diminished in 39 % of the cases with right *T1* and in 43 % of left *T1*.

Fig.7 TREATMENT RESULTS IN 30 MINUTES (T1) RIGHT EAR

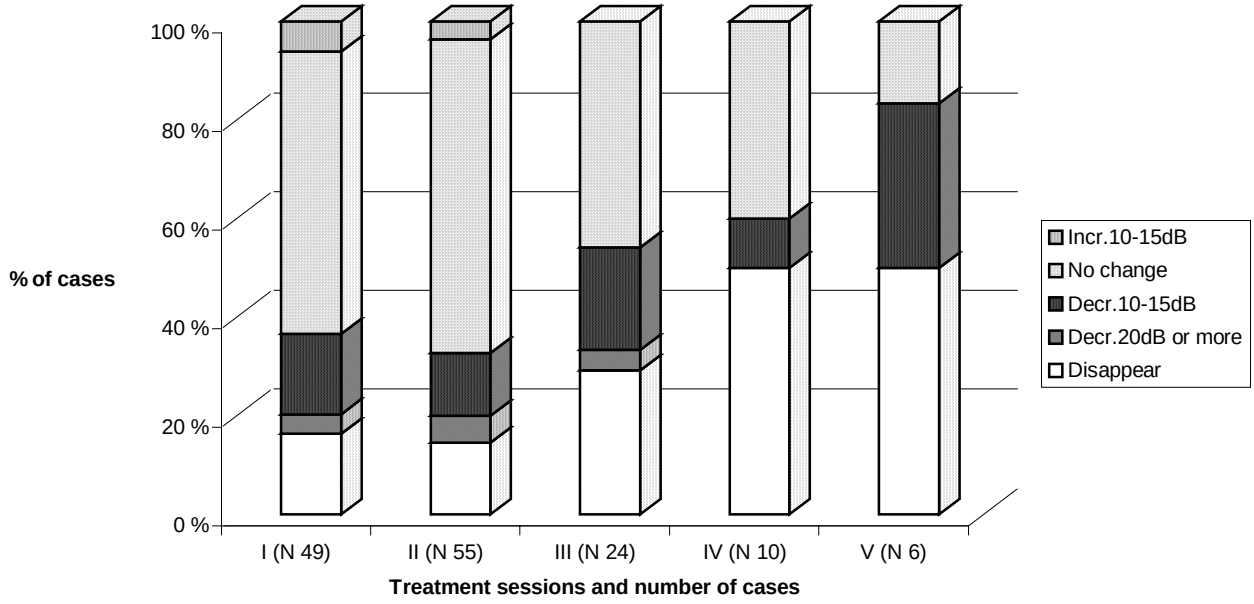


Fig.8 TREATMENT RESULTS IN 30 MINUTES (T1) LEFT EAR

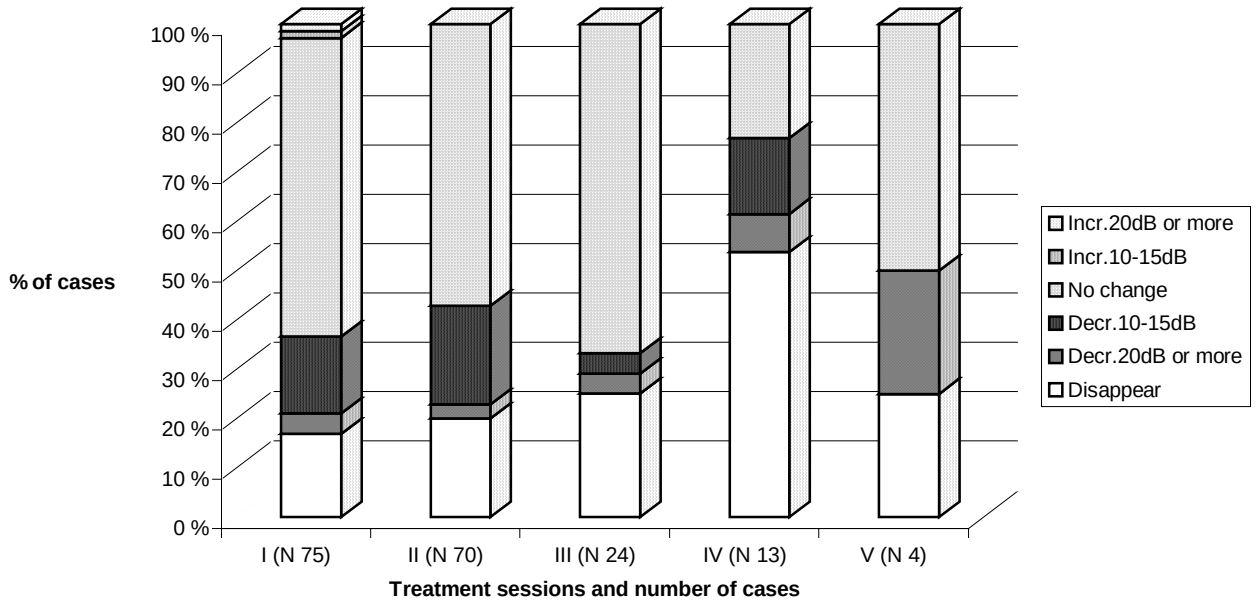


Fig.9 DELAYED TREATMENT RESULTS (T1) RIGHT EAR

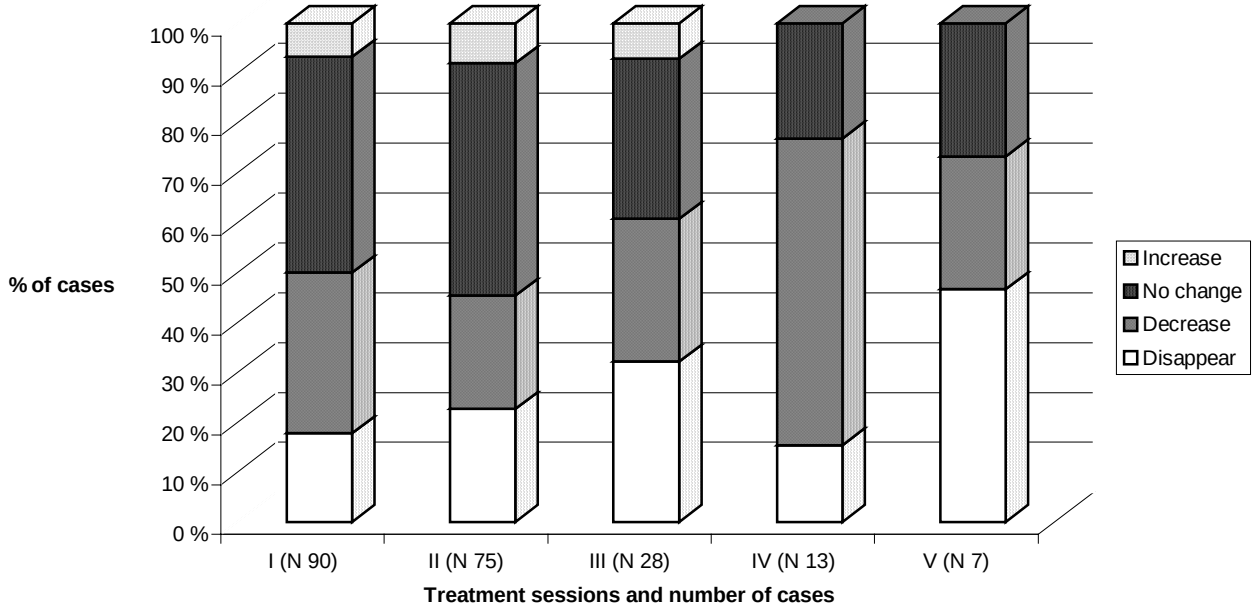
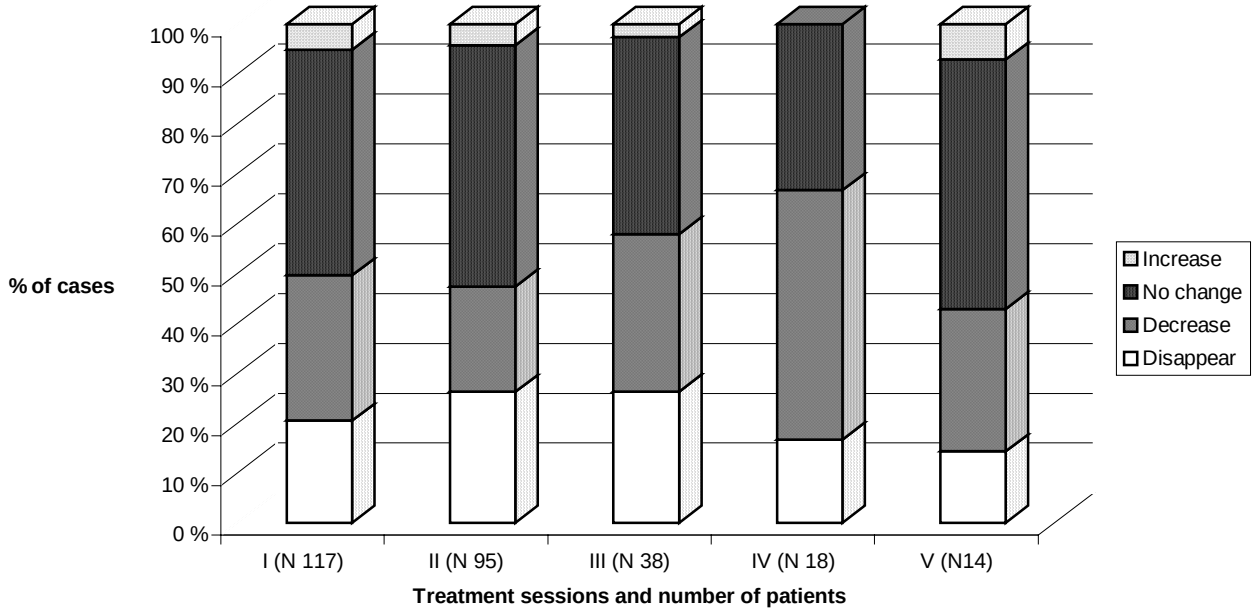


Fig.10 DELAYED TREATMENT EFFECTS (T1) LEFT EAR

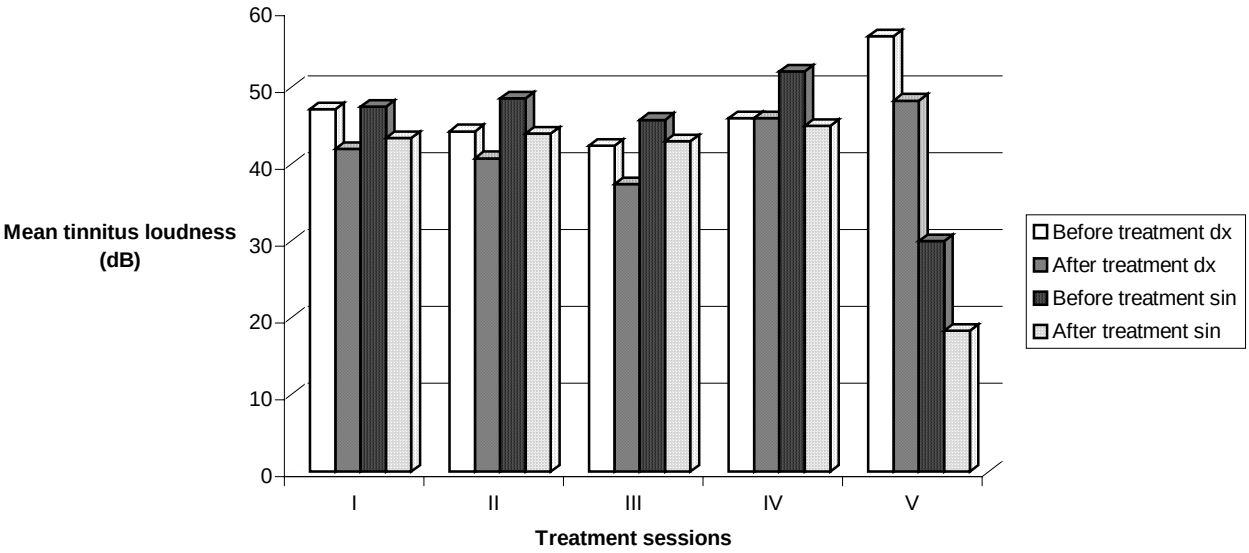


The volume of loudness changes. The loudness of tinnitus measured before the treatment period was compared to the one measured 30 minutes after the first treatment setting. The pretreatment mean loudness of right *T1* was 47.2 dB and posttreatment 42.0 dB ($p < 0.01$). The pretreatment mean loudness of left *T1* was 47.5 dB and posttreatment 43.4 dB ($p < 0.001$). The head *T1* sounds could not be compared because of scanty amount of cases in any treatments and the same problem existed with *T2* sounds. The mean loudness changes during the treatment period are presented in fig.11.

In the second treatment the mean pretreatment right *T1* was 44.3 dB and posttreatment *T1* 40.8 dB ($p< 0.01$). The mean pretreatment left *T1* was 48.6 dB, posttreatment *T1* 44.0 dB ($p< 0.001$). The mean pretreatment right *T1* and left *T1* values in the second treatment did not statistically differ from the pretreatment right *T1* and left *T1* values in the first treatment.

In the third treatment the mean pretreatment right *T1* was 42.4 dB and posttreatment *T1* 37.4 ($p< 0.05$). The mean pretreatment left *T1* was 45.8 dB and posttreatment *T1* 43 dB, (n.s). The mean pretreatment *T1* of the third treatment compared to the mean pretreatment *T1* of the second treatment differed statistically with right *T1* ($p< 0.01$) but not with left *T1*. In the fourth treatment no change in the volume of pre- and posttreatment loudness existed in right T1, it was 46 dB all the time. The mean value of the pretreatment left *T1* was 52.1 and posttreatment loudness 45 dB ($p< 0.05$). In the fifth treatment no significant changes existed partly due to the small amount of patients.

Fig.11 EFFECT OF TREATMENT TO MEAN TINNITUS (T1, dB)



The patients with repeatedly disappearing tinnitus. At the first treatment 8 cases of right *T1* disappeared during the 30 minutes of the session and in addition 16 cases of right *T1* ceased during the interval between the first and second treatment, (meaning 24 cases of right *T1* disappearances after the first treatment). Tinnitus usually reappeared after various intervals. At the second treatment 8 cases of right *T1* disappeared instantly and 17 cases later on during the week (altogether 25 right *T1* disappearances). At the third treatment 7 cases of right *T1* disappeared instantly and 9 cases later (altogether 16 right *T1* disappearances). At the fourth treatment 5 cases of right *T1* disappeared instantly and 2 cases later (altogether 7 right *T1* disappearances). At the fifth treatment 3 cases of right *T1* disappeared in 30 minutes and 3 cases later (altogether 6 right *T1* disappearances).

Right *T1* disappeared at least in two subsequent sessions in 9 cases (either at the treatment session or later on in the week). *T1* reappeared in those cases after various intervals but in the next session it ceased again after the treatment. In 2 cases the disappearance of right *T1* occured in three subsequent treatments. In 1 case the disappearance of tinnitus was repeated 5 times consecutively, in that patient it occured in 30 minutes.

At the first treatment 12 cases of left *T1* disappeared during the 30 minutes of the session and in addition 24 cases of left *T1* ceased during the interval between the first and second treatment (meaning 36 cases of left *T1* disappearances after the first treatment). At the second treatment 14 cases of left *T1* disappeared instantly and in 25 cases later on during the week (altogether 39 left *T1* disappearances). At the third treatment 6 cases of left *T1* disappeared instantly and 10 cases later (altogether 16 left *T1* disappearances). At the fourth treatment 7 cases of left *T1* disappeared instantly and 3 cases later (altogether 10 left *T1* disappearances). At the fifth treatment 1 case of left *T1* disappeared instantly and 2 cases later (altogether 3 left *T1* disappearnaces).

Left *T1* disappeared at least in two subsequent sessions in 15 cases (either at the treatment session or later on in the week). In 5 cases a disappearance of left *T1* occured in three subsequent treatments and in 3 cases in four treatments.

6.2.2. The changes in frequency

The mean baseline right *T1* frequency was 4612 Hz and in the first treatment the post-treatment right *T1* was 4296 Hz but the difference was not statistically significant. The mean baseline left *T1* frequency was 3631 Hz and in the first treatment the post-treatment left *T1* was 3606 Hz, the difference was not statistically significant. In the first treatment in 6 % of the right *T1* cases frequency changes occured (6 cases out of 109). In 5 % of cases with left T1 frequency changes occurred (7 cases out of 137). In the post-treatment period after the first treatment the pitch of right *T1* lowered in 2 cases and it rose in 4 cases. In the post-treatment period the pitch of left *T1* lowered in 6 cases and it rose in 7 cases.

In the second treatment the mean pretreatment right *T1* frequency was 4478 Hz and the post-treatment frequency was 4511 Hz, the difference was not statistically significant. The mean pretreatment left *T1* was 3819 Hz and the post-treatment 3784 Hz, the difference was not statistically significant. In the second treatment right *T1* frequency changed in 4 % of the cases and left *T1* frequency in 5 % of the cases. During the post-treatment period right *T1* frequency changed in 8 % of cases and left *T1* frequency in 5 % of the cases. In the third treatment right *T1* frequency changed in 9 % of the cases, left *T1* in 4 % of the cases. During the post-treatment period the pitch of right *T1* rose in 2 cases, the pitch of left *T1* lowered in 1 case and rose in 2 cases. In the fourth treatment the pitch of left *T1* lowered in 1 case in the

post-treatment period. In the fifth treatment the pitch of left *T1* changed in 1 case and in 1 case the pitch of left *T1* lowered in the post-treatment period.

6.3. The time interval between the treatment and result

In the first treatment 30 % of the right *T1* cases, 31 % of the left *T1* cases and 8 % of the head *T1* cases responded within 30 minutes; 35 % of the right *T1* cases, 46 % of the left *T1* cases and 17 % of the head *T1* cases responded between 30 minutes and 24 hour period; 9 % of the right *T1* cases, 7 % of the left *T1* cases and 17 % of the head *T1* cases responded during 2-7 day period. One right *T1* case and one left *T1* case had a response after 7 days. The same distribution of the time intervals seemed to occur in the subsequent treatments. In the right and left *T1* cases tinnitus responded during 30 minutes in a little more than one third of the cases and during the 24-hour-period in a little more than one third of the cases. The percentage of a response in head tinnitus was lower also in the following treatments. The time intervals are presented in fig. 12 and 13.

Fig.12 TIME LAG BETWEEN TREATMENT AND RESULTS (T1) RIGHT EAR

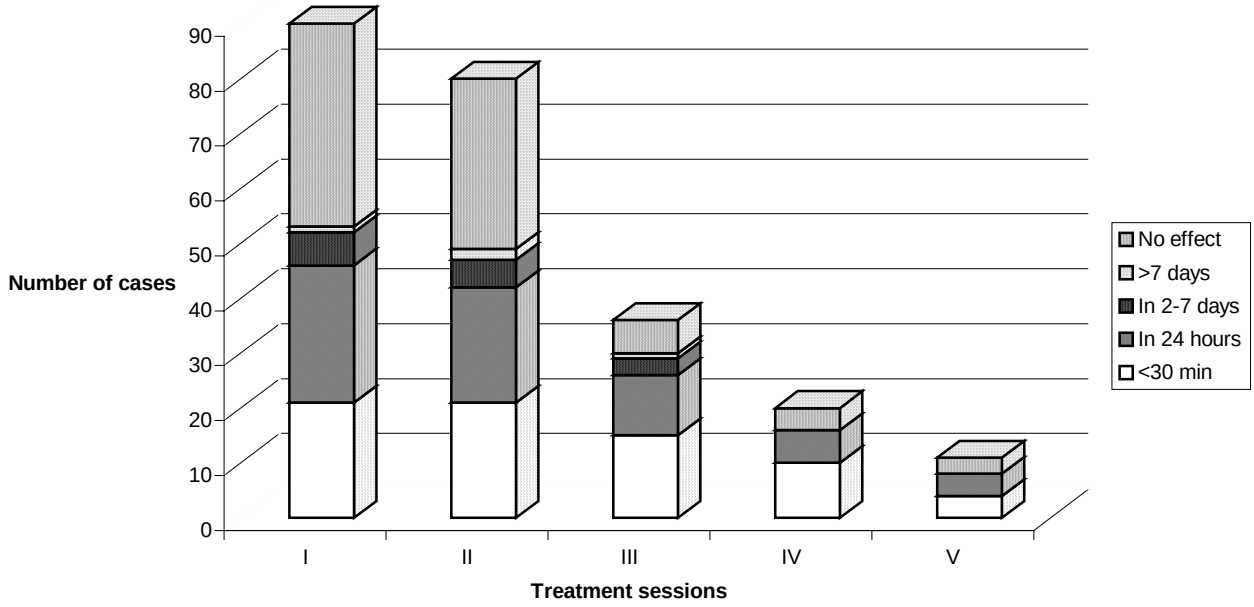
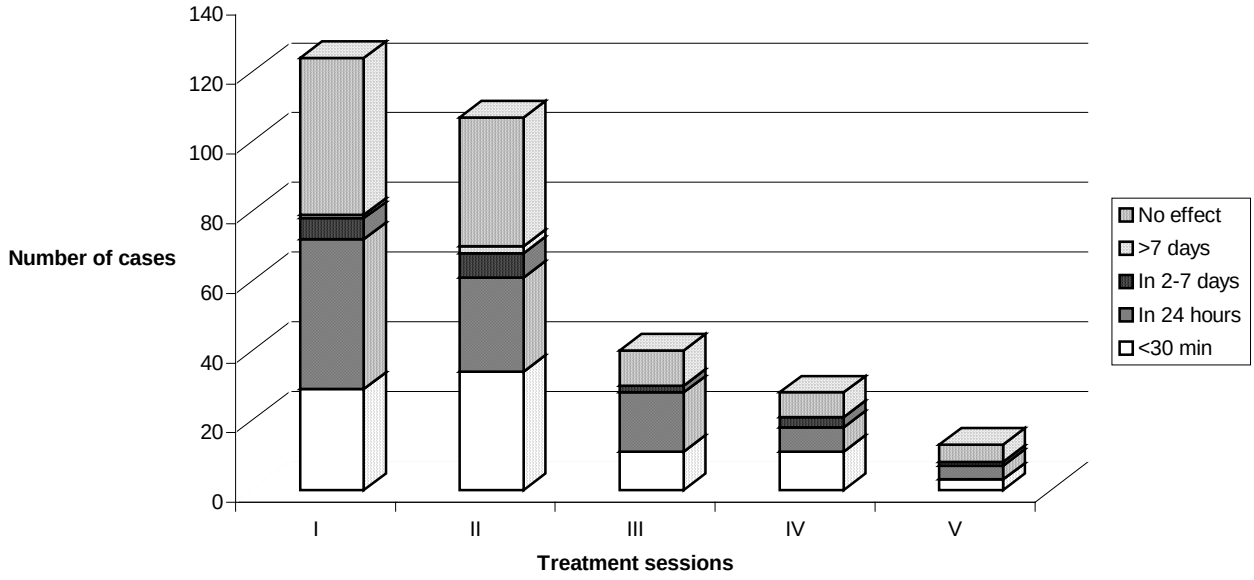


Fig.13 TIME LAG BETWEEN TREATMENT AND RESULT (T1) LEFT EAR



6.4. The duration of tinnitus changes

The first treatment affected tinnitus and the effect lasted at least a week in 22 % of the right *T1* cases and in 29 % of the left *T1* cases but in 0 % of the head *T1* cases. The change of tinnitus lasted hours or days in 32 % of the right *T1* cases and in 30 % of the left *T1* cases and in 27 % of the head *T1* cases. The duration of the effect during the treatment sessions are presented as the number of cases in fig. 14 and 15.

After the second treatment the treatment effect lasted at least hours in 47 % of the right *T1* cases, in 49 % of the left *T1* cases and in 27 % of the head *T1* cases. A relief was still reported at the time of the last report (reporting time varied from 2 weeks to 6 months) in 10 % of the right *T1* cases, in 9 % of the left *T1* cases but in no head *T1* cases. At least 1 day effect was reported by 41 % of the right *T1* cases, in 44 % of the left *T1* cases and in 27 % of the head *T1* cases.

In the third treatment the effect lasted at least 1 day in 57 % of the right *T1* cases, in 47 % of the left *T1* cases and in 27 % of the head *T1* cases. A response at least a week was reported in 25 % of the right *T1* cases, in 26 % of the left *T1* cases and in 18 % of the head *T1* cases. In the fourth treatment at least 1 day response was reported in 58 % of the right *T1* cases, in 61 % of the left *T1* cases and in 38 % of the head *T1* cases. At least one week response occured in 35 % of the right *T1* cases, in 32 % of the left *T1* cases and in 38 % of the head *T1* cases. In the fifth treatment the effect lasted at least a day in 57 % of the right *T1* cases and in 33 % of the left *T1* cases. At least a week response was reported in 21 % of the right *T1* cases and in 13 % of the left *T1* cases.

Fig.14 DURATION OF TREATMENT EFFECT (T1) RIGHT EAR

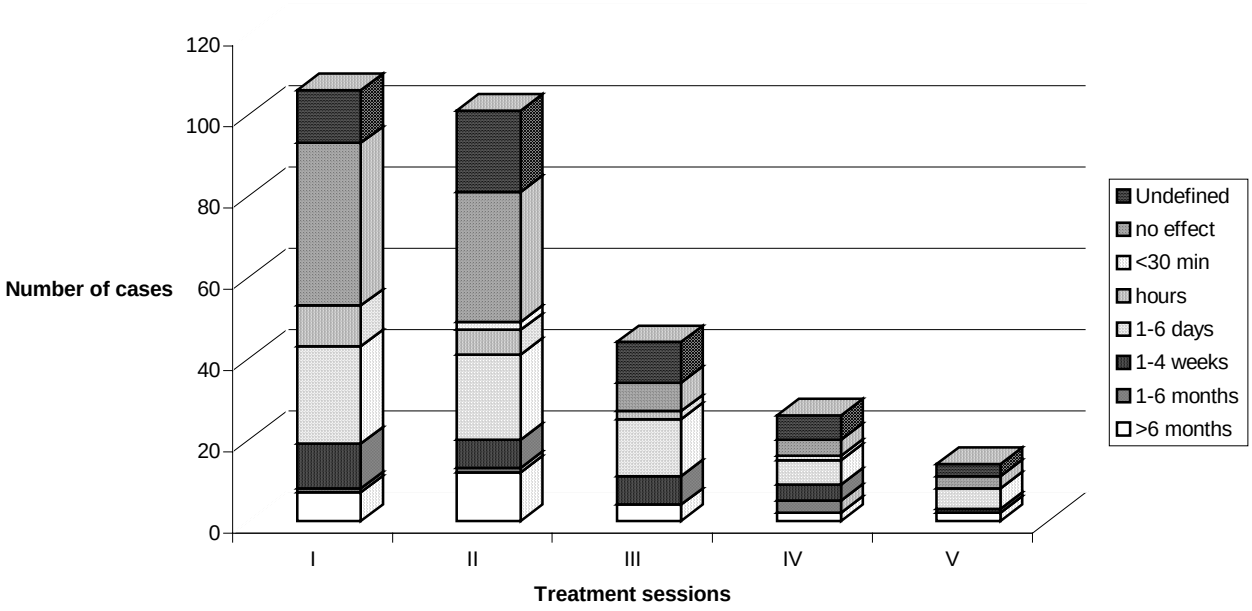
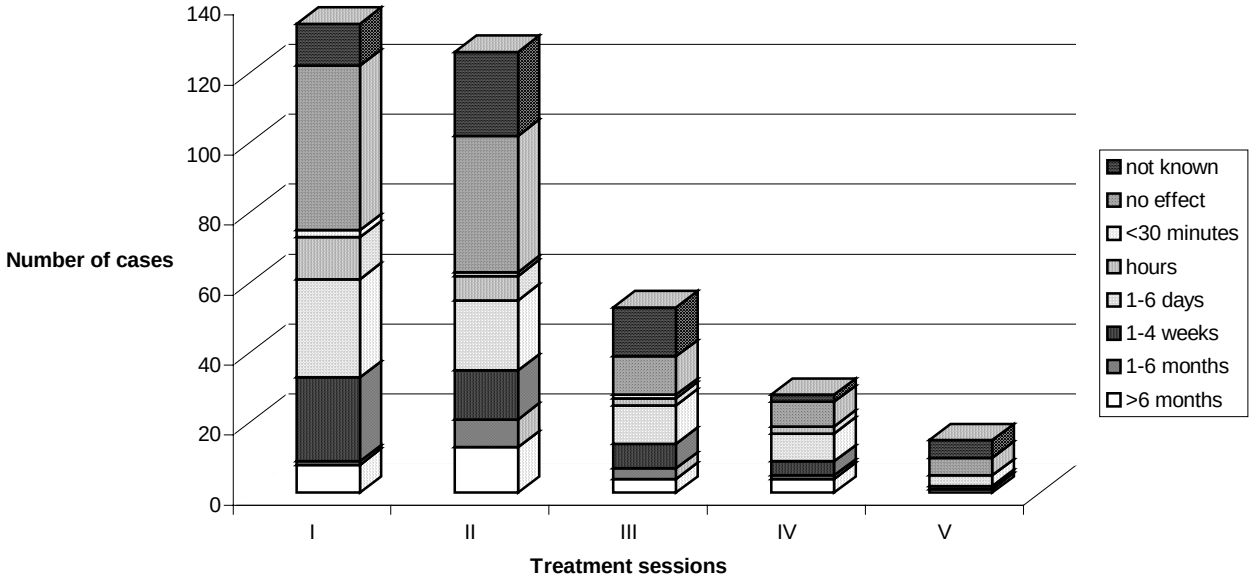


Fig.15 DURATION OF TREATMENT EFFECT (T1) LEFT EAR



6.5. The characters of the reappearing tinnitus

After the first treatment tinnitus did not return or was less loud if returned in 15 % of the right *T1* cases, in 18 % of the left *T1* cases and in no head *T1* cases. Tinnitus was in the pretreatment levels in 32 % of the right *T1* cases, in 34 % of the left *T1* cases and in 25 % of the head *T1* cases. Reappearing tinnitus was louder than originally in 5 % of the right *T1* cases, in 2 % the left *T1* cases but in no head *T1* cases.

After the second treatment tinnitus was less loud or did not return in 15 % of the right *T1* cases, in 15 % of the left *T1* cases but in no head *T1* cases. It returned to the pretreatment level in 28 % of the right *T1* cases, in 29 % of the left *T1* cases and in 20 % of the head *T1* cases.

After the third treatment tinnitus did not return or was less loud in 27 % of the right *T1* cases, in 15 % of the left *T1* cases and in no head *T1* cases. After the fourth treatment tinnitus did not return or was less loud in 16 % of the right *T1* cases, in 20 % of the left *T1* cases and in no head *T1* cases. After the fifth treatment tinnitus did not return or returned on a more quiet level in 21 % of the right *T1* cases and in 13 % of the left *T1* cases. The reappearing or recovering tinnitus levels compared to original values during the treatment sessions are presented in fig. 16 and 17.

Fig.16 LOUDNESS OF REAPPEARING TINNITUS (T1) RIGHT EAR

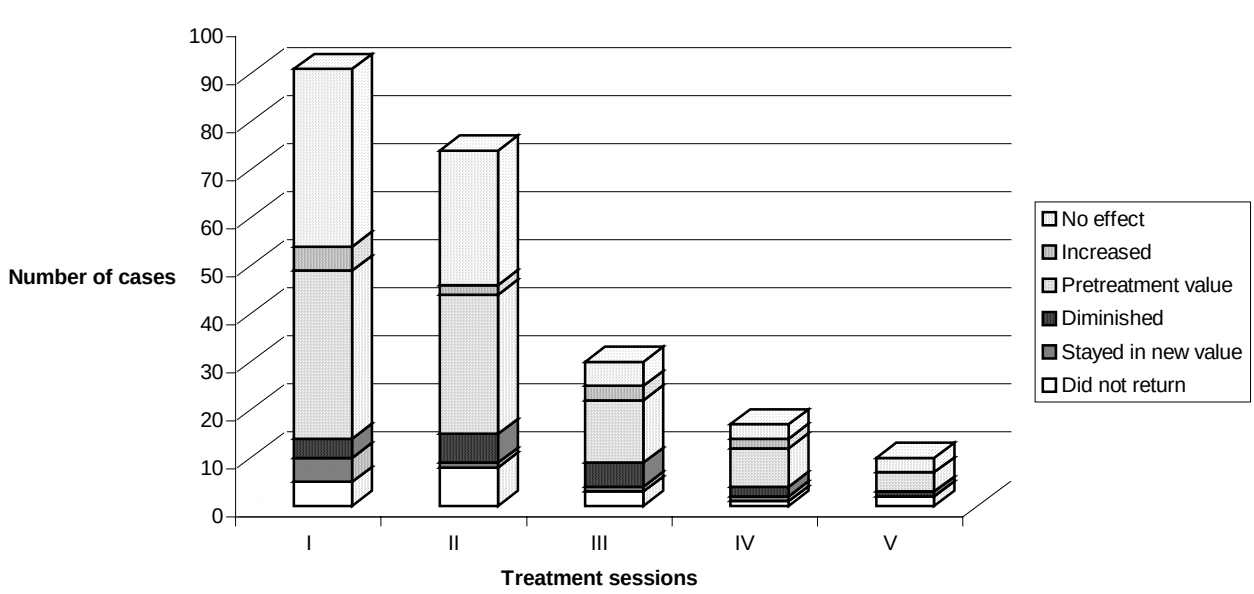
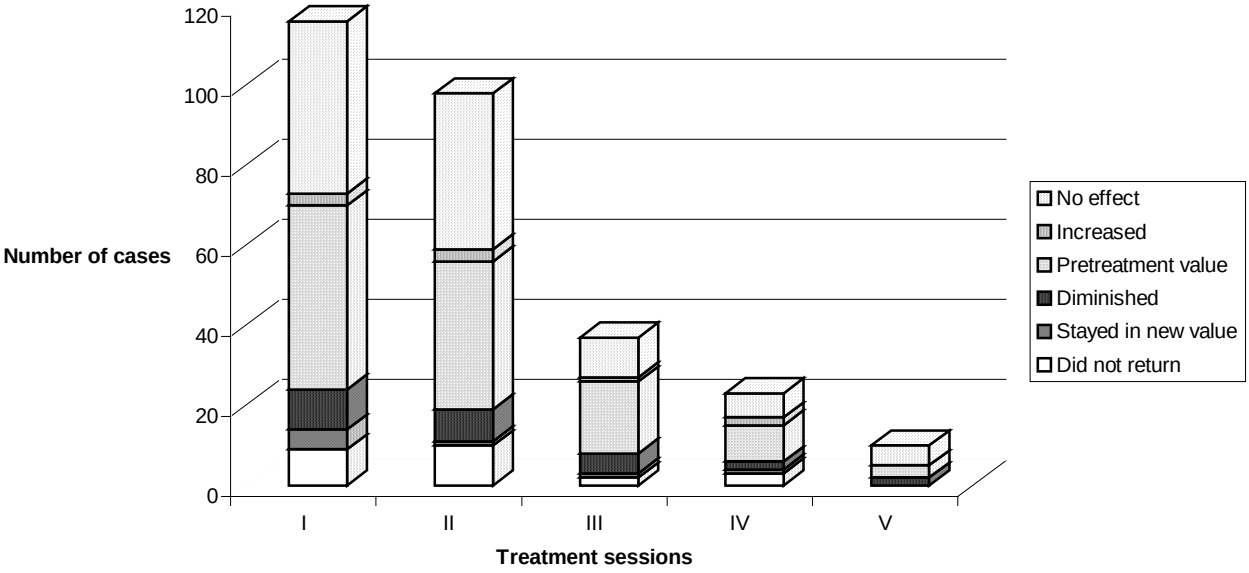


Fig.17 LOUDNESS OF REAPPEARING TINNITUS (T1) LEFT EAR



7. THE RESULTS AFTER THE TREATMENT PERIOD

7.1. The results after the last treatment

7.1.1. The volume of changes

At the last treatment sound disappeared or reduced at least 10 dB in volume compared to the initial tinnitus volume in 32 % of the right *T1* cases, in 31 % of the left *T1* cases and in 5 % of the head *T1* cases. *T2* sounds were so few and most of the time not measurable that they could not be analyzed.

The control patients were also measured about six months after the first interview. The right *T1* had disappeared or diminished at least 10 db in 33 % of the cases, the left *T1* in 25 % of the cases. Tinnitus was at least 10 dB louder in 14 % of the right *T1* cases, in 16 % of the left *T1* cases.

7.1.2. The comparisons of tinnitus changes among the treated and control groups

The loudness of right *T1* changed more in the treatment group ($p < 0.05$), but the loudness changes of left *T1* or head *T1* did not statistically differ between the groups. More frequency changes occured in the control group with right *T1* ($p < 0.05$). No difference in the frequency changes concerning the other groups of tinnitus occured.

7.2. The results 6 months after the end of the treatment period

7.2.1. The over-all rating of the change of the tinnitus sounds

The treated patients were requested to rate their tinnitus in the post-treatment period. The over-all rating of tinnitus (both *T1* and *T2* summarized) was better (either all the post-treatment time or at least part of the time) than in the pretreatment period in 27 % of the cases with right ear tinnitus, in 58 % of the cases with left ear tinnitus and in 36 % of the cases with head tinnitus.

The control patients were requested to rate their tinnitus in the 6 months interval. The over-all rating of tinnitus was better all the time or at least part of the time in 10 % of the cases with right ear tinnitus, in 25 % of the cases with left ear tinnitus and in no cases with head tinnitus.

When the groups were compared with crosstabs a statistically significant difference was found concerning to the rating of tinnitus between the treated patients and the controls. Right ear tinnitus was better in the treated group ($p < 0.001$) and left ear tinnitus was better in the treated group ($p < 0.01$). The difference in head tinnitus was not statistically significant. The results are presented in fig. 18, 19 and 20.

Fig.18 SUBJECTIVE TREATMENT EFFECTS AFTER 6 MONTHS (RIGHT EAR)

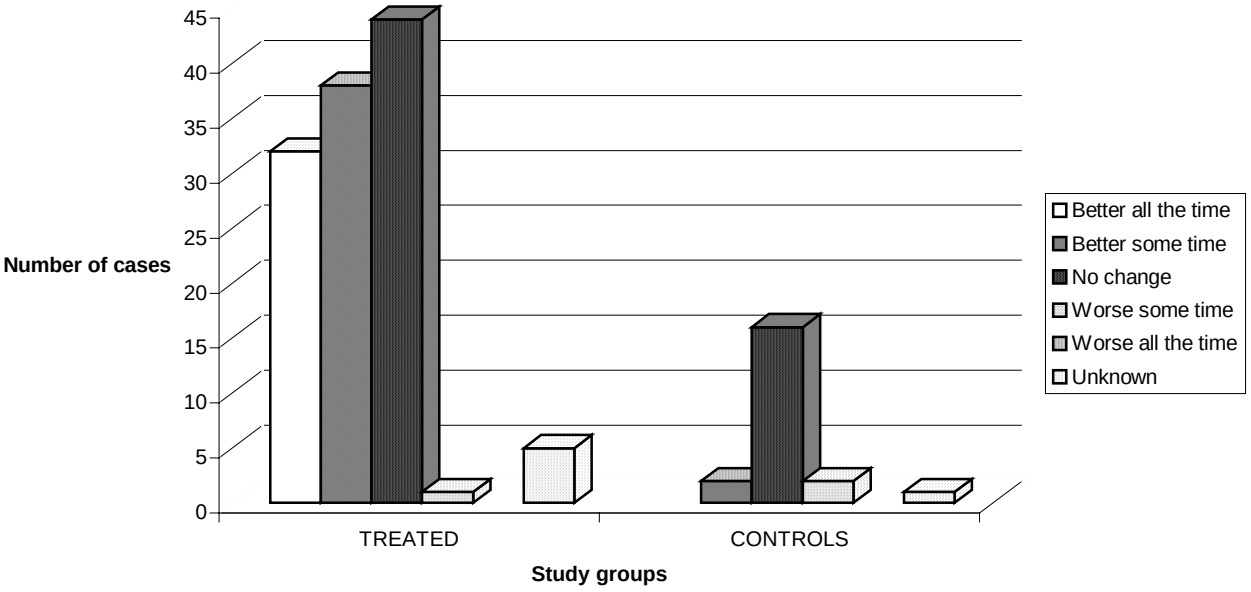


Fig.19 SUBJECTIVE TREATMENT EFFECT AFTER 6 MONTHS (LEFT EAR)

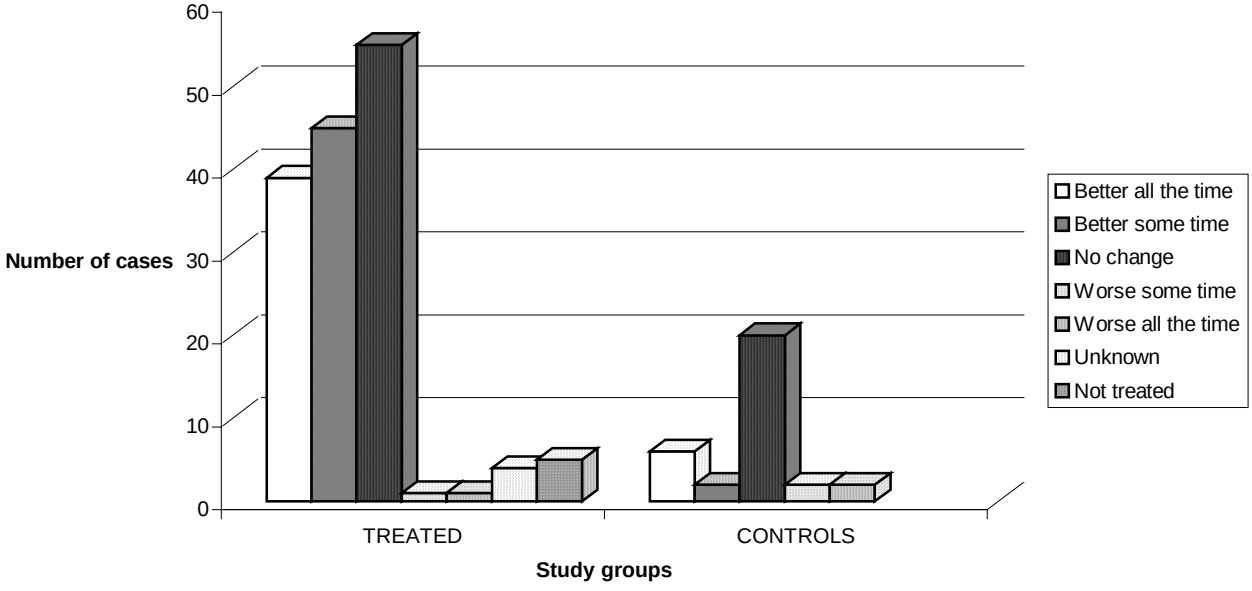
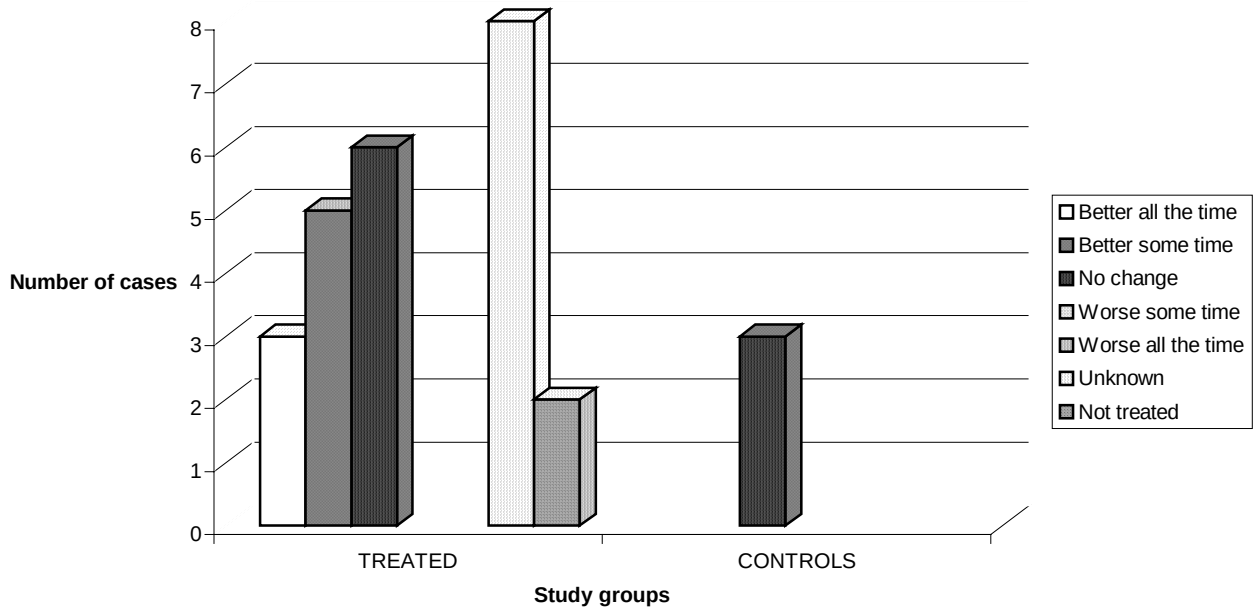


Fig.20 SUBJECTIVE TREATMENT EFFECTS AFTER 6 MONTHS (HEAD)



7.2.2. The duration of tinnitus change

The treated patients were asked to estimate how long their tinnitus change (tinnitus vanished or changed to better or worse) had lasted during the 6 months interval from the last treatment up to the inquiry. The right *T1* changes in tinnitus level lasted at least 10 % of the post-treatment time in 59 % of the cases, tinnitus was better at least 50 % of time in 30 cases (25 %). The left ear *T1* changes of tinnitus level lasted at least 10 % of the posttreatment time in 58 % of the cases and the changes lasted at least 50 % of the post-treatment time in 38 cases (26 %).

The control patients were also asked to estimate the change in tinnitus 6 months after the last measurement and how long the tinnitus change had lasted. The right *T1* in 3 cases (14 %) had ceased and was silent 100 % of time, in 16 cases (76 %) *T1* had not been different all that time. With left *T1* the *T1* sound was better 100 % of the post-measurement time in 3 cases (10 %), in 4 cases (13 %) *T1* stayed better for 90 % of time and in 1 case (3 %) it was better for 20 % of the time, in 19 cases (61 %) *T1* was not better during the post-measurement time. With head *T1* in 3 cases (100 %) no change occured during the post-measurement time.

When the treated and control patients were compared with crosstabs a statistically significant difference existed concerning the duration of the post-treatment results in the left *T1* between the groups, longer in the treatment group ($p < 0.005$). With the right ear *T1* a difference between the groups existed, longer effect in the treatment group, ($p<0.06$) but it was not statistically significant. No statistical significance was found with regard to the *T1* sound sensed in the head nor with any of the *T2* sounds. The results are presented in fig. 21 and 22.

Fig.21 DURATION OF TREATMENT EFFECT (SUBJECTIVE ESTIMATION AFTER 6 MONTHS) (RIGHT EAR)

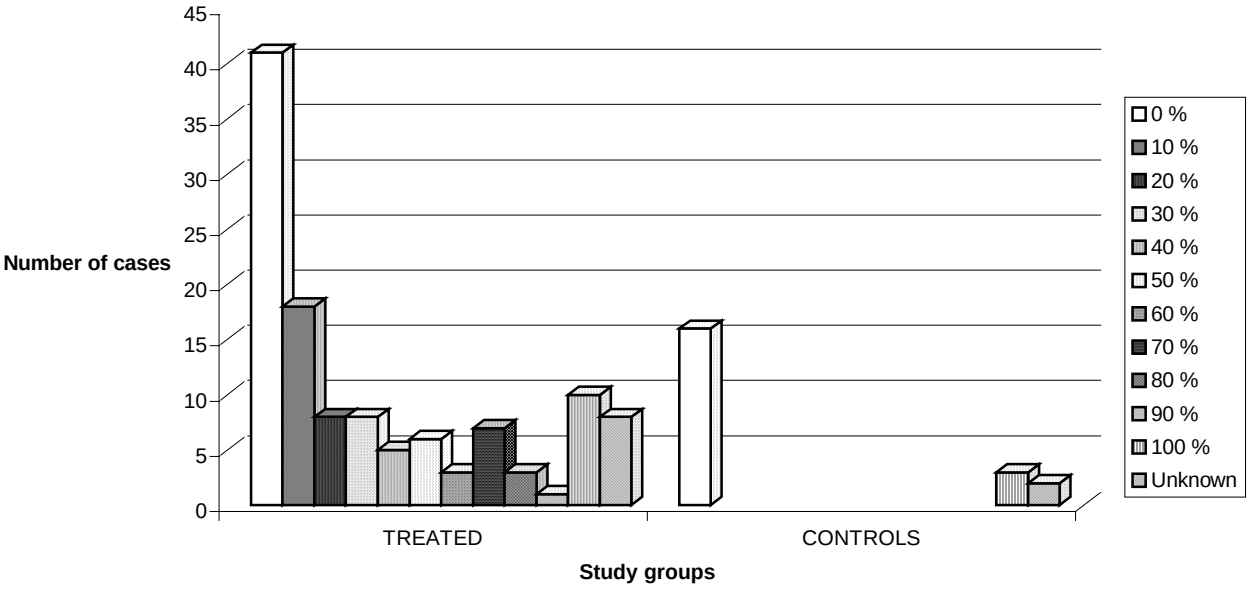
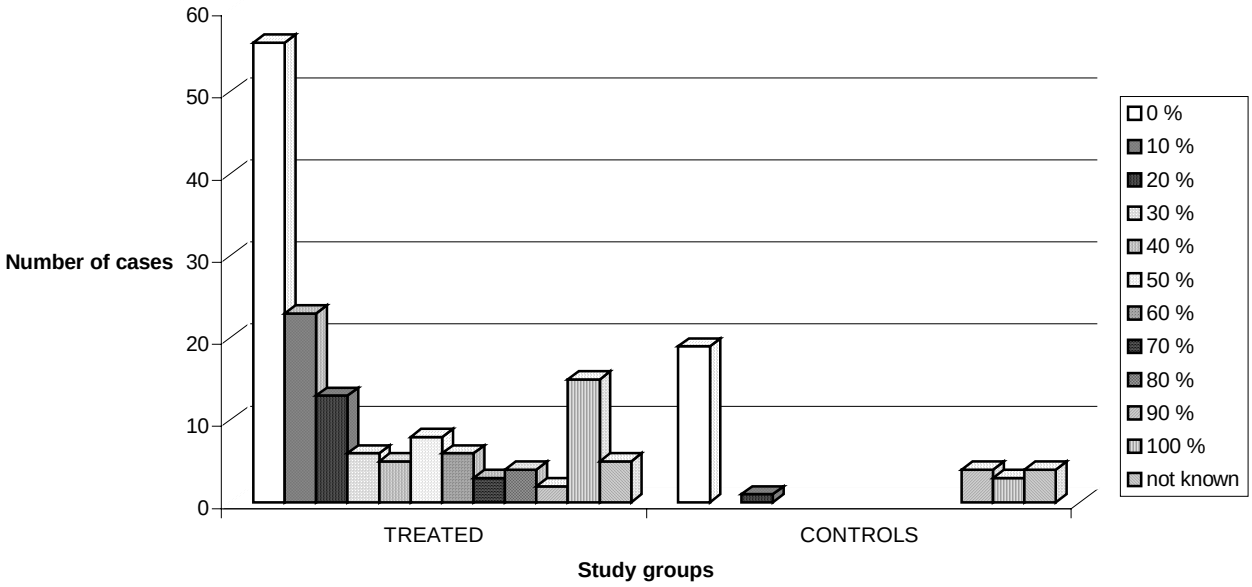


Fig.22 DURATION OF TREATMENT EFFECT (SUBJECTIVE ESTIMATION AFTER 6 MONTHS)(LEFT EAR)



8. THE RESULTS FOR THE TINNITUS-RELATED DISORDERS

40 cases with pressure in the ear existed before the first treatment (21 in the right ear and 19 in the left ear). 38 cases of these were treated, the symptom improved partly or totally in 7 right ear cases and in 2 left ear cases. In the second treatment 38 cases were treated; the symptom improved in 7 right ear cases and in 3 left ear cases.

Fullness of the ear was reported in 63 cases before the first treatment (32 in the right ear and 31 in the left ear). 59 of these were treated, the symptom eased or disappeared after the treatment in 8 right ear cases and in 4 left ear cases. In the second treatment the symptom improved partly or totally after the treatment in 12 right ear cases and in 6 left ear cases out of 58 treated cases.

Feeling of cracking of the sounds or diplacusis was experienced in 31 cases before the first treatment (16 cases in the right ear and 15 cases in the left ear). 24 cases of these were treated and ear was better in 3 right ear cases and 1 left ear case. In the second treatment the cracking of sounds or diplacusis eased after the treatment in 2 right ear cases and in 2 left ear cases out of 24 treated cases.

Hyperacusis to certain sounds was reported in 76 cases before the first treatment (42 cases in the right ear and 34 cases in the left ear). After the treatment (69 treated) the hyperacusis was relieved in 13 cases (9 right ear cases and 4 left ear cases). In the second treatment (8 right ear cases and 3 left ear cases) the hyperacusis improved in 11 cases out of the treated 65 cases.

Hearing impairment occured in 256 ears before the first treatment (125 right ear cases and 131 left ear cases). The subjective hearing improved only in 3 cases out of the treated 216 cases (2 right ear cases and 1 left ear case). In the second treatment the hearing improved in 10 cases (4 right ear cases and 6 left ear cases) out of the 207 treated cases.

9. COMPLICATIONS OF THE THERAPY

All the complications except volume and frequency changes during the treatment period are listed in table 11. The volume and frequency changes are reported earlier. The frequency rise was considered a complication if the new tinnitus was less tolerable for the patient but not if it was more pleasant.

Table 15

COMPLICATIONS OF THE THERAPY

(Reported complications during treatment sessions, number of cases)

	SESSIONS				
COMPLICATION	**I**	**II**	**III**	**IV**	**V**
No complications	161	168	71	41	21
Excess muscle pain	8	2	1		
Excess sleepiness	4	1	1		
Fainting or dizzy	5	3			1
Reflection in hand	1		1	2	
Skin reactions		1	1	1	2
Unknown			1	1	1

10. THE COMPARISONS BETWEEN RESPONDERS, NON-RESPONDERS AND CONTROLS

10.1. The divisions into groups

After the second treatment the treated patients were divided into two subgroups according to their own ratings; if their tinnitus had changed in the treatment they were classified as responders and if their condition was unchanged they were classified as non-responders. Then both groups were compared with each other and with the non-treated controls. After that the classification of the groups was as follows: group 1. responders, group 2. controls and group 3. non-responders.

10.2. The age and sex distribution of the groups

The age of subjects in the groups did not differ statistically. More females existed in the group 1 (82 females and 36 males) compared to the other groups. Group 2 included 21 females and 18 males and group 3 consisted of 22 females and 38 males. The difference in the sex distribution between the two treated groups was statistically highly significant ($p < 0.001$). The responders and the controls did not differ statistically in the sex distribution, neither did the non-responders and the controls. The male / female ratio was highest in the non-responder group and lowest in the responder group.

10.3. The comparison of ear diseases and hearing disorders between the groups

No statistical differences were found comparing ear diseases between any of the three groups with crosstabs or with logistic regression.

A difference concerning hearing disorders between the responders and the controls occured, more bilateral inherited or unclear hearing disorders in the control group ($p=0.01$). A statistically significant difference concerning subjective hearing impairment in the left ear existed between the two treated groups; significantly ($p< 0.05$) worse hearing in the non-responder group.

10.4. The duration and type of noisy work in the groups

The two treated groups did not differ in their working noise exposition neither did the non-responders and the controls. A statistical difference was found between the responders and the controls ($p< 0.05$), the former having been shorter times in a noisy work. As to the types of noise, exposure to paper industry noise and other noise seemed to be more common in the control group compared to the responders ($p<0.05$).

10.5. The comparison of the tinnitus character between the groups

10.5.1. The description of tinnitus

Table 16

THE DESCRIPTION OF TINNITUS IN THE GROUPS
(% of cases, right T1)

TYPE OF TINNITUS	RESPONDERS	NON-RESPONDERS	CONTROLS
Ringing	28	67	40
Chirping	23	13	24
Whoosing	18	18	8
Humming	12	2	8
Pulse-type	3		
Snapping			4
Other sound	12		16
NUMBER OF CASES	77	45	26

Table 17

THE DESCRIPTION OF TINNITUS IN THE GROUPS
(% of cases, left T1)

TYPE OF TINNITUS	RESPONDERS	NON-RESPONDERS	CONTROLS
Ringing	32	51	36
Chirping	28	17	16
Whoosing	14	19	16
Humming	12		10
Pulse-type	4		3
Snapping		2	
Other	9	6	19
NUMBER OF CASES	98	54	32

A statistically significant ($p< 0.001$) difference existed in the tinnitus character of the right *T1* between the responders and the non-responders. The tinnitus description among the responders was mostly either ringing (28 %) or chirping (28 %), the description of the non-responders was mostly ringing (67 %). A statistically significant ($p<0.05$) difference existed between the non-responders and the controls in the character of right *T1*. The description of the controls was mostly ringing (40 %) or chirping (24 %). No statistically significant differences existed between the responders and controls concerning the right *T1* tinnitus character.

A statistically significant ($p<0.05$) difference existed in the tinnitus character of left *T1* between the responders and the non-responders. In the responder group the most common descriptions were ringing (32 %) and chirping (28 %), in the non-responder group the most prevalent description was ringing (51 %). No statistically significant differences occured in left *T1* between the controls and the non-responders. The most prevalent description in the left ear T1 tinnitus sound in the control group was ringing (35.5 %). No statistically significant differences existed between the responders and the controls concerning the left *T1* tinnitus character.

No statistically significant differences in the character of the head *T1* occured between the groups. The most common description of the head *T1* sound in the responder group was whoosing (43 %), in the non-responder group head *T1* was ringing (43 %), in the control group only 3 head *T1* sounds existed, whistling was the most common description (2 cases). No statistically significant difference existed between the responders and the controls concerning the character of the head *T1*.

No statistical differences were found between the groups concerning to the amount of tinnitus sounds. No statistically significant difference between the groups existed concerning the duration of tinnitus.

10.5.2. The site of tinnitus

Tinnitus *T1* sounds tended to be more prevalent in the left ear in the responders compared to the control patients, but the difference was not statistically significant (p=0.08). No such difference existed among the non-responders and the control patients nor among the two treated groups. In the responder group patients tended to have the most prevalent *T1* sound in one of the ears, not bilaterally or in the head. In the non-responder group more bilateral cases existed, the difference between the treated groups was statistically significant (p<0.05).

10.6 The natural behavior of tinnitus

10.6.1. The natural fluctuations

Right *T1* disappeared spontaneously more often and for longer periods by the responders than by the controls (p<0.01) and the same observation was made also with left *T1* (p<0.01). Tinnitus was more constant in character (without fluctuations) in the non-responder group than in the responders, the difference was statistically significant in right *T1* (p<0.05). In left *T1* a similar difference was found but it was not statistically significant (p=0.07). No differences occured between the non-responders and the controls in the natural fluctuations and disappearance of tinnitus.

10.6.2. The reactions to stress or head and body movements

More fluctuations of right *T1* in physical stress occurred in the responder group than in the non-responders. In responders no change occured in 34 cases, aggravation occured in 38 cases and decrease of tinnitus in 4 cases. In the non-responder group no change occured in 28 cases, aggravation in 11 cases and decrease of tinnitus in 2 cases. The difference between the responders and the non-responders was statistically significant (p<0.05). In left *T1* more fluctuations occurred in the responder group than in the non-responders. In the responders no changes of left *T1* occured in 44 cases, aggravation occured in 46 cases and decrease of tinnitus in 4 cases. In the non-responder group no change of left *T1* occured in 35 cases, aggravation in 11 cases and decrease in 5 cases. The difference between the groups was statistically significant (p<0.05). No other differences existed between the groups.

10.7. The measurements of tinnitus

The audiometrically measured tinnitus in the respondes, the non-responders and the controls was compared. The loudness of tinnitus, either *T1* or *T2* did not statistically differ in either ear between the groups. The frequency of tinnitus (*T1* or *T2*) did not differ either. In the VAS-scale no difference existed between the groups concerning the loudness of any of the four most prevalent tinnitus sounds (*T1*, *T2*, *T3* and *T4*).

10.8. The earlier treatments of tinnitus

Vasodilative medication was more prevalent in the control group compared to the responders (p=0.01) or the non-responders (p=0.015). The analgesics were significantly more prevalent among the controls compared to the responders (p<0.001) or the non-responders (p<0.001). A difference between the treated groups was found about the usage of analgesics, more in the non-responder group (p<0.05). More antihistamin usage occured in the control group compared to responders (p<0.01). Physiotherapy as a treatment had been tried by more patients in the non-responder group than in the responder group (p<0.05). Patients in the

control group had tried physiotherapy more than non-responders ($p<0.001$) and even more than the responders ($p<0.001$). Masking was tried by just two patients in the non-responder group.

10.9. The comparison of status between the groups

10.9.1.The ORL- status and audiometric measurements

No statistical difference was found between the responder, the non-responder and the control groups concerning the ORL-status.

A statistical difference existed between the responders and the non-responders concerning the hearing in 500 Hz level in the right ear, being statistically worse in the non-responder group ($p<0.05$). A difference existed between the responders and the controls in 1000 Hz hearing in the right ear ($p<0.05$), worse in the control group. A statistically significant ($p<0.05$) difference existed between the responders and the non-responders in the hearing of the 8000 Hz in the right ear, worse in the non-responder group.

10.9.2. The muscular status

The muscular tension was situated more in the left side of the neck in the responders compared to mostly evenly situated tension of the non-responders ($p<0.01$) or the controls ($p<0.05$). No statistically significant differences were found in the muscular status in the shoulder or scapular area.

10.10. The objective loudness changes of tinnitus after the last treatment

A difference of the right *T1* audiometric loudness change at the end of the treatment period existed between the responders and the non-responders ($p< 0.001$). The difference of the left *T1* change between the responder and the non-responder group was statistically significant ($p<0.001$). No difference existed in head tinnitus between the responder and the non-responder group nor any difference between the same groups with regard to the *T2* sounds.

More tinnitus disappearances occured in the control group compared to the non-responders in left *T1* ($p<0.05$). No statistically significant difference existed between the responders and the controls in right *T1*.

10.11. The results after 6 months

10.11.1. The percentage of tinnitus free periods

A statistically significant difference existed between the responders and the non-responders in the right *T1* sound, tinnitus had stayed better for longer periods in the responder group ($p<0.005$). A similar difference existed in left *T1* between the responders and the non-responders, tinnitus had stayed better in the responder group ($p<0.001$). No difference existed in head *T1* between the two treated groups.

In right *T1* no difference was found between the non-responders and the controls in the tinnitus-free periods. A difference occured between the non-responder and the control groups in left *T1*, more total disappearance in the control group but more shorter disappearance among the non-responders ($p<0.05$). Longer better periods occured in the responder group compared to the controls in right *T1* ($p<0.001$) and in left *T1* ($p<0.005$).

10.11.2. The subjective judgement about tinnitus

The subjects were asked to estimate the over-all quality of their tinnitus (*T1* & *T2*) during the last 6 or more months. A statistical difference existed between the responders and the controls in right ear tinnitus (*T1* and *T2* together). The responders estimated statistically more often that their tinnitus had been better all the time or at least part of the time, the controls mostly reported no change ($p<0.001$). A statistical difference existed between the non-responders and the controls with right ear tinnitus. The non-responders stated that their tinnitus had been better for at least part of the time more often than controls ($p<0.01$). Between the two treated groups a statistically significant difference existed; the responders felt more often that their tinnitus had been better during the post-treatment period ($p<0.001$). With left ear tinnitus (*T1* and *T2*) similar difference existed between the groups. The responders considered their tinnitus better for all the post-treatment time or at least part of the time more often than the two other groups. The difference between the responders and the controls was statistically significant ($p<0.001$) and also between the responders and the non-responders ($p<0.001$). The non-responders had longer better periods than the controls, ($p=0.01$). With head tinnitus (*T1* and *T2*) less tinnitus existed in the posttreatment period in the responder group than in the control group ($p<0.05$) or in the responder group than in the non-responder group ($p<0.05$). No difference existed between the controls and the non-responders in head tinnitus.

10.12. Tinnitus related disorders

The pressure in the right ear relieved better in the responder group compared to the non-responders ($p<0.05$). The left ear pressure relieved better in the responder group compared to the non-responders ($p<0.05$). No other statistical differences existed between the groups in the other tinnitus-related disorders.

11. OTHER CONSIDERATIONS BASED TO THE RESULTS

11.1. The side of tinnitus compared to the muscular status

A correlation between the side of tinnitus and the side of the muscular tension in the cervical and upper thoracic area occured. Tinnitus was situated mostly in the side with more muscular tension. Rightsided tension seemed to co-exist with rightsided tinnitus and vice versa. In the cervical area the relationship was statistically significant ($p<0.001$). In shoulder area the relation between the side of tension and the side of tinnitus was statistically significant ($p<0.001$). A statistically significant relationship existed between the side of tinnitus and the side of tension in the scapular area although only 64 individuals had been tested for scapular area tension. The side with more tension in the scapular area was significantly more often also the side with tinnitus ($p<0.001$).

11.2. The most important correlations with good results

With regard to right ear tinnitus good outcome correlated primarily with description of tinnitus as chirping or humming ($p<0.0001$), female gender ($p<0.0001$), then with the situation of tension in the neck area ($p<0.005$) and then also with the tension in the scapular area ($p<0.005$). With regard to left ear tinnitus female gender seemed to be the most important factor to explain the good outcome ($p<0.0005$), then the description with the term chirping or humming ($p<0.005$), then the side of the tension of the neck ($p<0.01$) and also the side of the tension in the shoulder area. The most important factors that seemed to correlate with the good outcome of treatment were estimated and their importance was graded with logistic reggression. In right ear tinnitus the most important factor was the description of tinnitus ($p<0.001$) and in left ear tinnitus gender was the most important ($p<0.0005$). The term chirping correlated with female gender and thus the importance of the description could be also explained by the gender.

G. DISCUSSION

1. COMPARISON OF THE PATIENTS IN THE PRESENT STUDY TO OTHER STUDIES

1.1. Age

In the present study the medium age of the subjects was 63 years and both males and females were included, although females were more numerous in the treatment group. The medium age seemed to be the same as in other publications on treatment experiments. Henry et al (1992) had a mean age of 64.6 years. Baskill et al (1992) published a work with a mean age of 64.7 years. In a tinnitus self- help group Stouffer et al (1992) had a mean age of 60 years and Goebel et al (1992) got a mean age of 48 years with complex chronic tinnitus treatment group. Coles et al (1993) found tinnitus prevalence among people over 60 years to be more than twice the prevalence among 18 -40 years old people. Chung et al (1984) expressed tinnitus prevalence to be 6.6 % among 30 000 workers. The prevalence increased from 3.1 % in people under 19 years to 20 % in those over 70 years.

1.2. Other diseases

The diseases in the study group were quite commonly present in these age groups. In the city of Imatra the prevalence of hypertonia was 11.0%, coronary diseases 5.0%, epilepsy 1.0 %, diabetes 3.3 % and heart insufficiency 2.3 % in 1995. (KELA, Imatra according to the statistics about people receiving monetary compensation for medication). Among the study group the prevalence of hypertonia was 25.8 %, coronary disease 17.5 %, epilepsy 2.3 %, diabetes 6.0 % and heart insufficiency 17.1%. But the study group patients were probably older than the medium age of the inhabitants of the city of Imatra and the medication could not be compared to the medication of age comparable individuals. Davis et al (1992) found diabetes to be one general disease that seemed to carry an excess risk for prolonged spontaneous tinnitus (PST). The relationship in their study was complex and interfered with hearing impairment. They found another relationship with hypertonia and PST, and the relationship was most obvious in subjects with bilateral tinnitus. Medication, on the other hand, seemed to expose an individual to a risk of getting PST. Kraft et al (1996) published a survey with hyperinsulinemia in subjective idiopathic tinnitus and found it to be a major metabolic marker with diagnostic and therapeutic relationship to tinnitus. But also in that study the relationship was connected to hypertonia like in the study by Davis et al (1992). In the present study the existence of diabetes or hypertonia were just questioned. The state of hyperinsulinemia or the blood pressure levels were not measured, because the above studies were published after our data collection.

1.3. Hearing and ear diseases

When criteria of impaired hearing was set at a mean hearing threshold of 30 dB or worse at 500 to 2000 Hz and/ or 50 dB hearing loss at 2000 Hz, the prevalence of people with hearing loss needing rehabilitation was 3.2 % in a Finnish survey (Rahko et al 1985) among 65 year old healthy people. Rahko et al (1988) later showed that according to the same criteria and based on hearing threshold measurements of 11744 inhabitants of the city of Tampere, 1.5 % of the 35-54 year old people needeed hearing rehabilitation in Finland. More male than female attendants participated in the study and the hearing thresholds of the males were worse (Rahko et al 1988). Using the same criteria than Rahko et al (1988) (hearing 30 dB or worse in 500 Hz, 1000 Hz and 2000 Hz was an indication for hearing rehabilitation) in the present study gave the result that 7.2 % of the attendants should have been rehabilitated. The subjects of the present study were younger than the attendants in the study of Rahko et al (1985), but their mean hearing was worse. It seems that the hearing in subjects with tinnitus might be reduced. Davis (1989) studied the prevalence of self-reported hearing disability among adults (18- 80 years). Part of the individuals were audiologically tested. The level for significant hearing impairment was set at least to 25 dB average through frequencies 0.5 KHz, 1 KHz, 2 KHz, 4 KHz and with it 16 % of the population had a bilateral and 25 % unilateral hearing impairment. 10 % of the population reported bilateral hearing difficulty in a quiet environment.

In a study among tinnitus patients Stouffer et al (1990) showed that only 18 % of their patients, mean age of 49 years, had a hearing threshold at 1000 Hz and 4000 Hz better than 25 dB. Only 16.1 % of the subjects in the present study had a hearing threshold better than 25 dB at 1000 Hz and 4000 Hz in both ears, but the mean age in the present study was higher than in the study by Stouffer et al (1990). A positive, non-linear correlation between the hearing loss and tinnitus seemed to exist according to Chung et al (1984). When hearing loss got higher the amount of tinnitus cases increased at a faster rate. In a survey to search the diseases behind tinnitus Davis et al (1992) found that difficulty to comprehend a spoken language in a noisy situations is a reflection of hearing impairment as a whole but it also combined with the occurence of PST. A history of discharging ear seemed to be an important factor for PST. Chronic suppurative otitis media and a history of childhood otitis media both had some effect. In the present study former otitis media was not related to tinnitus.

The amount of chronic otitis media in the present study was 1.8 %. Ear surgery for chronic otitis media or cholesteatoma had been performed to 1.5 % of the subjects in the present study. Ruben (1992) studied the frequency of mastoidectomy and tympanoplasty in the U.S. and found that in spite of the increase in the amounts of operations during 1971 - 1978 the rate in the population remained the same, between 25-28 operations per 100 000. The rate of mastoiditis in a 100 000 population according to patient discharge from a short stay hospitals in U.S. was 5.7 in 1978 and likewise the rate of cholesteatoma was 4.2. The population in the present study is not comparable with the Ruben study population but more ear operations and chronic otitis media seemed to exist in the present study than in the normal population. Parisier et al (1984) found that the incidence of tinnitus was 34.1 % in chronic mastoiditis and cholesteatoma patient population preoperatively. After the surgery some improved but some cases of tinnitus started from the operation and the postoperative tinnitus incidence was 46.3 %.

2. COMPARISONS OF TINNITUS MEASUREMENTS AND CHARACTER

2.1. Tinnitus presentation in the present study

The way of reporting tinnitus by the amount of sites of its occurence and not by the amount of patients with tinnitus is exceptional. Engelberg et al (1985) gave the result of transcutaneous stimulation in tinnitus. Results were reported in two ways: the number of ears expressing improvement like in the present study and the number of patients with improvement. The tinnitus site "head" was not listed in their study. In the present study tinnitus did not always change similarly in every tinnitus site so this presentation was considered a better way to proper describe all the phenomena during the treatment period. For the same reason both the most troublesome tinnitus (*T1*) and the possible other tinnitus in the same place (*T2*) were reported as different sounds. It made possible to characterize the other tinnitus sounds and not only the most prominent one. Because of the difference in reporting tinnitus the comparisons to other studies are more complicated.

2.2. The duration of tinnitus

In the present study in 54.5 % of the subjects the tinnitus duration was 12 months or less, in 11.4 % of the subjects the duretion was 11 years or more. In many studies the duration of tinnitus has not been mentioned. In a follow-up survey by Goebel et al (1992) with 155 people, the mean duration of tinnitus was 5.8 years, range 1 to 32 years. Hallberg et al (1992) published a study about coping strategies in tinnitus with 72 males. The mean duration of tinnitus was 11.9 years in a group of bothersome tinnitus not needing treatment and 13.6 years in a group with severe tinnitus demanding treatment, no duration of tinnitus was given for the mild tinnitus group. Colding-Jorgensen et al (1992) stated only that in their series of 14 tinnitus patients most patients had suffered from tinnitus for more than 3 years.

The outcome of our treatment is not easy to compare with other treatments. In the literature the number of subjects involved in the treatment series are usually limited and the information on the duration of tinnitus varies. Shemesh et al (1992) mentioned the duration of tinnitus to be longer than 6 months; Briner et al (1992) from 1 to 60 years, average 15.29 years; Brookes (1996) between 1 and 10 years; Landis et al (1992) at least 3 years; Soussi et al (1994) from 6 months to 16 years. Ogasawara et al (1992) treated 99 patients with tinnitus with oxygen under high pressure. The duration of tinnitus was mentioned to be either under 2 weeks or more than 2 weeks and duration affected the outcome of the treatment.The duration of tinnitus did not seem to statistically affect the outcome of the treatment in the present study.

2.3. The site of tinnitus

The present study subjects had more tinnitus in the left side and it was also reported to be louder in that side (48 % the left side, 35 % the right side and 15 % symmethrical and 2 % in the head). Hazell (1981) reported more tinnitus in the left side and so did Meikle et al (1992). Axelsson et al (1989) reported tinnitus in the females: the left ear 31 %, the right ear 26 %, bilateral 31 % and the head 11 % and males: the left ear 36 %, the right ear 19 %, bilateral 47 % and the head 8 %. Stouffer et al (1990) found more tinnitus in the left side (20.1 % of males and 23.0 % of females). If descriptions of the site were summed up tinnitus in the left showed 40.7 %, right 34.6 % and the head or outside the head 10.4 % of the total amount of tinnitus in both sexes. In a unilateral tinnitus study by Ishikawa et al (1992) the affected ear was right in 46.3 % and left in 53.7 %. Axelsson (1996) published a matherial

with 478 tinnitus patients. In this study more tinnitus in the left side was found, in females left sided tinnitus was the most prevalent and in males both sides equal was the most common followed by left side tinnitus. On the contrary in the large NHS investigation (MRC institute 1987) no left side preponderance was found. However, the present study seems to conform the observations of the left side preponderance of tinnitus.

2.4. The pitch of tinnitus

2.4.1. The description of the sound

In the present study tinnitus was reported mostly as high pitched tinnitus. The most common description of *T1* was ringing (42 % in the right and 38 % in the left ear). In the head *T1* sounds whistling was the most prevalent (35 %). With the *T2* sounds wheesing was the most prevalent in the right (22 %) and in the left whistling and chirping were equally common (17 %). When the male and female answers were compared, they differed significantly in the description of *T1* sounds in the ear. No difference was found in the description of *T1* head sounds or in any of the *T2* sounds. Males tended to have mostly ringing (65 %) and females chirping (26 %) or ringing (23 %) in the right ear. In the left ear males had mostly ringing (59 %) and females chirping (27 %) and ringing (21 %). In the study by Stouffer et al (1990) the most common comment was ringing, in 41.3 % of male and 32.8 % of female patients, next was buzzing, which occured in 7.8 % of males and 15.3 % of females. The present study seems to confirm the observations of Stouffer et al (1990) that males tend to have mostly ringing character in their tinnitus and females seem to have more evenly distributed tinnitus character.

2.4.2. The measured pitch frequency

The tinnitus measurements in the present study gave a mean frequency value of 4640 Hz to the right ear T1 sound (but anyhow 50.6 % of the right ear T1 sounds had a pitch 6000 Hz or higher) and 3658 Hz to the left ear T1 sound (but 35.1 % of the left ear T1 sounds had pitch 6000 Hz or higher). That was fewer cases than was expected onthe grounds of the descriptions of tinnitus. Meikle et al (1992) measured also tinnitus pitch and found it to be from 3500 to 6499 Hz in 32.8 %, under 3500 Hz in 25.1 %, from 6500 to 8499 Hz 23.8 % and over 8500 Hz in 18.2 % of the cases. The division of cases in each frequency in the present study is not easy to compare to the study of Meikle et al (1992). In the present study the tinnitus division was different depending on the measured ear. Meikle et al (1992) did not mention the measured ear. The mean frequency was expected to be higher, because in earlier studies subjects themselves usually rated tinnitus high-pitched (Stouffer et al 1990). One reason is probably the audiometry, which enabled the measurements only up to 8000 Hz and higher pitched tinnitus could not be measured and was marked 8000 Hz.

2.4.3 The measured loudness of tinnitus

The mean measured HL tinnitus loudness value of the present study was of 43.4 dB for the right ear and 42.1 dB for the left ear (measured from the ipsilateral ear). In a survey by Ishikawa et al (1992), when the measuring ears were compared, the mean loudness of tinnitus was 49 dB in the ipsilateral and 40 dB in the contralateral side. The mean tinnitus values of Ishikawa et al (1992) are similar to the mean values of the present study. In the present study the mean loudness value of the loudest tinnitus (*T1* regardless of the place) in the VAS- scale was 5.5 and in case of two tinnitus sounds the mean loudness of the secondly loud tinnitus (T1 in another site or T2 in the same site than the loudest tinnitus) was 3.8. If

three tinnitus sounds existed the mean loudness of the third was rated 3.4 and if four the fourth 3.4. The loudness VAS- scale average was 6.3 in the publication by Stouffer et al (1990). The American Tinnitus Association reported an average of 5.4 in a VAS- scale. So the tinnitus loudness values of the present study are comparable to other studies.

When the measured tinnitus loudness values were compared to the hearing level at the same frequency, the difference between the measured tinnitus loudness and the hearing level was often small compared to the amount of distress tinnitus was causing to the individual. In several reports low figures of measured values of the SL of tinnitus have been published. According to Vernon (1987) tinnitus loudness values are mostly only 5- 10 dB above the sensation level. In spite of that people might complain excessively about the distress caused by their tinnitus and it could be assumed that the patients overestimate the loudness (Hazell 1981). More and louder tinnitus in the left side existed in the present study according to pretreatment questioning. But in fact the actual measurements about the loudness of tinnitus before the treatment did not confirm it. Hazell (1981) asked the tinnitus patients to rate their tinnitus in a VAS- scale and then the exact measurements of the loudness of tinnitus were made. No correlation was found between the two measurements (Hazell 1981). The effect of recruitment as an explaining factor has also been suggested, it would explain the intensity and distress caused by low sounds (Vernon 1987).

2.5. The fluctuations of tinnitus

2.5.1. The character of fluctuations

Tinnitus often fluctuated in different situations or positions. In the present study variations in pitch, loudness and expression of tinnitus seemed to exist. Fluctuations occured on everyday basis. *T1* sounds were not heard all the time, the most probable explanation is the masking effect of external sounds during daytime. Real disappearances may also exist, because *T1* often disappeared at least for minutes according to the patients. The *T2* sounds seemed to disappear even more.

In the present study the *T1* tinnitus sounds commonly fluctuated in loudness (38 % of right *T1*, 44 % of left *T1* and 47 % of head *T1*). Stouffer et al (1990) stated that 34 % of their patients reported an increase of the loudness of tinnitus ever since the beginning, 7 % reported a decrease and in 60 % tinnitus had stayed the same. 40 % of their patients had fluctuations of loudness on a day to day basis. Tyler (1992) reported that 31 % of the subjects experienced gradual changes and 23 % sudden changes in the loudness of tinnitus. The tinnitus loudness varied from day to day in 50 % of the patients. Since tinnitus had first begun 59 % considered that it had stayed in the same loudness level but 34 % stated that it had increased. Chéry-Croze et al (1996) analyzed the first 238 answeres and 39 % of the subjects reported fluctuations in the loudness. The comparison of the results of the present study to other studies about loudness fluctuations are not straightforward because of the difference in the tinnitus presentation. Many subjects in the present study had a prominent tinnitus sound in one ear and a softer one in the other. The fluctuations of all sounds were listed in the present study. In the study by Stouffer et al (1990), Tyler (1992) and Chéry-Croze et al (1996) only the most prominant tinnitus sounds were included. More fluctuations in the *T2* sounds occur so also more fluctuations in the *T1* sounds in the non-dominant tinnitus side could be expected.

2.5.2. The factors causing fluctuations

The head movements were expected to be an important agent to explain the fluctuations of tinnitus. But in the present study only a few subjects reported changes in the volume of tinnitus in head or body movements. The position causing most fluctuations of tinnitus was turning head to left ear to shoulder, most of the reported 20 cases were aggravations of tinnitus. Turning head to forward chin to the chest caused 18 cases of fluctuation, turning head right nose to shoulder 17 cases, turning head left nose to shoulder 16 cases, turning head right ear to shoulder 15 cases and turning head back 8 cases. It was assumed that some tendency could be noticed between the direction of the turning of the head and the influence and site of tinnitus like turning head right would aggavate the tinnitus in the left ear and decrease the tinnitus in the right ear and vice versa. No such tendency could anyhow be noticed. Axelsson (1998) noticed more aggravation than relief from tinnitus in different positions. Turning head right caused increase of tinnitus in 13 % of the cases in both ears and relief in about 3 % of the cases in both ears. Turning head left caused similar changes. Turning head forward increased tinnitus in 8.6 % of the cases in the right ear and in 9.5 % in the left ear. Turning head backwards caused increase of tinnitus in 12.8 % of the cases in the right ear and in 14.3 % in the left ear. Stouffer et al (1990) reported aggravation of tinnitus in 16 % of their patients when changing head position. The results of both above groups were similar to the results in the present study. If only aggravations were counted from all the positions, increase of tinnitus in the present study was reported in 24 % of the cases with right ear tinnitus and in 22 % of the cases with left ear tinnitus. The difference in the presentation of tinnitus makes the comparison difficult. In the present study all the offered positions might have affected the tinnitus in some subjects and in addition in both ears; the above results have been obtained by counting the percentages of all the cases and all the positions. Stouffer et al (1990) counted subjects with tinnitus and not ears or heads with tinnitus as in the present study. The figures of Axelsson (1998) are probably more comparable and similar to the results in the present study.

Not many reports with the effect of body or head positions could be found. The possibility that such a phenomen exists was not even mentioned in the causes of tinnitus-list of Axelsson (1992) or Davis (1996). Van den Abbeele et al (1992) mentioned that 35 % of their patients experienced the increase of tinnitus in different positions. Unfortunately they did not define the term position. In the present study some of the tinnitus sounds could only be heard in a specific body posture (19 cases) or in a specific head position (3 cases). Any comparable earlier reports of intermittent tinnitus sounds that appear only in a specific body or head position could not be traced.

In the present study one subject had a tinnitus which was audible only in weather with low pressure and disappeared in high pressure time. The same phenomenon was mentioned by Berliner et al (1987), who stated that even 23 % of their cochlear implant patients had aggravations of tinnitus with the change of weather. In present study fluctuations of tinnitus were reported due to temperature changes in 41 cases. Also sauna caused fluctuations of tinnitus, mostly worsening, but nearly 40% of all the reported fluctuations (23 of the total of 59 cases) observed relieves.

The influence of stress is a well known factor and in the present study 41 % of the cases with right ear tinnitus, 39 % of the cases with left ear tinnitus and 11 % of the cases with head tinnitus experienced an increase in physical stress. The psychological stress increased tinnitus in 27 % of the cases with right ear tinnitus, in 25 % of the cases with left ear tinnitus and in 28 % of the cases with head tinnitus. The aggravating influence of different kind of

stress has been reported also by others. House (1981) considered the relationship so that stress makes people less tolerant to tinnitus but does not, de facto, increase tinnitus itself. Evans et al (1981) listed stress and fatigue as exacerbating factors but did not estimate the importance of them. Dauman et al (1992) considered the influence of stress on tinnitus to be mediated through the autonomic nervous system.

The aggravating factors have also been listed in other studies and they seemed to be quite the same as were discovered in the present study. Van den Abbeele et al (1992) reported heightening of tinnitus in 35 % of their patients according to position, 31 % in loud noise, 15 % in altitude, 13 % with alcohol, 6 % with caffeine and 8 % when coughing. Berliner et al (1987) stated that with cochlear implant patients the most important aggravating factor seemed to be fatigue, 51 % of the patients reported it. Different kinds of stress were important and aggravations were reported with excitement in 28 %, lack of sleep in 21 %, nervousness in 40 % and anger in 16 %. Head and body positions affect in 12 %, physical activity in 26 % and even constipation in 4 %. Stouffer et al (1990) showed that a noisy or a quiet environment both seemed to increase tinnitus severity in one third (31- 36 %) of the subjects. Stress was an important factor for nearly as many (29 %). In the present study no statistical difference between the sexes existed concerning the effect of head and body position on tinnitus. Stouffer et al (1990) reported a statistical difference between sexes in two situations: changing head position and when first waking up in the morning, in both cases female patients reported more aggravations of tinnitus.

3. OTHER TREATMENTS IN THE STUDY GROUP

In the present study no benefit was achieved by drug usage in the pretreatment period, as no statistically significant improvement was found. Betahistine is a cerebral and cochlear vasodilator. Betahistine was tried by some subjects but it did not have a statistically significant effect on their tinnitus, single positive effects were however reported. The dose of betahistine in Ménière disease was raised higher by Coles (1996) than recommended by the manufacturer, namely 24 mg or even 32 mg three times a day (instead the normal 8 mg or 16 mg three times daily). The higher dose was found to be beneficial in Ménière patients with inadequate symptom control with normal doses. Coles (1996) did not give any facts about its usage by tinnitus patients without Ménière disease.

None of the subjects using antiepileptic drug and having simultaneous tinnitus reported any help for their tinnitus from the medicine, but antiepileptic drugs were not tried in tinnitus control in the present study. Antiepileptic drugs have been used for tinnitus treatment in several works (Goodey 1987). Guth et al (1992) listed drugs that have recently been used in the treatment of tinnitus and carbamazepine, primidone and valproate were in that list. Goodey (1987) considered carbamazepine as a drug of choice for tinnitus patients but he did not recall the effectiveness of it. In another study by Goodey (1981) a relief but not disappearance of symptoms with carbamazepine was achieved in 62 % of those tinnitus patients that had responded to the lidocain test. A newer anticonvulsive agent vigabatrin was reported to have shown some effect, but the number of subjects included in the study was limited (Beck et al 1992).

In the present study more benefit than disadvantage from the usage of alcohol was reported, a decrease of tinnitus occured in 25 cases and increase of tinnitus in 13 cases. Berliner et al (1987) reported only aggravation of tinnitus with alcohol. Excess alcohol seemed to induce tinnitus according to Goodey (1987). Goodey (1981) reported that some tinnitus sufferers seemed also to get help from alcohol. Stephens et al (1996) reported that 20 % of their

tinnitus patients used alcohol regularly because it helped their tinnitus. In the present study one male patient stated that only alcohol could keep his tinnitus on a reasonable level and he found it to be the only effective means of treatment.

4. THE SUBJECTIVE POTENTIAL ETIOLOGY OF TINNITUS

The etiology of tinnitus was unknown for a considerable number of subjects in the present study. Many causes may affect the final outbreak of tinnitus so the question was: “What do you think about this subject, could it have influenced the outbreak of your tinnitus?” Noise trauma (19 %) and muscular tension (20 %) were the most important factors. Hazell (1996) listed the factors, which trigger the onset of tinnitus and found workstress the most important (18 %), followed by domestic stress (10 %), negative counselling (9 %), acoustic trauma (8 %), surgery (8 %), injury (7 %) and acute respiratory illness (6 %). Stouffer et al (1990) asked patients: “What do you think originally caused your tinnitus?” Nearly half had no answer but 14.2 % considered noise and 12.9 % hearing loss. In Axelsson’s (1998) study NIHL was the most important (39%), followed by cervical spine degeneration (23 %), head trauma (20 %), hereditary hearing loss (18 %), malocclusion (16 %), barotraumatic hearing loss (16 %), hypertension (13 %) and heart problems (11 %). Axelsson (1998) also listed the most important co-factors for the onset of tinnitus and muscular tension was the most important 15.5 %. NIHL is an important cause of tinnitus in several reports (van den Abbeele 1992, Evans 1981, MRC Institute of Hearing Research 1987). Davis (1996) reported that only 4 major risk factors of tinnitus could be detected in his study : reported hearing disability, history of runny ears, low self regard of health status and for bilateral cases prolonged use of aspirin; other factors did not show any statistically significant increasing risk for tinnitus. The lists differ considerably from each other. The most likely explanation is the difference of questioning. The possibility of selecting more than one cause can explain some of the differences. The tinnitus contributing factor list of Axelsson (1998) is in agreement about the results of the present study.

5. TINNITUS- RELATED DISORDERS

Hyperacusis was reported by 40 % of the subjects in the present study at least sometimes in the right ear and 38 % in the left ear. That is more than in the report of Gabriels (1996), who reported sensitivity to sounds and a few percent of hyperacusis in 20.1 % of the subjects in her tinnitus material. The difference is probably the confusion of the terminology and the “part-time hyperacusis” in the present study (subjects, who had intolerance to sounds only part-time and the condition varied). Probably more intolerance to sound exists than is realized if the condition is expressing itself only occasionnally so subjects do not complain if not asked systematically. Accorging to Reich et al (1992) hyperacusis is by definition an unusual intolerance of normal environmental sounds, causing pain to the individual. Recruitment is an abnormal increase of the loudness of sounds making them intolerable loud. In spite of the clear definitions of these conditions, a confusion with the terms and expressions exist and terms like insensitivity to sound can mean either one of them. Jastreboff et al (1993) considered hyperacusis to be a pretinnitus phenomenon. Hazell et al (1992) considered hyperacusis to be a part of global sensitivity, because they found 20 % of their patients hypersensitive to other than auditive stimuli for instance to bright light or pain. Reich et al (1992) showed that 63 % of 104 hyperacusis patients had either temporomandibular joint disorders or some kind of problems with the bite. The amount of tinnitus among these patients was not mentioned but 75 % complained of the feeling of

fullness in their ears and also some of them had sensitivity to light and suffered from headaches or dizziness.

All the other tinnitus related disorders (fullness of the ear, pressure in the ear, distortion of hearing and diplacusis) do occur also without tinnitus, but in the present study they seemed to occur nearly always in the tinnitus side expressing the close relation to tinnitus. Fullness and pressure were experienced more in the left side like tinnitus. Gordon (1997) listed the disorders of Hear syndrome (hyperactive ear) to be tinnitus, audiosensitivity, hyperacusis, fullness of ear, dizziness, ataxia, pitch distortion, circadian variation, insomnia, depersonalization, neurashenia, nausea and vomiting or stomache upset, fear and paranoia and susceptibility to many drugs and motion sickness. In the TMJ- syndrome the disorders besides tinnitus were, according to Hazell (1987), fullness of the ear, dysacusis, tension headache and dizziness. Chole et al (1992) investigated 1032 subjects to find out the actual prevalence of otologic syndromes among temporomandibular joint disorder (TMD) patients compared to the normal population. The occurence of tinnitus, dizziness, vertigo and otalgia were statistically significantly more common in the TMD- group. The TMJ-area was not studied in the present study but some of the above listed disorders existed also in some subjects of the study.

Subjective hearing loss is a phenomen, when the subjects have a normal hearing level measured by the audiometer but complain about insufficient hearing and especially difficulty to comprehend spoken information although they hear that something is spoken. In the present study 5 subjects with hearing in both ears 25 dB or better complained about insufficient hearing. Hosoda et al (1993) gave intravenous lidocain to tinnitus patients and used contingent negative variation (CNV) audiometry to measure hearing thresholds at the same time. In the tinnitus- decreased group a CNV threshold decrease (4- 6 dB) was found in 22 % of the cases but in the tinnitus- unchanged group only small changes were found. They came to the conclusion that tinnitus itself has a masking effect like an external sound The clearing up of hearing can be explained by the diminishing of tinnitus in tinnitus patients, but the author of the present study has discovered it also in patients with subjective hearing loss without tinnitus. So far not enough of these patients exist to make statistical analysis of the used treatment in this disorder but the results seem to be repeatable in some patients. In the discussion of a seminar presented by Goodey (1981), Jackson and Goodey both reported that with intravenous lidocain some small or no differences in hearing thresholds could be detected, but people stated that they could hear more clearly.

6. TREATMENT

The treatment was accomplished by two ways: spray and strech method and trigger point injections, both standard ways to treat pain. Fluori-Methane is a vapocoolant, which is applied over the muscle with restriced motion and the muscle is streched to its full length (Travell et al 1983). It resembles in that way a regular physiotherapeutic treatments. Comparing Fluori-Methane spray streching and normal physiotherapeutic treatment is difficult. Only one subject reported a relief of tinnitus after regular massage but in several (41) occasions Fluori-Methane was used successfully alone. In the first treatment 15 cases were treated only with Fluori-Methane and in the second treatment 8 cases. After the second treatment all the individuals treated with Fluori-Methane alone responded.

Trigger point injections are beneficial to certain patients but not for all. Lidocain or procain are commonly used local anesthetics (Travell et al 1983). Other local anesthetics and isotonic saline have also been used. No difference in efficacy or mode of action have been

found between them (Han et al 1997). The method in the present study did not differ from the treatments used for pain.

7. RESULTS

7.1. The effect on tinnitus

In the present study at least some benefit was experienced by nearly half of the subjects with tinnitus located in the ear and one third of the cases with head associated tinnitus after the first treatment. In the second treatment the amount of cases feeling at least some relief was over 40 % in ear associated tinnitus and 27 % with head tinnitus. The positive results were also confirmed by measuring the loudness of tinnitus. The difference in the pretreatment and posttreatment *T1* values was about 4 dB (30 min after the treatment) and that was statistically significant in ear associated tinnitus ($p<0.01$ for the right ear and $p<0.001$ for the left ear) in the first treatment. In the second treatment the mean *T1* loudness change was about 4 dB ($p<0.01$ for the right ear and $p<0.001$ for the left ear). Earlier reports about measured tinnitus volume changes after treatments were not available.

In the present study frequency changes could be produced to 5.5 % of the cases with right *T1* and in 5.1 % of the cases with left *T1* in the first treatment. No published reports of the measured frequency changes after tinnitus treatments were available. The site of tinnitus changed in several treatments. The from-ear-to-another-ear cases could be explained by the possibility of already existing softer tinnitus in the contralateral ear, sensed when the most prominent tinnitus was removed. The site of tinnitus moved from ear to ipsilateral occipital area in three subsequent treatments in one subject and moved from ear to be heard outside the head in one subject in 10 minutes.The site changes might be explained by the genuine effects of trigger points: If the most active trigger points have been inactivated, the nearby latent ones might activate and produce new symptoms to a new area (Travell et al 1983). This is also known with pain treatments, all the pain producing trigger points should be inactivated otherwise the pain is only moved to another area (Travell et al 1983). The muscular status in the cervical area, the shoulder area and in the scapular area in the present study was found to be more tensive in the tinnitus side.

7.2. Why the effect is not from lidocain alone

Lidocain is known to suppress tinnitus by intravenous administration (Goodey 1987). It is also used as a test to investigate the behavior of different types of tinnitus (Goodey 1987). In an experiment with 2 mg / kg body weight lidocain administered intravenously 63 % of the patients experienced either partial or total relief from their tinnitus for 10 to 30 minutes, occasionally up to three days (Goodey 1981). Israel et al (1982) with intravenous lidocain in a double-blind cross-over study could confirm its effect when compared to saline. Duckert et al (1984) reported that 100 mg bolus of intravenous lidocain caused relief from tinnitus in 40 % of the subjects. It also made tinnitus worse in 30 % of them and 80 % suffered from side-effects. 20 subjects of them were selected and explained, that they would be given lidocain. Instead they were given intravenous saline and 40 % of them responded to saline. The authors considered that a double-blind test protocol is not possible with lidocain because of the high amount of lidocain-combinding adverse effects. The subjects who received lidocain earlier could detect which substance they were receiving. Thus the effect of lidocain was partly produced by the expectations of the patients.

The results with local lidocain injections in the present study could not be from lidocain alone via accidental intravenous administration for several reasons.

1) The intravenous injection was careful avoided by repeatedly controlling the place of the needle.

2) The amount of lidocain was too small (mean amount under 30 mg of lidocain) to cause the responses.

3) Among the subjects of the present study were 10 individuals who had had some local operation accomplished in the pretreatment period while already suffering from tinnitus. Lidocain was used subcutaneously to get local anesthesia but in no case any effect on the tinnitus was experienced.

4) Similar responses have been achieved with saline injections than with lidocain in treating pain. The effects of injections without lidocain have been demonstrated with the trigger point injections in other publications (Han et al 1997, Travell et al 1983).

5) Duration of the result; the influence of lidocain lasted 10 to 30 minutes, occasionally up to 3 days (Goodey 1981). In the present study the effect varied from 2 weeks to up to 6 months after the second treatment in 9 % of the cases with ear associated tinnitus. Because the non-responders were advised to stop the treatment after the second treatment and if they were unsure at least after the third treatment, all the subjects attending the fourth treatment had responded earlier. After the fourth treatment an effect lasting at least a week was experienced by more than 30 % of all the cases with tinnitus. Because the effects of lidocain were mostly under 3 days and considerable amount of effects in the present study lasted longer than 3 days, the explanation by the effect of lidocain seems unlikely.

6) The time lag between the treatment and result was experienced by more than a third of the subjects in the present study. In more than 6 % of the cases the response was delayed 2 to 7 days after the treatment. Lidocain effect starts instantly (Goodey 1981).

Wyant (1979) published two tinnitus patient cases undergoing treatment of pain with trigger point injection. They both got benefit for their pain, but in addition their tinnitus improved repeatedly. Each of the following places were injected with 1% lidocain 15 ml and triamcinolone 60mg. The used injection points with patient 1 were 1) left paraspinous muscle at the level of C2 close to midline, 2) another point 1 cm lateral from the previous, 3) 1 cm distal in relation to the upper fibres of the trapezius muscle, 4) 1 cm further distal from it, 5) within the sternocleidomastoid 0.5 cm cephalad to the fourth one, 6) 1 cm cephalad to the fifth one. The injection points with patient 2, with right ear tinnitus were 1) right scalenus medius, 2) right splenius capitis. With injection of each place with lidocaine 0.5% 2.5 ml and triamcinolone 20 mg the pain and ringing of the right ear ceased. The recurrence of tinnitus was successfully re-treated by injecting additional trigger points and with repeated injections. Patient 1 had a maximum relief for four months and patient 2 for four weeks after injections. The places of injection and the duration of the relief from tinnitus are similar to the present study. The trigger points were ipsilateral to tinnitus in the patient 2, the observation is identical with that of the present study. Biesinger (1998) presented a treatment program of tinnitus with functional treatment of upper cervical spine. The fundamental components of the therapy were physical therapy and manual therapy by a physican trained as an orthopedist. He used also local anestethic to the insertion of sternocleidomastoid muscle at the mastoid; to the insertion of superior obliquus muscle, major rectus posterior muscle and minor rectus muscle of the head; to spinal processes of the axis and to the insertion of the levator muscle of the scapula at the level of C2-C3 and sometimes at its insertion. They were all (especially the last one) commonly used injection places also in the present study.

In the present study 24 cases with right ear tinnitus and 36 cases with left ear tinnitus experienced complete disappearance of tinnitus either instantly or later during the

posttreatment period after the first treatment. In the second treatment 25 cases with right ear tinnitus and 39 cases with left ear tinnitus experienced a complete disappearance of tinnitus. This effect could be produced repeatedly to some subjects (twice to 9 cases with right ear tinnitus and 15 cases with left ear tinnitus; the maximum was 5 times in consecutive settings). The placebo effect seems a very unlike explanation for the subjective and objective improvement in tinnitus.

The treatment effects were asked 6 months after the treatment and subjective evaluation of improvement had been experienced, depending on the tinnitus site, by about one third or more of the treated cases. In the control group one fourth or less reported improvement after similar waiting time. The difference after 6 months was statistically significant with right *T1* ($p<0.001$) and with left *T1* ($p<0.01$) but not with head tinnitus. As can be perceived, the tinnitus sounds can improve also spontaneously even in a considerable amount of cases. Because of the long observation time and statistically significant difference between the groups explanation of the result by placebo effect alone is unlike.

7.3. The results for other disorders

In the present study the pressure in the ear improved in 9 ears out of 38 treated in the first treatment and ten out of 38 treated in the second treatment. The fullness of ears improved in 12 cases out of 59 treated in the first treatment and in 18 cases out of 58 treated in the second treatment. The result indicates positive outcome for one third of all the ears treated for tinnitus and having a tinnitus-related problem at that time (in part of the ears intermittent problems existed and they were symptomfree during that time). The fullness and pressure in the ear with tinnitus have been reported in temporomandibular disorders (Chole et al 1992, Vernon et al 1992). Treatment experiments with these symptoms were not found.

The hearing disorders often present with tinnitus were hypacusis, hyperacusis, cracking of sounds and diplacusis. Hypacusis is mostly connected to tinnitus also as a causative agent. Tinnitus might itself produce hypacusis. In the present study hearing improved subjectively in 3 ears out of 256 treated after the first treatment. In the second treatment subjective improvement of hearing occurred in 10 ears out of 207 treated. The connection of cervical disorders and hypacusis has been reported earlier (Hülse 1994, Brügel 1991, Pihakari 1998). Objective improvements in audiograms have been produced with manipulation of the cervical area according to Hülse (1994). Brügel (1991) reported that hearing defects might be caused by manipulations of the cervical area and advised to be careful with them. Hyperacusis is a distressing symptom with no proven widely accepted means of treatment. In the present study hyperacusis improved in 13 ears out of 69 ears treated in the second treatment in 11 ears out of 65 treated in the first treatment. One subject needing hearing rehabilitation suffered from severe hyperacusis and was inable wear a hearing aid. She was successfully desensitized to sounds while her tinnitus was treated and after more than a year with repeated treatments hyperacusis subsided and she was able to wear a hearing aid. Hyperacusis has been treated by maskers by sensitizing the patient to constant noise, Hazell (1987) reported a reduction of loudness discomfort with maskers. In a study with 30 patiens with hyperacusis, 73 % of them reported improvement after using a masker for 6 months (Hazell et al 1992). The effect of maskers in treating hyperacusis has been questioned by Gabriels (1996) who considered that part of the effectiveness might just be time and not the device. She reported good results with maskers in spite of skepticisim against the treatment of hyperacusis with them. The number of cases with hyperacusis in the present study is too small for comparing the results. The cracking of sounds and diplacusis improved in the first treatment in 4 ears out of 24 ears treated and could be reproduced in the second treatment to all of them. Most of the treatments did not give permanent cure to any of the symptoms, but

the good effects could usually be reproduced for the responding patients in every treatment just like most of the cases with tinnitus.

8. PSYCHOLOGICAL ASPECTS IN TREATING TINNITUS

In the present study fluctuations of tinnitus occured in stressful situations. In many reports tinnitus patients have more depression and stress or they have inability to relax when compared to subjects without tinnitus (Stephens 1992, Tyler et al 1983). Succesful treatments of tinnitus with relaxation (Hallam 1987, Hazell 1987), biofeedback (House 1978, Landis 1992, Lindberg 1987, Grossan 1976) and other similar therapies (Hallam 1987) have been published. They include psychological caring of the patient enabling him to cope with the situation (Hallam 1987, House 1981, Goebel 1992, House 1978, Landis 1992). Tinnitus in several subjects is alleviated by proper investigation of the disorder and discussion with a therapist (Coles 1992). According to Coles (1992) even half of his patients are sufficiently helped by proper counselling. In the present study the effect of counselling cannot be underestimated. The control group was given the same counselling and investigation, but treated group had in addition the treatment visits that allowed more frequent counseling and may bias the results.

The results in this study are subject to a lot of criticism. With an open study all the participants could choose themselves whether or not they wanted to take part in it. That will favour individuals who are optimistic and maybe easily convinced of new ideas. Duckert et al (1984) did a survey with a 5cc bolus of placebo saline injection given intravenously to 20 people suffering from tinnitus. 40 % of them reported a change of their tinnitus after the injection. 6 people reported a decrease and 2 an increase of at least 25 %. Unfortunately they did not tell how long the placebo effect lasted. It is hard to believe that all previous results are placebo phenomena because the first time tinnitus disappeared, it was the patient who reported that her tinnitus was gone. The longest results with this method were more than a year tinnitus-free periods. Besides the relief to tinnitus could be produced over and over again with some individuals. The patients were a heterogenous group. It would have been easier to compare individuals with the same type of tinnitus and the same type of hearing to each other. But because tinnitus itself was investigated too, it was good to have as many types of people and tinnitus as possible in the research.

9. THE CONNECTION OF TINNITUS AND MUSCULAR TENSION

In the present study the side of muscular tension in the cervical area was connected to the ipsilateral tinnitus in both ears ($p<0.001$). A similar connection could be detected from the shoulder area ($p<0.001$) and in the scapular area ($p<0.01$). Tinnitus and vertigo occured more often with TMD patients compared to the normal population (Chole et al 1992). In 338 of TMD patients tinnitus and vertigo symptoms were significantly more prevalent (59.0 %) than in the 2 control groups (13.8 % and 32.5 %) (Chole et al 1992). Bjorne (1991) reported that lateral pterygoid muscle seemed to be more tensive in the tinnitus side and with lidocain injection a relief from tinnitus was achieved. Vernon et al (1992) tried to find symptoms associated with TMD generated tinnitus. They could not find any conclusive symptoms but there seemed to be more pain and fullness in the ears, tinnitus was more complex, for instance it consisted of several sounds, was difficult to mask and had often a gradual onset. Rubinstein et al (1992) reported a significantly higher prevalence of frequent headaches and symptoms of cranio-mandibular disorder (ear, facial and cervical pain, swallowing and chewing difficulties, limitations of mandibular movements and jaw stifness) in tinnitus

patients. Bruxism was also three times more prevalent in tinnitus group. The connection of tinnitus and cervical disorders has been suggested also earlier, Brügel et al (1991) described tinnitus and low- frequency hearing loss in patients with cervical disorders or after cervical manipulations. The connection of trigger points and tinnitus was noticed by Eriksson et al (1996), who reported a greater number of trigger points in the tinnitus patients compared to controls. Cervical spine degeneration was also the most important contributing factor for tinnitus according to Axelsson (1998).

The effects of cervical muscular tension to tinnitus has not been reported with this extent earlier, because the side of tension and the side of tinnitus have not been mentioned in several earlier reports (Rubinstein et al 1992, Axelsson 1998). The results of the present study point out that a resiprocal connection between these two variably exists although their internal relationship may be complex. The tension seems to follow a hearing defect after acoustical trauma and in these cases tinnitus and hearing defect are the first to appear. Hülse (1994) reported a connection between cervical receptive field and cochlear nucleus. That might also explain the results with the connection of the side of tension and ipsilateral tinnitus. He found also a connection between hearing disorder, otalgia and cervical tension; they occured in the same side. Some of the patients in the present study reported improved hearing after the treatment. Hülse (1994) had patients with subjective hearing disorders, which were reversible after manipulation, and reported improvement in the audiometric recordings. The experiences of Wyant (1979) point to the fact that it is important to find all the trigger points and inactivate them all, otherwise tinnitus returns. The patient of Wyant, who had the sidedness of tinnitus mentioned, had trigger points in the ipsilateral side.

Chole et al (1992) quote twelve studies containing 25 to 1391 patients with TMD, 14 -76 % of them had tinnitus, 3 -100 % had otalgia and 6 -62 % had fullness of the ear. The authors evaluated the reasons for that coincidence according to theories expressed by others. First of them is Eustachian tube blockage but it has never been shown to actually happen. Second is hyperactivity in masticatory muscles via trigeminal nerve but it would make tinnitus objectively audible which is uncommon. The TMD-associated aural symptoms have also been explained as a reflection to a direct mechanical stimulation of malleus through the anterior mallear ligament, which has shown to exist in a human fetus. By Excessive Somatic Concern Hypothesis tinnitus is generated by underlying distress. The theory is based on the observation that there are more people with emotional disorders among TMD patients. The authors did not express their own theory about the coincidence between TMD and otological disorders (Chole et al 1992).

Ferber-Viart et al (1998) noticed that brief intense clicks cause short-latency cervical muscle microcontraction, verified by recording of myogenic vestibular evoked potentials (MVEP). They recorded trapezius reactions and found them to depend on both vestibular and cochlear afferents. Colebach et al (1994) found some similar reactions to brief intense clicks on EMG recordings from sternocleidomastoid muscle originating from cochlea. Cooper et al (1991) treated people with craniomandibular disorders with transcutaneous electrical neural stimulation. The patients complained of dizziness, tinnitus, otalgia and fullness of ear. The electromyographic recordings were made bilaterally from masseter, anterior temporalis and digastic muscles. The creation of new occlusal position with an ortodontic appearance correlated with a significant decrease of complaints and also the electromyographic recordings were normalized, thus the normal muscle activity seemed to be connected to the reduction of otological symptoms and vice versa.

10. THEORETICAL EXPLANATION MODEL OF THE TREATMENT

When comparing the results of all tinnitus treatments a strikingly equal benefit with completely different methods seems to exist (Doyle et al 1987). Drugs, operations, maskers, trigger point injections or relaxation therapy may not all affect the same locus of tinnitus generation. The knowledge about inner ear in tinnitus is limited (Kemp 1981, Wilson 1987, Johnstone 1992). Various diseases are known to connect with tinnitus like TMD, depression and pain. (Rubinstein et al 1992, Chole et al 1992, Gordon 1997, Tyler et al 1983)

It seems obvious that tinnitus is not generated the same way in every case. It is generally considered a symptom and not a disease itself. In Ménière disease the cause of it is probably within the inner ear. This however might not be true with other types of tinnitus. In a study by Wable et al (1996) about perilymphatic pressure, the tinnitus patients were compared to age-matched or hearing-matched controls and no systematic differences in perilymphatic pressure were obtained. In noise induced hearing loss the degeneration of OHCs has been shown in the electromicroscopic pictures (Ward 1980, Nielsen et al 1986). The weakness of the explanation of OHC loss as the only causative factor is the fact that it exists also in ears with only hearing loss without tinnitus (Hinojosa 1980). In addition the hair cells seemed perfectly normal in some cases with tinnitus (Vernon 1977). By light microscopy no specific morphologic changes could be found post mortem in the cochleas of tinnitus patients compared to controls, in fact more hair cell pathology was found in the controls. The difference was not significant (Oliveira et al 1990).

Some authors consider the central nervous system at least a part of tinnitus generation (Romand 1992). Central nervous system could act as a modulator so, regardless of the generation place of tinnitus, the detection of it could be switched on or off by the central nervous system. The influence of stress to the fluctuations of tinnitus seems to point some kind of modulator role of central nervous system in the sensing of tinnitus. Hazell considered the influence of trigger point injections to be mediated via the routes from the ear to the conscious mind. The tinnitus as a phenomen does still exist after the treatment, but the route to the higher centers is blocked and the tinnitus is no longer detectable (Hazell 1993).

A lot of the knowledge about the morphologic changes in inner ear in tinnitus have been studied with rats, guinea pigs and cats with salicylate-induced tinnitus (Jastreboff 1992, Kellerhals 1992, Eggermont 1992, Jastreboff 1994). It is hard to know if chemically induced tinnitus is similar enough to human tinnitus. Part of the animal work has also been accomplished by noise-induced tinnitus but the noise has always been very intense sound of a short duration, which is not the most common way to get tinnitus in humans (Axelsson 1992).

Levine (1999) published his theory of two different kinds of tinnitus: somatic (craniocervical) and otic tinnitus. He considered otic tinnitus to be due to disorders of the ear. Somatic tinnitus can begin even with perfecly normal ear, the reason is often some disorder in the head or upper neck. Other characters of somatic tinnitus are localization of tinnitus ipsilateral to the somatic disorder, no vestibular complaints, no abnormalities in the neurological examination and normal hearing. Levine (1999) considered somatic tinnitus to be modulated at the site of CN, especially in DCN, because it is the site on auditory pathway where the nonauditory inputs interact with the auditory system. At higher centers the neuronal crossing is higher and tinnitus is perceived binaurally.

The autonomic nervous system has a very rich network in the cervical area and it continues all the way up to the inner ear and central nervous system (CNS). The cervical sympathetic

system has an origin in the CNS. The postganglionic fibres follow blood vessels, cervical or caudal brain nerves to their destination (Spoendlin 1981). Another, perivascular, adrenergic innervation seems to exist in the inner ear, originating from the stellate ganglion (Spoendlin 1981). Laurikainen et al (1993) concluded that there must be a dual sympathetic innervation (stellate and superior cervical) in the control of cochlear blood flow. Vass et al (1997) described also connections from trigeminal ganglion to the cochlea.

The active trigger points can cause autonomous reactions referred to a distance from the point itself (Travell et al 1983). It is possible that tinnitus, which can be treated through trigger points, is also mediated by autonomic nervous system via the route described by Hülse (1994). Aran et al (1992) pointed that tinnitus might be related to a peripheral impairment which can trigger abnormal autonomous functioning in the central auditory system. That way it is understandable that tinnitus can diminish or even disappear when the influence of the autonomic nervous system to the inner ear is altered. It would also explain the autonomic phenomena that are reported sometimes by the patients when they are treated (thermal changes, increase in the nasal discharge). Levine (1999) considered the DCN to be the coordination center of tinnitus. Both otic and somatic tinnitus activate the output of DCN to higher centers. He considered that the sensory inputs go via trigeminal nerve and the spinal trigeminal tract from the face; via vagus nerve, facial nerve and glossopharyngeal nerve from the external and middle ears and via C2 dorsal root and fasciculus cuneatus from the neck to medullary somatosensory nuclei (MSN). From MSN the fibers project to ipsilateral DCN.

If the autonomic nervous system plays a part in tinnitus generation it might explain the fact that tinnitus does not disappear with the dissection of the cochlear nerve. The influence of autonomic nerves does not disappear with that because the autonomic innervation of the inner ear does not follow the cochlear nerve.

In the present study the positive outcome seemed to correlate with female gender, chirping and humming character of the tinnitus sound and ipsilateral tension in the cervical and shoulder area. No other clear signs to indicate positive outcome could be found. Positive signs were expected to be short duration of tinnitus, normal or nearly normal hearing, spontaneous disappearances or fluctuations in tinnitus, low-frequency character of the tinnitus sound (especially non-tonal tinnitus sound matched to a narrow band noise) and clear trigger effects with head or body movements. Neither of those seemed to be very important. All of the positive signs could be explained with female gender. Women have more chirping or humming character in their tinnitus, women seemed to have more tension in the cervical area (Travell et al 1983) and also the tinnitus in women seemed to respond more to trigger point injection techniques.

Different routes in tinnitus generation probably exist and one of them has a statistical correlation with the ipsilateral muscle tension. It is possible that one of the originating areas of this kind of tinnitus is the autonomic nervous system. The route may proceed via the route described by Hülse (1994), Biesinger (1998) and Levine (1999). Even tinnitus caused by loud noise could be explained with reactions originating from the autonomic nervous system. The first place of detection of noise is the inner ear. Too loud a sound causes a shock reaction in the inner ear and the process proceeds via the autonomic nerves combined with the vascular system or through the nucleus cochlearis. The route leads retrogradically from nucleus cochlearis to the cervical area causing reflectory muscular spasm to the corresponding neural arch reflectory area. This kind of reactions in the sternomastoid muscle have been recorded with EMG after brief clicks played through headphones by Colebach et al (1992), the recording place was upper half of each sternomastoid muscle. In an other study by Colebach et al (1994) in all of the 10 volunteers the 95 dB click was followed by tonic

neck flexor activation. Ferber-Viart et al (1998) studied myogenic vestibular evoked potentials (MVEP) after brief intense clicks. The recording place was on both trapezius muscles. In case of total cochleovestibular damage the MVEP was absent, in case of either cochlear or vestibular lesion the MVEPs could be recordered but the amplitudes were reduced. This reflex arch from ear to the cervical area would explain the observation that after acute noise induced unilateral acustical trauma the ipsilateral cervical area is more tensive than the contralateral side (personal observation).

In reflex dystrofia, a mysterious disorder occuring mostly in the limbs, the causality of the disorder is explained with following theory : After some, sometimes only a minor accident in the limb, the autonomic nerves start overreacting causing swelling, cutaneous changes and very intense pain in the limb. The developement of such a disorder necessitates a natural sensitivity of the patient and abnormal sympathetic reflex. The pain is not managable with anti-inflammatory analgesics and the situation leeds to a severe handicap when allowed to proceed to the irreversible point. If it is promply managed with treatments blocking the sympathetic nerves, the progress of the disorder might be reversed (Pohjolainen 1995). In the study by McNulty et al (1994) the trigger points were shown to react during psychological stress although the other parts of the same muscle (trapezius) were electrically silent. The tension in the trigger points is supposed to be mediated by the autonomic nervous system but the nontrigger parts of a muscle are mediated by voluntary corticospinal nervous system (McNulty et al 1994). Levine (1999) pondered the differences in susceptibility to tinnitus, some otic or somatic insult will cause tinnitus to some individuals and not to others. One explanation could be the somatic-auditory interactions. He considered that it might explain why 1) some patients with a hearing disorder develop tinnitus and others do not, 2) some patients develop tinnitus with chronic progressive hearing loss and others do not, 3) patients with symmethric hearing loss can develop tinnitus to only one ear and 4) how craniocervical movements can modulate tinnitus.

Wyant´s (1979) and my experiments with trigger point technique in tinnitus treatment seem to bear some similarity with the result of reflex dystrofia. It could thus be assumed that at least some of the tinnitus cases are in fact triggered first by some disorder, disease or environmental event. The development of a minor disorder to a chronic tinnitus state is caused by the overreaction of the autonomic nerves just like in reflex dystrofia. If that is the case tinnitus should be quickly treated in order to prevent it to become a chronic just as is the case with dystrofia. This assumption needs of course still a lot of studying but seems to be in agreement with the theory of Levine (1999) and could explain many of the conflicts still existing in the theories of tinnitus generation.

If autonomic nerves play part in tinnitus generation and tinnitus is generated in the DCN level the way Levine (1999) suggests, it might explain why:

1) Tinnitus might start after various different general diseases - the disease could affect the autonomic nerves triggering reflectory effects in the central nervous system.

2) Tinnitus might start after loud noise or other disorder affecting hearing in one individual but not in another although both get similar kind of hearing defect - it depends on the sensitivity of the autonomic nerves of the individual like in reflex dystrofia.

3) Tinnitus seems to be affected by psychological aspects - they are important also in the developement of reflex dystrofia, which is a disease connected to autonomic nervous dysfunction.

4) Various other disorders are quite frequently connected to tinnitus like hyperacusis and fullness of ear - the autonomic nerves conduct vague sensations and they might explain these type of disorders better than the clearer neural connections of pyramidal tract nerves.

5) Tinnitus can start and remain even if the inner ear or cochlear nerve are destroyed - the inner ear or cochlear nerve are perhaps not needed in tinnitus generation at least in some cases of tinnitus. Tinnitus might be produced in the nucleus cochlearis after neural excitation via autonomic nerves and the triggering point might be situated outside the ear.
6) Tinnitus can sometimes disappear although hearing defect is permanent - if the OHC destruction would be the reason for tinnitus in every case it is hard to explain how the OHC could regenerate in cases when tinnitus disappears but the hearing defect reflecting the degree of OHC degeneration does not. In spite of the hair cells, if the autonomic nervous excitation is reduced, tinnitus influenced by it might be reduced as well.

These theories are summarized in fig.23.

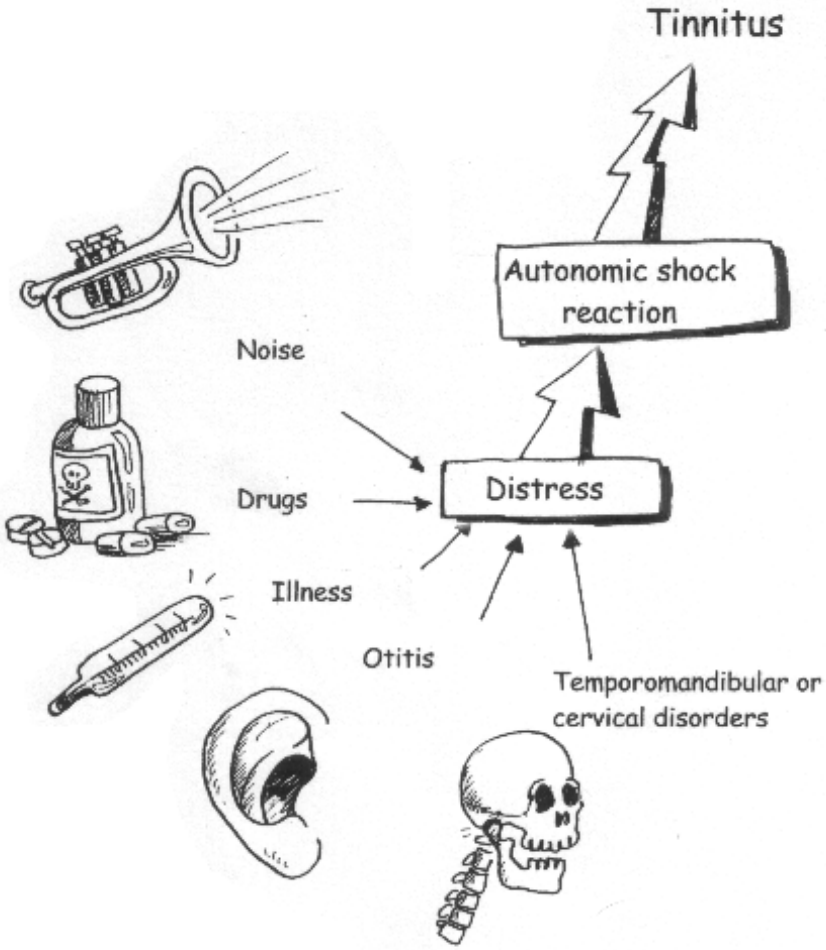


Fig.23 Tinnitus generation

H. SUMMARY AND CONCLUSIONS

1. The tinnitus character in primary care ENT patients (217 people, 178 treated and 39 control patients) was analyzed. This meant 122 cases with right ear tinnitus, 152 cases with left ear tinnitus and 13 cases with tinnitus in the head in the treated group and 26 cases with tinnitus in the right ear, 31 cases with tinnitus in the left ear and 3 cases with tinnitus sensed in the head in the control group. The volume and frequency of tinnitus were measured. The character of tinnitus was mostly ringing. In the treated group 42% of right ear tinnitus, 37% of left ear tinnitus and 27% of tinnitus sensed in the head was described as ringing. In the control group 40% of right ear tinnitus and 35% of left ear tinnitus was ringing, none of the three head tinnitus cases were discribed as ringing. The women had statistically ($p<0.001$) more chirping character in their tinnitus and male more ringing. In some cases several tinnitus sounds were sensed.

2. The loudness measures of tinnitus were low in spite of the distress to the patients. The mean VAS of the loudest tinnitus was 5.5, the second loudest tinnitus 3.8, the third loudest tinnitus 3.4 and the fourth loudest tinnitus 3.4. The measured mean tinnitus volumes were 44 dB for the right ear and 43 dB for the left ear.

3. The maximal cervical muscular tension situated statistically more often ($p<0.0001$) in the same side than tinnitus. This seems to point to a correlation with them.

4. The treatment was accomplished by first manually searching the trigger points in the cervical and upper thoracic area. The most prevalent trigger points especially in the ipsilateral side were injected with 1% lidocain without adrenalin. The treated cases were analyzed in the end of the treatment period and at least 6 months after the last treatment. The results were compared with the untreated control patients with tinnitus

5. The effects of trigger point injections on tinnitus were analyzed and more than one third could benefit from it. The effect of lidocain injections on trigger points overlasted the duration of drug influence and could not be explained with the usage of lidocain alone. The treatment effects were mostly temporary and partial but a few permanent improvements occured. After 6 months the treated patients had statistically better outcome with their tinnitus than the control group (right ear $p<0.001$, left ear $p<0.01$).

6. Chirping type of tinnitus seemed to be connected with better outcome of tinnitus treatment than other types ($p<0.05$ in the left ear, $p<0.001$ in the right ear). The females had better outcome of the treatment and it seemed to be connected to the gender directly ($p<0.0001$).