Osteopathy in idiopathic sudden hearing loss

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by ***Uwe Staffa***

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DECLARATION IN LIEU OF AN OATH

I hereby declare that I have written the submitted Master Thesis on my own.

All passages that have been taken over literally or roughly from other persons' published or unpublished work have been marked as such. All sources and aids I have used for the thesis are mentioned. The thesis of the same content has not yet been presented to another examination authority.

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Date Signature

Abstract:

Objective: The purpose of this work was to find out if there is a better recovery of hearing threshold in patients with sudden hearing loss who were treated osteopathicly after the standard treatment course with steroids and rheologic remedies compared to those who were not.

Design and Methods: Controlled clinical trial of patients with sudden hearing loss with persistent hearing impairment after a one week inpatient therapy series with corticosteroids and rheologic drugs.

Results: In the intervention group there was an over all improvement of hearing threshold of 5dB mean and in the controls of 4,4dB mean improvement. The intervention group showed some improvement between 2dB and 7,5dB in the lower frequencies (125Hz – 1kHz) and in the higher frequencies (3kHz – 8kHz) between 3dB an 14dB. In the controls improvement appeared in the frequencies from 125Hz to 2kHz between 5dB and 8dB. Above 3kHz there was almost no difference.

Conclusion: The question if osteopathic treatment, following drug therapy in care of sudden sensorineural hearing loss, leads to better recovery of hearing thresholds than no additional treatment can not be answered sufficiently after this work. In the end the composition of the two groups was too different to allow a correct comparison.

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1 Introduction

I had the idea to write a master thesis on this topic when I once treated a patient who had sustained a cerebro-cranial trauma. Among other things, he was suffering from a unilateral hearing impairment due to the severe head injury. The treatment mainly focussed on problems in the area of the locomotor system and so I was very surprised when the patient told me one day that the result of audiometry suddenly had improved very clearly within a week. The patient underwent audiometry twice within a week and in the meantime he was treated with osteopathy. As a result, the question arose whether the improvement of hearing and the treatment were connected.
From this point in time on, I started to take an interest in the topic of hearing and hearing disorders. Although the loss of hearing does not constitute a direct physical risk, it may have serious mental and social consequences for the person affected. (Davidson, 2002) The sense of hearing has a central function for the human being for social communication and as warning and orientation system (Probst et al, 2004) In my opinion, the impairment is especially important when the hearing disorder affects the frequency range of human speech (500 Hz – 4000 Hz) since then verbal communication with one's fellow human beings is heavily disturbed. Completely everyday things such as for example a conversation in company become unbearable because of the increased noise level in the vicinity.

It turned out, however, that hearing loss or hearing impairment acquired through trauma is relatively uncommon. Therefore, I looked for a more common form of hearing loss and drew up a plan for implementing this topic in a master thesis. My preliminary research showed that the incidence of sudden hearing loss in this country and the frequency of its treatment in hospital should be enough for performing this study in the way it has been planned. Approximately 200 patients suffering from sudden hearing loss are treated as in-patients in hospitals in my catchment area every year (personal communication by Dr. Summesberger, 2005; Dr. Hollenstein 2006; Dr. Kanonier, 2006) On account of the information indicating that there is a sufficient number of patients who do not respond well or not at all to treatment with drugs, I decided to choose this topic for my master thesis. Contrary to all expectations, it turned out bit by bit that the rate of remission was extremely high and thus only a few patients were eligible for the study.

This work deals with the effects of osteopathic treatment on the hearing threshold determined using tone audiometry in patients with idiopathic sudden hearing loss.

Is there a difference in the recovery of the hearing threshold after idiopathic sudden hearing loss between a group of patients treated with osteopathy and a group of patients not receiving any further treatment?
First of all, the patients of both groups received an in–patient treatment up to one week with corticosteroids and rheologic infusions with pentoxifylline and hydroxyethyl starch (HES). After the in-patient stay, the medication was continued for all patients up to 10d with cortison and Trental orally and was reduced gradually. Patients who, after the in-patient drug treatment, still showed a clearly lowered hearing threshold in the audiogram were eligible for the study. The patients of the experimental group were treated with osteopathy four times within four weeks when there still was a clear hearing impairment after the in-patient treatment. The patients of the control group did not receive any other treatment in addition to the drugs administered orally and reduced gradually. At the end of the osteopathic treatment of the experimental group, a tone audiometry was carried out and the results of the two groups were compared.

Objective of the work
The following work aims at finding out whether osteopathic treatments performed following drug therapy contribute to a greater improvement of the hearing threshold than with drug treatment alone.

This work was only possible thanks to the good cooperation with the team of the Otorhinolaryngologic Department of the hospital LKH Feldkirch which volunteered kindly to help me to recruit patients and to carry out the status and follow-up examinations.

2 Theoretical fundamentals

2.1 Anatomy and physiology

For this chapter, also see Reiss (2003) and Probst (2004).

2.1.1 Anatomy of the ear

The ear is made up of 3 separate sections: 1. external ear, auris externa, 2. middle ear, auris media, 3. inner ear, auris interna.

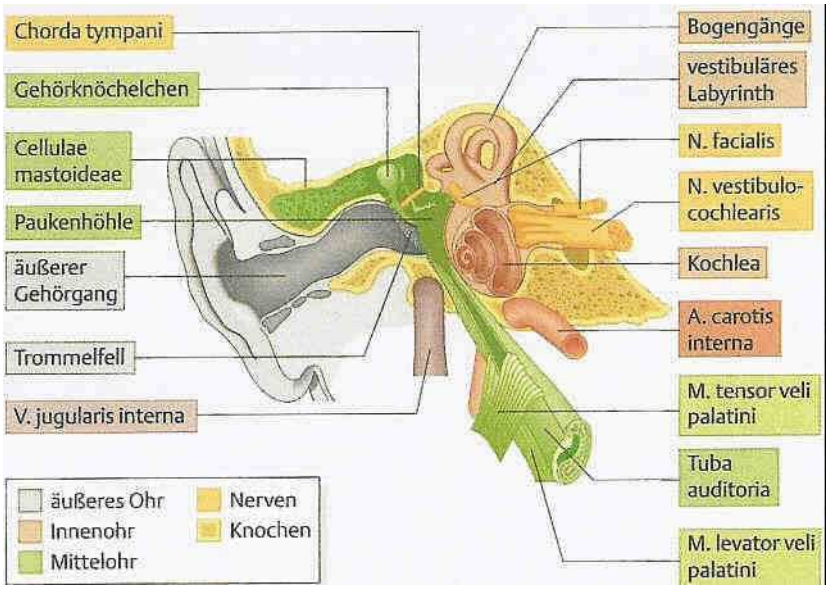


Fig. 1: Peripheral hearing organ, from Hals-Nasen-Ohrenheilkunde (Probst et al, 2004, page 154)

2.1.1.1 External ear

The external ear consists of the pinna of the ear (auricle), the external acoustic meatus (meatus acusticus externus) and the eardrum (membrana tympanica) as medial limitation.

2.1.1.2 Middle ear

The middle ear consists of aeriferous spaces lined with mucous membrane, namely the tympanic cavity (cavitas tympanica), the mastoid cells (cellulae mastoideae) and the Eustachian tube (tuba auditoria). The

tympanic cavity is the main space of the middle ear, it is separated from the external acoustic meatus through the eardrum and contains the auditory ossicles - hammer (malleus), anvil (incus) and stirrup (stapes) – as well as the two inner muscles of the middle ear – tensor tympani muscle and stapedius muscle. These two muscles of the middle ear regulate the state of tension of the eardrum. The hammer is connected with the eardrum on one side and on the other side it is movably connected with the anvil which again has an articulated connection with the stirrup and through the footplate establishes the contact to the oval window of the inner ear. This chain of the auditory ossicles is for transmitting and amplifying the vibration from the eardrum to the perilymph of the inner ear.
The middle ear is connected with the rhinopharynx through the Eustachian tube.

2.1.1.3 Inner ear

The inner ear is a unity from an anatomical and evolutionary point of view. Functionally, a distinction is drawn between the organ of hearing (organum cochleare) and the organ of equilibrium (organum vestibulare).
It is found in the petrosa of the temporal bone and consists of several connected bony ducts called labyrinth. The bony labyrinth (perilymphatic space) is lined by the membranous labyrinth (endolymphatic space) and the perilymphatic space remaining free is filled with liquid rich in sodium (perilymph). The perilymphatic and endolymphatic spaces consist of two vesicular structures each (utriculus and sacculus) in the vestibule (vestibulum) of the three semicircular ducts (ductus semicirculares) and the cochlear duct (ductus cochlearis).
Urticulus, sacculus and semicircular ducts belong to the organ of equilibrium and are connected to the cochlea (organ of hearing) through the Hensen’s canal and are filled with endolymph (liquid rich in potassium). The thin endolymphatic duct (aquaeductus vestibuli) that ends up in the endolymphatic sac on the back area of the petrous bone branches off the utriculosaccular duct, the connecting passage of utriculus and sacculus. It is a contentious issue whether the endolymphatic sac is located between the two layers of the dura mater or between the periosteum of the petrous bone and the dura mater. It is assumed to be involved in the regulation of the endolymph, in immune processes and secretory activities.

The perilymphatic space of the bony labyrinth is connected with the subarachnoid space through the perilymphatic duct (aquaeductus

cochleae). It starts under the round window in the tympanic scala and ends up at the back pyramidal area under the internal acoustic pore. It is probably regularly open only in children and closed through connective tissue in adults.

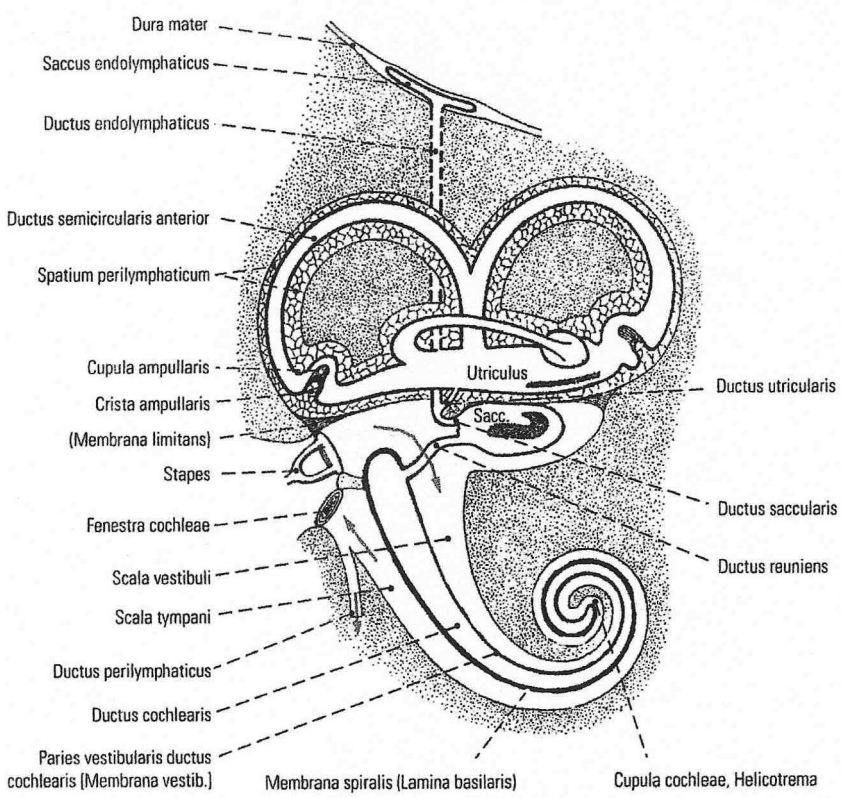


Fig. 2: Scheme of the membranous labyrinth and the perilymphatic spaces in the human being (Reiss, 2003, p. 614)

2.1.1.3.1 Cochlea

The bony canal of the cochlea has a length of approx. 3 –3.5 cm and winds itself in spiral paths round the cone-shaped modiolus. A fine osseous lamella consisting of two parallel layers, the lamina spiralis ossea, runs round the modiolus.
Together with the spiral membrane (lamina basilaris), the spiral ligament and the cochlear duct, the lamina spiralis ossea divides the winding of the cochlea into two stairs, the vestibular scala and the tympanic scala, that are connected with each other at the tip through the helicotrema.

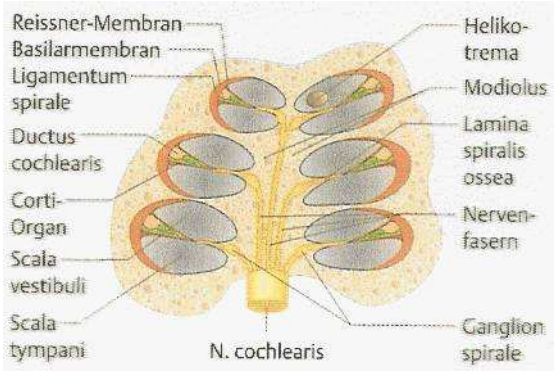


Fig. 3: Axial section through the cochlea (Probst et al, 2004, p. 160)

The lamina basilaris is narrow and thick at the base of the cochlea and becomes broader and thinner towards the tip. This changes its mechanical properties in the course of the cochlea. The basilar membrane is essentially stiffer and less flexible in the area of the base of the cochlea than at the tip of the cochlea. That is how the resonance of the basilar membrane is tuned to high frequencies at the base and to low frequencies at the tip. These are the mechanical properties tonotopy, the projection of certain frequencies to defined areas of the basilar membrane, is based on. This is how every frequency can be assigned to certain nerve fibres.

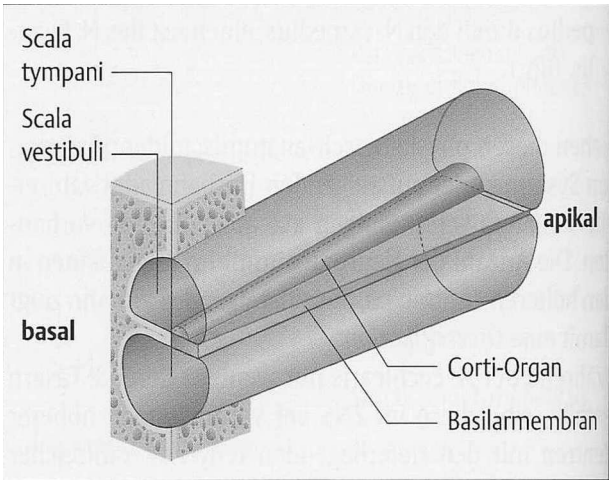


Fig. 4: Model of basilar membrane (Probst et al, 2004, p. 160)

The vestibular scala starts at the base of the cochlea in the area of the oval window and contains perilymph. It is located above the cochlear duct and is separated from it through the thin Reissner's membrane.

The tympanic scala is filled with perilymph as well and is located under the basilar membrane. It runs from the helicotrema downwards to the round window that is closed by a mobile membrane for pressure equalisation.
The cochlear duct of triangular cross-section is limited at the bottom by the basilar membrane that supports the organ of Corti, at the top by the Reissner's membrane and laterally by the stria vascularis.
The stria vascularis is a multilayered epithelium that lines the spiral ligament of the cochlea and through its numerous capillaries ensures the metabolism of the cochlea as well as the production of the endolymph.

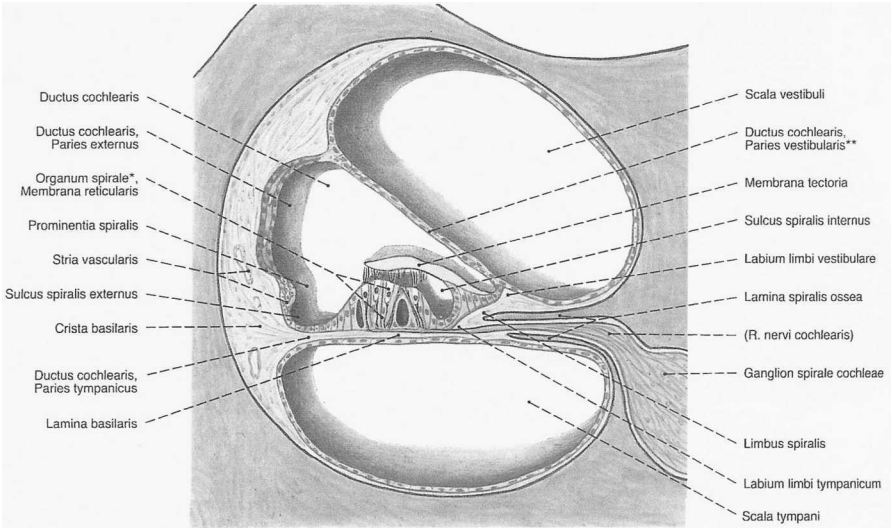


Fig. 5: Cochlea with organ of hearing, schematised sectional view (Sobotta, 2004, p. 396)

The organ of Corti (organum spirale) lies on the basilar membrane and constitutes the sound receiver. It contains supporting and sensory cells (outer and inner hair cells). The tectorial membrane starts from the lamina spiralis ossea and covers the sensory cell area of the organ of Corti. The hair cells are mechanoreceptors carrying a bundle of stereocilia (sensory hairs) of different length on their surface. When the stereocilia are moved/bent through movements of the basilar mambrane and/or through wave motions of the endolymph, an adequate stimulus is created for the sensory cell – the transformation of an acoustic information into a neural signal. The approx. 3000 inner hair cells of the cochlea are located more medially on the basilar membrane and are surrounded by supporting cells. They are arranged in a row. Every inner hair cell has several afferent fibres of the cochlear nerve.
The outer hair cells are located laterally of the inner hair cells. The number of outer hair cells is approximately three to four times higher than that of the inner ones. Normally, they are arranged in three rows. Their

stereocilia of the outer hair cells are firmly connected with the tectorial membrane. The cochlear nerve supplies the outer hair cells mainly with efferent fibres and only a few afferent fibres. They are considered to be the engine of the cochlear amplifier.

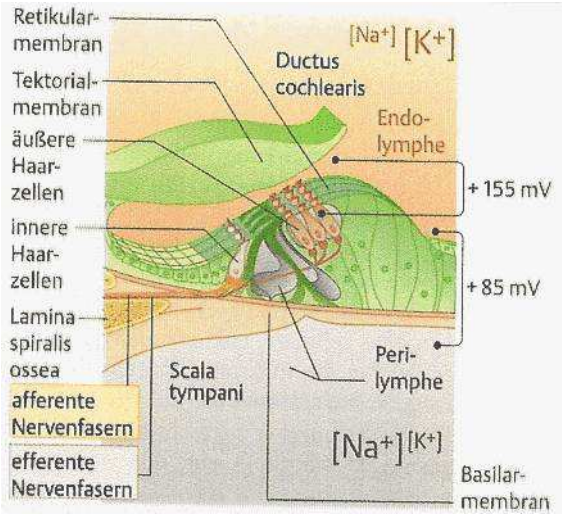


Fig. 6: Corti-Organ (Probst et al, 2004, S. 161)

2.1.1.3.2 Meatus acusticus internus

The internal acoustic meatus (internal ear canal) connects the labyrinth with the posterior cranial fossa. It ends with an oval hole, the internal acoustic pore at the posterior surface of the petrous pyramid. The cerebral membranes reach inside the internal ear canal and the subarachnoidal space extends up to here and communicates with the perilymphatic spaces of the inner ear. The facial nerve, the intermedius nerve and the vestibulocochlear nerve as well as the labyrinthine artery with sympathetic plexus and the labyrinthine veins run through the inner ear canal.

2.1.1.3.3 Blood supply of the inner ear

The inner ear is supplied with blood by the labyrinthine artery. The labyrinthine artery originates from the basilar artery or the anterior inferior cerebellar artery. It has fine branches towards the dura and runs to the inner ear through the internal acoustic meatus, where it divides into the vestibular artery and the cochlear artery.
The blood flows off the labyrinth through several labyrinthine veins to the inferior petrosal sinus and the superior bulb of the jugular vein.

2.1.1.3.4 Nerve supply of the ear

The muscles of the external ear are innervated by the facial nerve. The sensitive innervation of the external ear is accomplished by several nerves. The posterior surface of the pinna of the ear is supplied by the great auricular nerve and the minor occipital nerve. The auriculotemporal nerve of the mandibular nerve of the trigeminal nerve accomplishes the sensitive innervation of the anterior outer surface of the pinna of the ear, the anterior upper part of the external ear canal and the anterior upper part of the external surface of the eardrum. The auricular branches of the nerves VII, IX and X supply the bottom and the posterior wall of the external ear canal and the external surface of the eardrum.

Several nerves run in the middle ear, but they supply the tympanic cavity to a low extent only. The motor innervation of the stapedius muscle is accomplished by the stapedius branch of the facial nerve and the tensor tympani muscle is supplied by the medial pterygoid nerve of the 3rd branch of the trigeminus nerve.
The mucosal membrane of the middle ear is supplied by the tympanic plexus. It is formed by the tympanic nerve of the glossopharyngeal nerve, sympathetic fibres of the plexus of the internal carotid nerve (caroticotympanic nerves) and by a communicating branch of the facial nerve. The cord of tympanum (intermedius nerve) runs through the tympanic cavity.
The vestibulocochlear nerve is a purely sensory nerve that mainly contains afferent fibres leading to the vestibular and cochlear nuclei in the brain stem. It is functionally made up of the vestibular nerve and the cochlear nerve.

The different nerve fibres from the elements of the vestibular end-organ (utriculo-ampullary nerve, saccular nerve, posterior ampullary nerve)

unite in the vestibular ganglion on the bottom of the internal ear canal to form the vestibular nerve.

The afferent fibres of the cochlear nerve originate in the bipolar nerve cells of the spiral ganglion in the cochlea. The peripheral processes end at the hair cells of the organ of Corti and the central processes unite to form the cochlear nerve.
The efferent fibres of the auditory pathway originate in the superior olivary nucleus and reach the organ of Corti through the olivocochlear tract and the cochlear nerve.

In the internal ear canal, the two parts unite to form the vestibulocochlear nerve of macroanatomic unity. It runs through it together with the facial nerve, the intermedius nerve, the labyrinthine artery and the labyrinthine veins into the cranial cavity. It enters the brain stem in the cerebellopontine angle together with the facial nerve and the intermedius nerve. The fibres of the cochlear nerve end in the cochlear nuclei, the fibres of the vestibular nerve end in the vestibular nuclei.

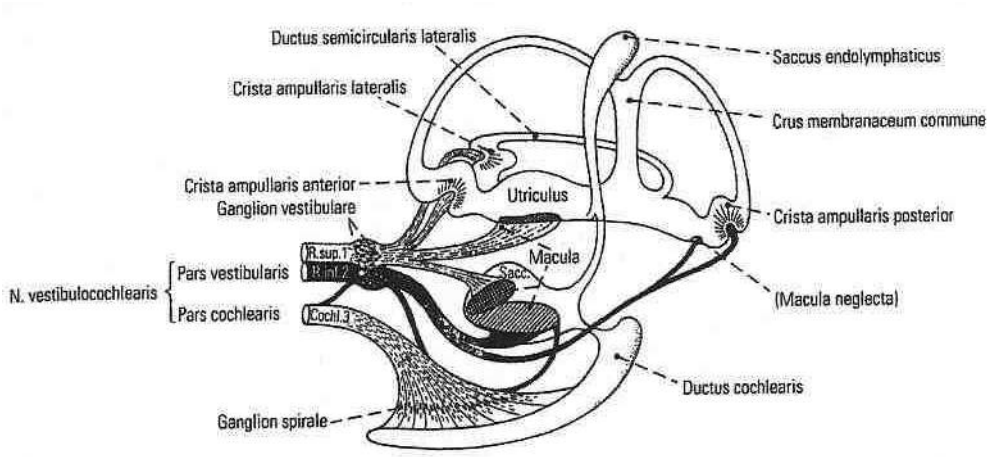


Fig. 7: Branching of the vestibulocochlear nerve (Reiss, 2003, p. 617)

2.1.1.3.5 Central auditory system

The central auditory system and with it the processing of acoustic information starts in the brain stem in the cochlear nucleus representing the end of the cochlear nerve. The cochlear nerve receives its afferences exclusively from the same side, then the large part of the fibres runs to the opposite side and reaches the areas of the auditory cortex mainly located in the temporal lobe through the two lower olivary complexes, the inferior colliculus of the midbrain and the thalamus.

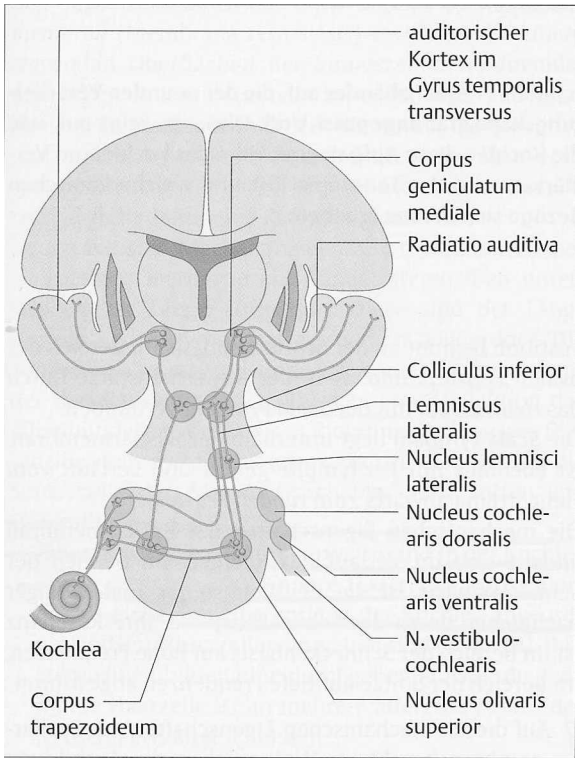


Fig. 8: Central auditory pathway (Probst et al, 2004, p. 159)

2.1.2 Physiology of hearing

The human ear is able to perceive air vibrations of frequencies from 16 Hz to 20 000 Hz as sound. Very high and very low frequencies within this range of the hearing spectrum cannot be heard very well. The frequencies in the middle of the range from 1000 Hz to 4000 Hz are perceived best. (Mrowinski and Scholz, 2002) Sound waves reaching the ear are reflected by the elevations and depressions of the pinna of the ear and conducted through the external ear canal to the eardrum which is set vibrating through the sound pressure. The vibrations of the eardrum are then transmitted to the oval window (limit of the inner ear) by the auditory ossicles. The size ratio between the eardrum and the footplate of the stirrup, that closes the oval window, is 17:1. This ratio results in sufficient pressure amplification in order to transform the vibrations of the eardrum into an effective liquid motion of the perilymph. The shock waves in the perilymph run through the vestibular scala in the cochlea upwards to the helicotrema and downwards through the tympanic scala until the round window that equalises the pressure. The travelling wave triggered by this causes movements of the basilar membrane against the tectorial membrane through which the sensory hairs of the outer hair cells of the organ of Corti are stimulated. The activation of the outer hair cells results in an activation of the inner hair cells.
The inner hair cells are responsible for transduction, that is the transformation of acoustic vibrations into nerve potentials, that are then transmitted by the cochlear nerve and processed centrally.

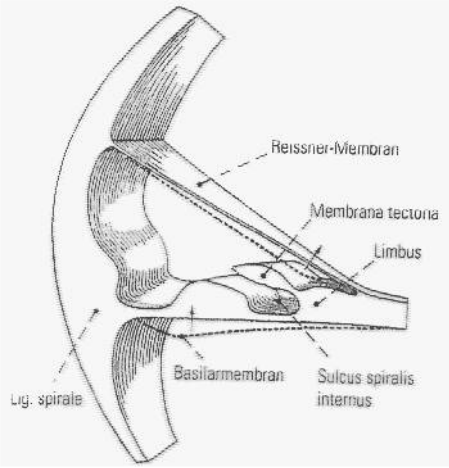


Fig. 9: Travelling wave, deflection of the basilar membrane (Reiss, 2003, p. 623)

2.2 Idiopathic sudden hearing loss

2.2.1 Definition

> *"Der Hörsturz ist eine ohne erkennbare Ursache plötzlich auftretende, in der Regel einseitige Schallempfindungsschwerhörigkeit cochleärer Genese von unterschiedlichem Schweregrad bis hin zur Ertaubung. Schwindel und/oder Ohrgeräusche sind zusätzlich möglich."* (http://www.leitlinien.org, 2004)
> [Sudden hearing loss is a hearing difficulty due to impaired sound perception that occurs suddenly without identifiable cause and normally on one side and is of cochlear genesis, of different degree of severity to deafness. Vertigo and/or tinnitus are possible as well.]

In literature, sudden sensorineural loss of hearing is commonly defined as loss of hearing developing within a period of less than 72 hours and marked by a hearing impairment by 30 dB and more in three adjacent frequency ranges. (Ahmed et al, 2004; Chang et al, 2005; Halpin and Rauch, 2003; Miller, 2003; Wazen and Ghossaini, 2003) Loss of hearing can set in suddenly – virtually at a moment's notice – or develop within several hours to a few days. (Wazen and Ghossaini, 2003)

2.2.2 Aetiology / pathogenesis

The precise developmental mechanism of idiopathic sudden hearing loss has not been clarified exactly up to now.
There are various theories on the development of sudden hearing loss which, however, have not been confirmed yet. In addition to circulatory disorders (of vascular or rheologic origin) and infectious diseases (viruses), autoimmune processes, mental factors and disorders of the cervical spine are assumed possible aetiologies. (Krisch, 2005; Yasar, 2001)

A clear cause can be shown in not more than 10 – 15% of the cases in spite of comprehensive examinations (Fritsch et al, 2003; Slattery et al, 2005; Zadeh et al, 2003).
Fayad and De La Cruz (2003) describe SSNHL (sudden sensorineural hearing loss) even as a syndrome and not as an own diagnosis. The aetiology of sudden hearing loss remains unclear in most of the patients and is considered to be idiopathic.

Wazen and Ghossaini (2003) state in their article "The Diagnostic and Treatment Dilemma of Sudden Sensorineural Hearing Loss" that it is usually the greatest challenge to find out the causes of the hearing loss and aetiology often remains doubtful despite comprehensive examinations.

"Ätiologie und Pathogenese des Hörsturzes sind weitgehend unbekannt. Folgende Pathomechanismen werden diskutiert (Tab. 1):

- Störungen der Durchblutung (Gefäßdysregulationen, z.B. durch Vasospasmus und /oder Endothelschwellungen und Dysfunktionen und/oder rheologische Störungen)
- Störungen der Ionenkanäle der Haarzellen mit zellulärer Dysfunktion
- Synaptische Störungen infolge Neurotransmitter-Dysfunktion (Insuffizienz oder Toxizität)
- Efferente Fehlsteuerungen
- Störungen der Ionenkanäle der Zellen der Stria vascularis mit nachfolgenden Elektrolytstörungen in der Endolymphe, u.U. mit Hydrops
- Entzündliche Veränderungen (z.B. endolymphatische Saccitis)
- Unbekannte Pathobiochemie und Pathophysiologie" (http://www.leitlinien.org, 2004)

[Aetiology and pathogenesis of sudden hearing loss are unknown to a large extent. The following pathomechanisms are discussed (Tab. 1):

- disorders of blood circulation (vascular dysregulations, e.g. due to vasospasm and / or endothelial swelling and / or dysfunctions and / or rheologic disorders)
- disorders of the ion channels of the hair cells with cellular dysfunction
- synaptic disorders as a result of neurotransmitter dysfunction (insufficiency or toxicity)
- efferent malfunctions
- disorders of the ion channels of the cells of the stria vascularis with subsequent electrolyte disturbances in the endolymph, perhaps with hydrops
- inflammatory changes (e.g. endolymphatic saccitis)
- unknown pathobiochemistry and pathophysiology]

(http://www.leitlinien.org, 2004)

Various opinions on some common theories on aetiology of sudden hearing loss are presented in the following.

2.2.2.1 Circulatory and vasomotor disturbances:

The stria vascularis and the cells of the organ of Corti always react in the same way to the different noxae. The hurtful influence leads to cellular decay, as a result of which an oedema is developed that leads to an occlusion of the functional terminal vessels and a blockage of the microcirculation in the cochlea. The resulting lack of oxygen in the cochlea results in the standstill of the functional metabolism in the end causing the hearing loss – irrespectively of the type of noxa. (Beck, 1984)

Halpin and Rauch (2003) state that several authors suggest vascular insufficiency as cause of SSNHL. This assumption is primarily based on the sudden development of the symptoms – as if the blood supply to the cochlea was interrupted.
It has to be countered that the systemic treatment with primarily vasodilatory substances turned out to be not effective for acute disturbances of the inner ear and even is contraindicated on account of vascular "steal syndromes". In this case, the general vascular dilatation locally in the affected area may result in a further deterioration of the arterial blood circulation. (Plontke, 2005)

Haematological diseases such as leukaemia, sickle-cell anaemia, macroglobulinaemia and polycythaemia are mentioned as possible causes of SSNHL. Regarding these diseases, vasospasm, thrombosis, embolism and acute bleeding into the inner ear are assumed to be pathogenetic, pathologic evidence of this perceptio, however, has not been provided yet. (Wazen und Ghossaini, 2003)

According to Fayad and De La Cruz (2003), there are several arguments against a circulatory aetiology. These include the high rate of spontaneous regression, the significant incidence in rather young patients, the absence of an apparently increased incidence in diabetics and the fact that most of the patients do not suffer from vertigo.

Possible pathomechanisms:

- haemorrhage
- thrombosis, embolism
- microcirculatory disorders
- vasospasm
- endothelial swelling
- rheologic disturbances (such as: increased thrombocyte adhesiveness and aggregation,...)
- vertebrobasilar ischaemia (VBI)

According to http://www.hoersturz.info (2005), occlusions of the blood vessels through small blood clots are supposed to be the most common cause of circulatory disorders of the inner ear. Thus, factors that increase the coagulability of the blood and result in a thickening of the blood contribute to the occurrence of sudden hearing loss.

Sudden deafness can be the initial presentation of VBI (vertebrobasilar ischemia). Hearing loss due to VBI often has a good outcome. (Lee and Baloh, 2004)
The vertebrobasilar system supplies the largest part of the auditory system including the inner ear. Therefore, vertebrobasilar insufficiency is able to cause serious hearing disorders. (Lee and Baloh, 2004)

In their study, Lee and Baloh (2004) found an incidence of sudden hearing loss of 8% (n=29) in 364 VBI patients within a period of 3 years and 9 months. In nine of these patients, an isolated audiovestibular disorder was the initial symptom of VBI. The brain MRI showed an infarction in the area of the anterior inferior cerebellar artery in more than three quarters of the patients suffering from sudden hearing loss with vertebrobasilar ischaemia.

2.2.2.2 Infectious diseases

Infectious diseases of the upper airways are particularly frequently stated in literature as possible causes of sudden hearing loss. They may have occurred shortly before or simultaneously with the sudden hearing loss. (compare: Fayad and De La Cruz, 2003; Fritsch et al, 2003; Halpin and Rauch, 2003; Wazen and Ghossaini, 2003)
The list of infectious causes is long and might include each pathogen. A causal connection between sudden hearing loss and pathogens such as

syphilis, mumps, measles, herpes zoster and adenovirus is established more often than for other pathogens since they have a known pathologic effect on the inner ear. (Wazen and Ghossaini, 2003)

Severe sensorineural hearing loss may occur as complication together with apparently viral infections. 28% of the patients suffering from sudden hearing loss report a virus-like infection of the upper airways within a period of up to one month before the onset of the impairment of hearing. (Fayad und De La Cruz, 2003)

> *"For viruses like mumps, measels, and rubella, there is evidence that they may be the cause of idiopathic sudden sensorineural hearing loss (ISSNHL). For other viruses, there clearly is an association with idiopathic sudden sensorineural hearing loss, although a convincing evidence of a causal relationship is lacking."* (Fayad and De La Cruz, 2003, p. 2)

Several authors showed an association of SSNHL with an active viral disease of the upper airways and determined antibodies to certain viruses as well. (Halpin and Rauch, 2003)

In an animal experiment, Stokroos et al (1998) induced a herpes simplex virus 1 labyrinthitis that caused hearing loss and a structural damage of the cochlea, but without showing the symptoms of herpetic infection and without the occurrence of HSV specific antibodies.

A virus infection of the cochlea is not absolutely necessary for a sudden hearing loss to occur. A disease of the upper airways is enough to trigger reactions in the inner ear. (Halpin and Rauch, 2003)

Adams (2002) detected biomolecules, so-called inflammatory cytokines, that are produced in the cochlea (in the spiral ligament) as reaction to a systemic inflammatory process without symptoms. This may cause a disorder of the function of the potassium ion recycling in the spiral ligament and result in a sensorineural hearing loss.

Histopathologic examinations of the inner ear showed that the changes in the cochlea occurring in case of sudden hearing loss are the same as in case of virus infections: atrophy of the organ of Corti, the tectorial membrane and the stria vascularis. (Schuknecht et al, 1962)

Wazen and Ghossaini (2003) state that meningitis is a known cause of sudden sensorineural hearing loss with a possible spread of the pathogen into the inner ear and/or the internal ear canal.
Sensorineural hearing loss may also occur in case of syphilis or HIV infections in rare cases.

2.2.2.3 Autoimmune processes

Sensorineural hearing loss may be caused by autoimmune diseases (autoimmune inner ear disorders – AIED) as well. Well known autoimmune diseases such as for example Cogan's syndrome, systemic lupus erythematodes and polyarteritis nodosa are associated with sensorineural hearing loss.
Regarding autoimmune processes, however, it has to be mentioned that the clinical picture slightly differs from the one of sudden hearing loss. In these cases, for example, the impairment in hearing often occurs bilaterally. It develops quickly and progressively with a tendency to a further deterioration of the impairment of hearing when it remains untreated, but the suddenness of the sudden hearing loss is missing. (Fayad and De La Cruz, 2003)
The theory of autoimmunologic causes of sudden hearing loss is based on positive serologic findings and the presence of other immunologic signs and symptoms. (Wazen and Ghossaini, 2003)

2.2.2.4 Trauma

Head injuries are possible causes of sudden sensorineural hearing loss. In this case, the impairment in hearing can be the result of the loss of hair cells, rupture of the cochlear membrane, fracture of the petrous bone with involvement of the labyrinth or a perilymph fistula. (Fayad and De La Cruz, 2003)

2.2.2.5 Functional disturbance of the cervical spine

It is generally accepted today that functional disturbances in the area of the upper cervical spine may be the causes of cervico-encephalic symptomatic complexes such as cephalgias, but also cervical vertigo. The existence of a "cervicogenic hearing disorder" is a controversial subject. Several authors report hearing losses by up to 20 dB that can be influenced well through manual therapy. (Hülse, 1994)

Functional disorders of the upper cervical spine, such as e.g. blockage of the segment C1/C2, were observed concomitantly in patients suffering from sudden hearing loss. (Reißhauer et al, 2006)
The study results of Reißhauer et al (2006) suggest that functional disorders of the cervical spine are common findings in case of tinnitus. This study showed also that the reduction of the global mobility of the cervical spine has a significantly greater influence on the tinnitus than segmental functional disorders.

Hülse (1994) describes cervicogenic hearing disorder as follows:

- lowering of the hearing threshold, in particular in the low-frequency range by up to 25–30 dB
- commonly (67%) unilateral cochlear symptoms, often recurrent
- subjective impairment of hearing, often feeling of ear pressure
- TEOAE clearly reduced (at 90%) – in most cases under 50%
- functional deficiency of C0/1 and /or C2/3 on the side of impaired hearing
- cervicogenic impairment of hearing is reversible, can be influenced by manual therapy

The following pathomechanism is assumed mainly on account of the positive influence of manual therapy:
Due to the functional impairment of the head joints, there are changes of activity in the area of the proprioceptive and nociceptive afferences of the upper cervical spine – mainly of C2. Such afferentiation disorders reach the areas of cranial nerve nuclei through indirect and direct neural connections. (Hülse, 1994) Pfaller and Arvidsson (1988) showed direct afferent connections from the dorsal root of C2 to the ventral cochlear nucleus in animal experiments with rats.

Hack et al (1995) demonstrated a relation that is very interesting from the osteopathic point of view. They confirmed the direct connection made of connective tissue between the rectus capitis posterior minor muscle and the posterior atlantooccipital membrane lying underneath and between this membrane and the spinal dura mater in eleven dissections.

There are cases, in which a connection between the cervical spine and the efferent part of the hearing system has been shown in a clinically clear manner, that is cases of tinnitus occurring due to a reflex after manipulations of the cervical spine. (Reißhauer et al, 2006)

According to Hülse (1994), experience acquired up to now suggests that labyrinthine hearing loss by more than 35 dB cannot exclusively be attributed to a functional disorder of the head joints. Nevertheless, elevations of the threshold by up to 20 dB could be reached using manual therapy in case of rather distinct hearing disorders and clear functional deficits of the head joints, which was found very positive.

2.2.2.6 Mental stress

In their study, Schüßler et al (1992) came to the conclusion that psychosocial factors play a large role in the genesis of sudden hearing loss. According to that study, medium difficult to difficult circumstances of life (e. g. injury, illness, separation, death of relatives, personal conflicts) prior to the illness were found in 82 % of the patients. Sudden hearing loss most commonly occurs with simultaneous physical and mental stress.
A connection between emotional stress and conflict situations and the occurrence of sudden hearing loss has been observed. (http://www.hoersturz.info, 2005)

2.2.2.7 Surgeries

There are several reports in literature on sudden hearing loss after surgical interventions. Rifai et al (2005) report in their study on a high incidence of severe sudden hearing loss in patients after hepatic transplantation. In fourteen patients out of sixteen in the study, sudden hearing loss was bilateral and four patients out of sixteen reported a sudden hearing loss. A neurotoxic mechanism depending on the dosage of the immunosuppressive medication (e.g. cyclosporine or tacrolimus) is assumed to be responsible for the sudden hearing loss.

Sudden hearing loss with or as a result of cardiac surgical interventions or other non-otologic surgeries under general anaesthesia is a rare complication. (Walsted et al, 2000)
Evan et al (1997) studied 18 cases of sudden sensorineural hearing loss after non-otologic surgeries (and not including cardiopulmonary bypass surgeries) under general anaesthesia and came to the conclusion that there are probably several aetiologies of sudden hearing loss after general anaesthesia.

SSNHL after bypass surgery is assumed to be the result of microembolism, calcium, fat and deposits. (Walsted et al, 2000)

Mak und Tumber (2003) report on a case of postoperative sudden sensorineural hearing loss after a posterior lumbar decompression. In this case, the patient sustained a rupture of the dura during a surgery of the vertebral column under anaesthesia. A severe and persisting unilateral sudden hearing loss occurred as complication.
Perilymph fistula after rupture of the cochlear membrane as a consequence of the high inner ear pressure with intraoperative Valsalva's manoeuvre, lesions caused by nerve compression due to the displacement of the brain as a consequence of changes in pressure in abdominal position, persistent loss of cerebrospinal fluid and the amount of blood loss are discussed as causes of the sudden hearing loss in this case. (Mak and Tumber, 2003)
In her study, Walsted (2000) found a connection between the loss of cerebrospinal fluid during surgery and impairment in hearing. The hearing losses found in this study were considered to be a direct consequence of the loss of cerebrospinal fluid since there is a direct communication between cerebrospinal fluid and perilymph through the cochlear aquaeduct. Consequently, a decrease in the volume of cerebrospinal fluid results in a reduction of the volume of perilymph in the osseous cochlea, which may cause changes in position of the round window and/or spreading of the endolymph in the cochlear duct. (Walsted, 2000)
In his article, Wang (1986) describes a case of bilateral loss of hearing after spinal anaesthesia. The simultaneous occurrence of the post-spinal headache syndrome and the bilateral hearing loss suggests the generally accepted explanation that the loss of cerebrospinal fluid may result in an affection of the vestibulocochlear nerve.

Further possible causes of sudden sensorineural hearing loss are listed under the item Differential diagnosis. They are not mentioned here separately since they are established diagnoses and therefore are irrelevant to the idiopathic sudden hearing loss.

2.2.3 Symptoms

The manifestation of the sudden hearing loss is characterised by acute subjective hearing impairment to total loss of hearing.
The impairment in hearing sets in suddenly, approximately 50 % of the patients sustaining the sudden hearing loss in the morning. Some of these patients, however, have already noticed a feeling of pressure in the ear on the eve of the incident. (http://www.leitlinien.org, 2004; Yasar, 2001)
In the vast majority of cases, the impairment in hearing occurs unilaterally. A bilateral loss of hearing is developed by 2% - 4% only. (Fritsch et al, 2003; Wazen and Ghossaini, 2003)
Tinnitus very commonly occurs together with the sudden hearing loss. The figures on the incidence of tinnitus in combination with sudden hearing loss vary from 70% (Wazen und Ghossanini 2003) and 80% (http://www.hoersturz.info, 2005) to 70-90% (Yasar 2001).
Vertigo is another possible concomitant symptom. The information in literature on the incidence of vertigo vary from 30% (http://www.hoersturz.info, 2005; Yasar, 2001) to 40% (Fayad and De La Cruz, 2003) and 50% (http://www.hear.it.org, 2005; Wazen and Ghossaini, 2003).

There may also be other symptoms on the incidence of which, however, there are no precise data available.

These include:

- sensation of pressure in the ear (fullness of the ear)
- vestibular disturbances (nystagmus, disturbance of equilibrium)
- numbness sensation around the pinna of the ear (periauricular dysaesthesia)
- hyper-/diplacusis

(http://www.leitlinien.org, 2004)

Depending on the area affected in the cochlea, the impairment in hearing can occur in different ranges of frequency. A distinction is drawn between high-, mid-, low-frequency and pancochlear hearing loss. Deafness and impairment of hearing coming close to deafness are possible as well.

2.2.4 Diagnosis

When a cause is found and a diagnosis can be made, the disease is referred to as symptomatic sudden hearing loss.

In the far greater number of cases, however, no cause can be found despite comprehensive examinations. Then the pseudo-diagnosis of idiopathic sudden hearing loss is made. (Probst et al, 2004, p. 267)

The diagnosis is established by ruling out other diagnoses, therefore the level of examination can be considerable in the individual case.

The following examinations are performed on patients suffering from a sudden hearing loss as a matter of routine at the LKH Feldkirch:

- exact anamnesis: previous hearing problems
- ENT findings
- otoscopy
- testing of hearing (tuning fork, tone audiometry)
- testing of the vestibular nerve (testing of equilibrium, nystagmus)
- laboratory: blood count, infectious serology – neurotropic pathogens (HIV, varicella-zoster virus, herpes virus, borrelia,...)

Further examinations are necessary in individual cases:

- CT
- MRT
- otoacoustic emissions (OAE)
- brain-stem audiometry (BERA)
- interdisciplinary examinations (e.g. orthopaedics, neurology, internal medicine)

(personal communication Dr. Summesberger of 11 nov. 2005)

2.2.5 Differential diagnosis

A clear dividing line has to be drawn between the idiopathic sudden hearing loss and other clinical pictures that can also cause a sudden impairment in hearing and the aetiology of which is known. (Krisch, 2005; Yasar, 2001)

"Folgende Ursachen für eine akute Innenohrschwerhörigkeit werden diskutiert:

- Virale Infektionen (z. B. Adenoviren, Zoster, Mumps, HIV)
- Encephalitis disseminata (multiple Sklerose)
- Autoimmunvasculitis (z.B. Cogan-Syndrom)
- Toxische Einflüsse (z.B. Arzneimittel, Drogen, Gewerbegifte)
- Dialysepflichtige Niereninsuffizienz
- Tumoren (z.B. Akustikusneurinom, Hirnstamm- und Felsenbeingeschwülste)
- Perilymphfistel (innere und äußere)
- Barotrauma
- Akutes Schalltrauma, akustischer Unfall
- Funktionsstörungen der Halswirbelsäule (z.B. Trauma, Fehlstellung)
- Bakterielle Labyrinthitis (z.B. bei Mittelohrentzündung, Lues, Borreliose)
- Liquorverlust-Syndrom (z.B. nach Liquorpunktion)
- Meningitis
- Hereditäre (genetische) IOS, Innenohrmissbildung
- Genetisch bedingte Syndrome (z.B. Usher-, Pendred-Syndrom)
- Hämatologische Erkrankungen (z.B. Polyglobulie, Leukämie, Exsikkose, Sichelzellenanämie)
- Herz-Kreislauferkrankungen (z.B. [relative] Hypotonie, Operation am offenen Herzen)
- Psychogene Hörstörungen
- Andere Ursachen"

(vergleiche: http://www.leitlinien.org, 2004)

[The following causes of sudden labyrinthine deafness are discussed:

- viral infections (e. g. adenoviruses, zoster, mumps, HIV)
- disseminated encephalitis (multiple sclerosis)
- autoimmune vasculitis (e.g. Cogan syndrome)
- toxic influences (e.g. drugs, addictive drugs, industrial poison)
- renal insufficiency requiring dialysis
- tumours (e.g. acoustic nerve neurinoma, tumours of the brain stem and the petrous bone)

- perilymph fistula (internal and external)
- barotrauma
- acute acoustic trauma, acoustic injury
- functional disturbances of the cervical spine (e.g. trauma, abnormal position)
- bacterial labyrinthitis (e.g. in case of middle ear inflammation, lues, borreliosis)
- CSF loss syndrome (e.g. after cerebrospinal fluid puncture)
- meningitis
- hereditary (genetic) labyrinthine deafness, malformation of the inner ear
- genetic syndrome (e.g. Usher's, Pendred's syndrome)
- haematologic diseases (e.g. polyglobulism, leukaemia, exsiccosis, sickle-cell anaemia)
- cardiovascular diseases (e.g. [relative] hypotension, open-heart surgery)
- psychogenic hearing disorders
- other causes]

(compare: http://www.leitlinien.org, 2004)

2.2.6 Classification

2.2.6.1 High-frequency hearing loss

The hair cells on the base of the cochlea are affected in case of high-frequency hearing loss (basocochlear hearing impairment). The perception of high frequencies is disturbed. It is the most common form of labyrinthine deafness and occurs in particular as age-related hearing loss, noise-induced hearing loss, in case of sudden hearing loss and as a consequence of the use of ototoxic drugs. (Mrowinski and Scholz 2002, S.25)

2.2.6.2 Low-frequency hearing loss

Low frequencies are perceived in the area of the apex of the cochlea (apicocochlear). The loss or impairment of the ability of hearing low frequencies mainly occurs with Menière's disease. (Mrowinski and Scholz 2002, S.25)
A drift of the hearing threshold in the low-frequency range by up to 30 dB, however, also may occur in the context of cervicogenic hearing disorders. (Hülse, 1994)
Low-frequency hearing loss is assumed to be the result of an endolymphatic hydrops. A local circulatory disturbance of the lamina spiralis with hypoxic tissue damage and disturbance of the electrolyte homoeostasis is possible as well. (http://www.leitlinien.org, 2004)

2.2.6.3 Mid-frequency hearing loss

In case of mid-frequency hearing loss, the audiogram shows a tub-shaped lowering of the hearing threshold in the medium range of frequency. This type of hearing impairment is relatively rare and pathogenetic factors have hardly been studied yet. Possible causes such as for example local circulatory disorders in the area of the lamina spiralis ossea with hypoxic damage to the organ of Corti or genetic defects are discussed. (http://www.leitlinien.org, 2004)

2.2.6.4 Pancochlear hearing loss

Pancochlear hearing loss affects all frequencies, therefore minor hearing impairments are already found serious by the persons affected. Functional disorders of the vascular stria and/or of the afferent vessels in terms of a circulatory disorder and tissue hypoxia are possible pathogenetic factors. (http://www.leitlinien.org, 2004)

2.2.6.5 Deafness / impairment of hearing coming close to deafness

Deafness and impairment of hearing coming close to deafness are characterised by the large extent of the hearing loss throughout the entire frequency range. Embolic or thrombotic occlusion of the common cochlear artery or the spiral modiolus artery with hypoxic strial insufficiency are possible causes. (http://www.leitlinien.org, 2004)

2.2.6.6 Other types of hearing loss

Other hearing losses are courses of the sound threshold that cannot be classified, including highly fluctuating courses of the hearing threshold and sudden hearing loss with progression of the hearing impairment under therapy. (http://www.leitlinien.org, 2004)

2.2.7 Therapy

The AWMF online Leitlinien [online guidelines of the Association of the German Scientific Medical Societies] (2004) indicate that not every sudden hearing loss has to be treated immediately and that, provided that the patient is informed and only has a slight hearing loss, it is possible to wait for some days and see whether a spontaneous regression takes place. By contrast, in case of pronounced hearing loss, previous damage to the ears as well as additional vestibular complaints and/or tinnitus, it is not indicated to wait and see.

> *"Das grundsätzliche Problem der Hörsturztherapie liegt darin, dass wegen im Einzelfall unklarer Ätiologie gesicherte Behandlungsmethoden mit reproduzierbaren Ergebnissen nicht existieren und deshalb die heute angewandten Therapieformen lediglich empirisch abgesichert sind."* (http://www.leitlinien.org, 2004)
> [The fundamental problem of the therapy of sudden hearing loss is that, because of unclear aetiology in the individual case, there are no established treatment methods with reproducible results and therefore the forms of therapy applied today only are supported by empirical evidence.]

The current general therapy approaches for sudden hearing loss are treatment with glucocorticoids, rheologic therapy, ionotropic therapy, reduction of the endolymph volume, administration of antioxidant agents, lowering of fibrinogen through apheresis, hyperbaric oxygenation and inhibition of thrombocyte aggregation and are based on pathopysiologic considerations and disease models as well as on results of clinical examinations. In the end, the decision on a certain form of treatment of sudden hearing loss is made on an individual basis according to the pathophysiology suspected, the best available external evidence, considering risks and adverse effects and the patient's personal desires for therapy. (Plontke, 2005)

2.2.7.1 General principles of the therapy of sudden hearing loss

- rheologic therapy (haemodilution, volume effect, improvement of fluidity, lowering of plasma viscosity, etc.)
- antioedematous therapy (glucocorticoids, etc.)
- ionotropic therapy (influencing of ion channels, etc.)
- reduction of the endolymph volume (osmotherapy, bolus of glycerol, etc.)
- antioxidant agents
- inhibition of thrombocyte aggregation
- lowering of fibrinogen through apheresis
- hyperbaric oxygenation (HBO therapy)

(compare: http://www.leitlinien.org, 2004)

2.2.7.2 Rheologic therapy

Rheologic therapy aims at improving the flowing properties of the blood and so at an improved microcirculation of the inner ear. An intact microcirculation is absolutely necessary for an ideal supply of the inner ear with oxygen and energy carriers as well as the discharge of metabolic end products. (Plontke, 2005)

The most important aim of the treatment of sudden hearing loss in terms of pathophysiology is to improve the circulation and the metabolism of the inner ear and so to normalise the oxygen supply of the sensory cells in the cochlea. (http://www.oxymed-hbo.de, 2006)

Various drugs are used in different combinations for rheologic therapy:

- Dextrans having a pronounced effect on the blood volume. Low-molecular dextran (dextran 40) improves the microcirculation and reduces erythrocyte and thrombocyte aggregation. Anaphylactic reactions may occur as adverse effects after the application of dextran. These adverse effects, however, can be avoided through pretreatment with Dextran1.

- Solutions of hydroxyethyl starch (HES) nearly have the same properties as dextran solutions. The anticoagulant effect of HES, however, is essentially weaker. The disadvantage of HES is the itching that is relatively common, in particular with high doses, persists for a long time and is resistant to therapy.

- Pentoxifylline and naftidrofuryl are mainly used for treating peripheral circulatory disorders. They cause a reduction of blood viscosity, an improvement of erythrocyte fluidity, disaggregation of thrombocyte aggregates and inhibit thrombocyte aggregation.

When these substances are used for treating sudden hearing loss, possible adverse effects and possible benefit have to be weighed up. (Plontke, 2005)
The actual benefit of rheologic measures, however, is a contentious issue. Some studies show that infusions of pentoxifylline and also of dextran with pentoxyfilline added do not achieve a statistically significant gain in hearing. (http://www.leitlinien.org, 2004)

2.2.7.3 Glucocorticoids

It is a big problem that there is no generally accepted standard therapy for the treatment of sudden hearing loss. The therapy with glucocorticoids, however, has gained worldwide acceptance as "quasi-standard therapy" since it seems to achieve the best average treatment results in most cases of sudden hearing loss. (Plontke, 2005)
Steroids counteracting the inflammatory cascade spare the cochlea from continued damage and may reverse some of the loss. (Slattery et al, 2005)

The rational basis of the universal application of glucocorticoids for cochleovestibular diseases such as e.g. sudden hearing loss is the blocking of inflammatory processes. The antiphlogistic effect takes place independently of the cause (bacterial, viral, immunopathologic, chemical, physical or ischaemic-hypoxic).
In some pathologies of the inner ear, the Na/K balance in the fluids of the inner ear can be disturbed. The sodium-, potassium- and chloride-associated water inflow may result in cell swelling and electrophysiologically measurable functional losses. The Na/K balance is regulated through the Na/K-ATPase and is of great importance for the normal function of the inner ear. The influence on the ion transport through the stria vascularis is discussed to be the most important aspect of the effect of glucocorticoids. (Plontke, 2005)

Controlled, prospective and randomised studies (of relatively high evidence) on the effectiveness of glucocorticoids show a rate of remission of 59-87%. (http://www.leitlinien.org, 2004)
The commission of the *AWMF Leitlinien* (2004) recommends glucocorticoids as initial therapy for sudden hearing loss.
In addition to intravenous and oral administration of the drugs, the intratympanic injection of steroids into the middle ear is gaining more and more in importance. (Slattery et al, 2005)

2.2.7.4 Ionotropic therapy

Many years ago, local anaesthetics have already been recommended for the treatment of diseases of the inner ear and their symptoms and have since been used time and again for the therapy of tinnitus and sudden hearing loss. The mechanism of action thanks to which in particular tinnitus is reduced is still unknown. (Plontke, 2005)
The intravenous administration of large-dose local anaesthetics such as procaine or lidocaine may influence the ion transport processes of sensory cells (transduction channels), of the cells of the stria vascularis (ion transport) as well as of the afferent synapses of the inner hair cells (e.g. NMDA-receptor-associated ion channels) in the auditory system. Ionotropic therapy has to be performed in hospital since convulsive seizures, central respiratory paralysis and cardiovascular failure may occur in case of overdosage. (http://www.leitlinien.org, 2004)

2.2.7.5 Reduction of the endolymphatic volume

Plontke (2005) states in his work that clinical studies showed that sudden low-frequency hearing losses are treated better with a dehydrating therapy than with a primarily rheologic therapy. This applies in particular when there are electrophysiologic indications of an endolymphatic hydrops. Dehydration therapy according to Vollrath is based on the sugar alcohol mannitol. Diuresis takes place because this osmotic diuretic is filtered in the glomerules, but not or only partially absorbed in the tubules, which causes water to be held back in the renal tubules.

2.2.7.6 Antioxidant agents

So-called free radicals (cytotoxically reactive species of oxygen and nitrogen, ROS, NOS) that are neutralised by endogenic cellular antioxidative chemical compounds and enzymes are developed physiologically in all cells. A disturbance of the balance of the production of reactive oxygen and nitrogen compounds and the release of endogenic antioxidant agents in the inner ear may occur as a result of excessive exposition to noise, circulatory disorders induced otherwise or ototoxic substances. (http://www.leitlinien.org, 2004; Plontke, 2005)

Several antioxidant agents showed potential benefit in the animal experiment, but did not come up to the expectations in clinical studies. The therapeutic effectiveness of these substances - in particular of alpha lipoic acid - in the therapy of sudden hearing loss still has to be examined in clinical studies. (Plontke, 2005)

2.2.7.7 Inhibition of thrombocyte aggregation

is a possible therapy of acute diseases of the inner ear in the case of suspected vascular cause. Acetylsalicylic acid, prostaglandins and related products of the arachidonic acid metabolism inhibit thrombocyte aggregation. (Plontke, 2005) It has to be kept in mind, however, that acetylsalicylic acid is (reversibly) ototoxic in high doses. Controlled studies on the effectiveness of inhibitors of thrombocyte aggregation are not available. (http://www.leitlinien.org, 2004)

2.2.7.8 Lowering of fibrinogen through apheresis

Apheresis is a treatment eliminating pathogenic proteins, protein-bound pathogenic substances or pathogenic cells from the blood outside the body. (Plontke, 2005) The effectiveness of lowering of fibrinogen in the therapy of sudden hearing loss is proven through two prospective randomised studies. (http://www.leitlinien.org, 2004)
H.E.L.P. apheresis (heparin-induced extracorporeal LDL precipitation) is a method of blood purification in which substances inhibiting the blood flow or promoting blood coagulation (LDL cholesterol, lipoprotein (a) and fibrinogen) are filtered out of the blood. This improves the flow properties of the blood and the regulation of vascular width and the circulation in the small vessels of the inner ear increases. (http://www.hoersturz.info, 2005)

2.2.7.9 Hyperbaric oxygenation

The therapy with hyperbaric oxygen (HBO) is a treatment in which the patient is enclosed in a pressure chamber breathing in pure oxygen. Thanks to the elevated oxygen partial pressure in the blood more oxygen per unit time can diffuse from the blood into the tissue. (Plontke, 2005)

Animal experiments showed that hyperbaric oxygenation causes an increase in the oxygen partial pressure in the perilymph by several hundred per cent compared to the initial value. The elevated values can still be found even hours after the end of the hyperbaric oxygenation. This effect is found both in the unaffected and in the affected inner ear.
Recovery of affected cells of the sensory epithelium and the acoustic nerve fibres in the cochlea is favoured on account of the increased oxygen supply to these structures. (Plontke, 2005)

There are different, partly controversial study results on the benefit of HBO treatment in the therapy of sudden hearing loss.
In his work, Plontke (2005) states studies that prove the benefit of HBO therapy compared to rheologic therapy and also in addition to prednisolone, rheologic agents and stellate block.
This offers possibilities of using HBO as measure in addition to the "standard therapy" or as secondary therapy for patients suffering from sudden hearing loss in whom the primary therapy was not successful.

2.2.7.10 Manual therapy

The success of manual medicine for certain complaints in the ENT region can hardly be denied, it is however true that its importance is still assessed divergently. (Hülse, 2004) Several authors report that chiropractic treatment of the cervical spine improves symptoms such as vertigo, tinnitus and hearing loss. (Kessinger and Boneva, 2000)
In his article "Effektivität der manuellen Medizin in der HNO" [Effectiveness of manual medicine in ENT medicine], Hülse (2004) reports on the patients' good therapy satisfaction in case of complaints such as vertigo, hearing disorders and disorders of voice. After six months, 72% of the patients with vertigo, 59.2% of the hearing disorders and even 83.3% of the voice disorders and still 29.8% of the patients suffering from tinnitus have improved or were free of complaints.
The cochlear symptoms with cervicogenic hearing disorder are completely reversible and disappear when the functional disorder of the head joints has been cured through manual therapy. (Hülse, 1994)
Moderate hearing impairments by more than 35 dB, which, according to Hülse (1994), should not be classified as purely cervicogenic hearing impairments any more, and that show functional problems in the area of the head joints, benefit from manual therapy as well. In these cases, the improvement of the hearing threshold amounted to max. 20 dB – which the patients, however, already found very positive.
In their case study, Kessinger and Boneva (2000) report on a geriatric patient with episodes of vertigo, pain and sensation of pressure in the left ear and hearing loss in both ears that got increasingly worse. After a few weeks of chiropractic therapy, she no longer had any attacks of vertigo and her hearing improved considerably. The audiogram showed improvements in all frequencies after 13 weeks of treatment. The most important changes were found in the high-frequency range which showed improvements by 15 dB – 35 dB. The final audiometry 9 ½ months after the beginning of the treatment showed improvements in some frequencies - in particular in the high frequencies - and regression in other frequencies.

2.2.8 Prognosis

According to Halpin and Rauch (2003), sudden sensorineural hearing loss is one of only a few sensorineural disorders that are known to be reversible in some cases.

In all, the range of possible results is very large. There are for example untreated cases with complete remissions as well as treated cases without any improvement. (Halpin and Rauch, 2003)

The percentages given in literature for spontaneous remissions range from 30 % to 70 % and normally occur within the first two weeks. (Fayad and De La Cruz, 2003; http://www.hear.it.org, 2005; http://www.hoersturz.info, 2005; Wazen and Ghossaini, 2003; Zadeh et al, 2003)

The time between the hearing loss and the start of therapeutic measures is an important factor in prognosis. (Fritsch et al, 2003; Miller and Schein, 2005) Zadeh et al (2003) report significantly better recovery of patients who receive treatment within 3 days.
Presence of vertigo is generally considered to be an indicator of a rather bad prognosis. (Chang et al, 2005; Fayad and De La Cruz, 2003; Fritsch et al, 2003 ; Slattery et al, 2005; Wazen and Ghossaini, 2003) Zadeh et al. (2003), however, state that in their study vertigo did not turn out to be an indicator of a rather bad prognosis in contrast to earlier reports.
The more serious the initial hearing loss, the worse is the prognosis. (Fayad and De La Cruz, 2003; Slattery et al, 2005; Wazen and Ghossaini, 2003)
The patient's age at the onset of the disease is an essential factor for prognosis. Zadeh et al (2003) showed that patients who are younger than 40 years at the time of the sudden hearing loss have a statistically significantly higher rate of remission ($P<0.05$).

Several authors report that the initial course of the audiogram may provide indications of the recovery to be expected. Chang et al (2005) performed a study on this matter. According to that study, "mid-tone loss patterns" have the best prognosis.

According to Schüßler et al (1992), stable circumstances of life, an emotionally stable personality without psychiatric disorders and a reasonable change of the lifestyle are predictors of a favourable course.

2.2.9 Epidemiology

There are no confirmed data on the incidence of sudden hearing loss available in literature. The data spread for the most part are estimations. According to *AWMF online Leitlinien* (2004), the number of new cases in Germany and Austria amounts to 20 / 100,000 inhabitants / year.
In Japan, the number of patients treated for sudden hearing loss in university hospitals constantly increased from the seventies to the nineties. (Nakashima et al, 2000) According to Nakashima et al (2000), 30 % of the patients suffering from sudden hearing loss were treated in university hospitals in Japan in 1993. According to that, the estimated incidence amounts to 13 cases per 100,000 inhabitants per year.
In the United States, the incidence is estimated at values between 5 and 25 new cases / 100,000 / year. (Fayad and De La Cruz, 2003; Halpin and Rauch, 2003; Miller and Schein, 2005; Wazen and Ghossaini, 2003; Zadeh et al, 2003)
The incidence in men and women is approximately equal. (http://www.leitlinien.org, 2004; Wazen and Ghossaini, 2003)
The average age of illness is between 40 and 50 years, however with downward trend. (Wazen and Ghossaini, 2003) Approximately 60 % of the patients are aged between 30 and 60. (Yasar, 2001) Children, adolescents and young adults under 20 are only very rarely affected by sudden hearing loss. (http://www.leitlinien.org, 2004; Yasar, 2001)
Zadeh et al (2003) report in their study on a higher incidence with increasing age.

3 Materials and method

3.1 Study design

This study is a controlled user observation, with an intervention group and a control group. 15 patients were included in the user observation – five in the intervention group, ten in the control group. The user observation is not blinded and not randomised. The patients of the intervention group were recruited within the period from the early January 2006 to the end of August 2006 in a match controlled manner. The control group was determined retrospectively by means of analysis of the audiograms.

3.1.1 Intervention group

The intervention group consists of five patients (three male, two female patients) at the age of 50 – 62 (mean: 54.4a) and in three patients the right and in two patients the left side is affected. The patients sought medical treatment between the first and the eleventh day after the sudden hearing loss, two patients (2, 5) having already had ear problems (sensation of pressure, tinnitus) some time (2 approx. two weeks, 5 approx. 10 weeks) before the actual sudden hearing loss. In-patient treatment took seven days in four patients and eight days in one patient. One patient (3) had already had a sudden hearing loss earlier on the same side with a permanent hearing impairment of 40 %, corresponding to a hearing threshold of approximately 40 dB. (Probst, 2004). One patient (5) has a permanent hearing impairment on the opposite side. All patients underwent an examination by the ENT specialist and were treated with drugs as in-patients before the osteopathic treatment. The interval between the last osteopathic treatment and the audiometry the value of which were then used as endpoints for comparison amounted to one to eleven days (mean 5.8d). Three patients (1, 2, 3) had an infection of the upper airways immediately before the sudden hearing loss to approximately one month before it. Two patients (3, 4) had a cardiopulmonary bypass surgery 8 and 16 months respectively before the sudden hearing loss and two patients (2, 5) were exposed to stress immediately before the event.

Patient	Age	Sex	Side
PU1	58	M	Right
PU2	52	M	Right
PU3	62	M	Left
PU4	50	F	Left
PU5	50	F	Right

The table shows age, sex and side of the affected ear of the patients of the intervention group.

3.1.1.1 Patient recruitment

Recruitment of patients for the intervention group mainly was accomplished through the Otorhinolaryngologic Department of the hospital LKH Feldkirch under the direction of Prim. Dr. Wolfgang Elsäßer and Dr. Rene Summesberger. When it turned out that too few patients were eligible for the study, I included additional ENT specialists for assistance in recruiting ten weeks after the beginning of the study: Dr. Peter Wachter (ENT specialist with cottage-hospital affiliation in the LKH Bludenz), Dr. Georg Hollenstein (ENT specialist with cottage-hospital affiliation in the LKH Bregenz), Dr. Georg Kanonier (ENT specialist with cottage-hospital affiliation in the KH Dornbirn) and Dr. Wolfgang Feuerstein (otorhinolaryngologist in own practice in Dornbirn).

3.1.1.2 Inclusion criteria

On account of the difficulties regarding the recruitment of patients, inclusion criteria are very broad. It turned out during the course of the study only that only a very small number of patients suffering from a sudden hearing loss still had significant hearing impairments after the in-patient infusion therapy and injections of cortisone.

- sudden unilateral hearing loss
- hearing impairment by at least 30 dB in three adjacent frequencies
- sudden hearing loss since one to at most three weeks
- in-patient drug treatment completed
- age between 30 and 75 years
- persistent objective hearing impairment
- voluntary participation

3.1.1.3 Exclusion criteria

- sudden hearing loss longer than 3 weeks ago
- missing patient compliance

3.1.2 Control group

The control group included 10 patients aged 38 – 71 years (mean: 52.4a), seven male and three female patients. The left ear was affected in eight patients and the right ear in two patients. No data are available on the duration of the in-patient treatment and on the interval from the occurrence of the sudden hearing loss to the start of treatment.

Patient	Age	Sex	Side
PK1	70	M	Left
PK2	53	F	Left
PK3	42	F	Left
PK4	38	M	Left
PK5	51	M	Left
PK6	45	M	Left
PK7	71	F	Left
PK8	60	M	Right
PK9	54	M	Left
PK10	40	M	Right

The table shows age, sex and side of the affected ear of the patients of the control group.

3.1.2.1 Inclusion criteria

The control group was established retrospectively. To this effect, the audiograms of 146 patients who were diagnosed with sudden hearing loss and underwent in-patient treatment in the LKH Feldkirch from early November 2005 to the end of October 2005 were examined.

- one-sidedness of the hearing impairment
- hearing impairment by at least 30 dB in three adjacent frequencies
- age between 30 and 75 years
- persistent hearing impairment after in-patient therapy – evident from the follow-up audiogram
- availability of a follow-up audiogram at least 2 weeks after the audiogram of the initial condition

3.1.3 Target parameter

The target parameter of this study is the change in the hearing threshold determined by means of pure tone audiometry (PTA) and presented in the audiogram.

3.2 Pure tone audiometry

Comparability of the individual patients and of the two groups is based on the hearing threshold determined by means of tone audiometry and shown in the audiogram.

The determination of the hearing threshold is one of the most important and most frequently performed examinations in audiology. (Mrowinski and Scholz, 2002)
Hearing threshold means the sound pressure level of an acoustic stimulus on which this stimulus can just be perceived. (Probst, 2004)
Pure tones are generated by an audiometer and played to the patient through headphones with increasing sound pressure level (volume). In practice, the frequencies from 125 Hz to 8 kHz are checked. The patient then states from which sound pressure level on he/she hears the sound. The frequencies and the corresponding decibel values of the sound pressure level are then entered into the audiogram. (Probst, 2004)

Accuracy of the determination of the threshold depends less on the audiometer than on the patient's attention. Repeated measurements may well show deviations by up to 10 dB in air conduction. The audiogram will become clearer and will not put on wrong accuracy if the measuring points are entered accurately to 5 dB even if the audiometer allows more accurate adjustments. (Mrowinski und Scholz, 2002)

Possible inaccuracies when entering and/or reading the values in the audiogram are another source of error.

This thesis compares and evaluates the hearing threshold values of both groups determined at the beginning and at the end of the period of observation by means of audiometry. The audiometry includes the frequencies of 125 Hz, 250 Hz, 500 Hz, 1 kHz, 1.5 kHz, 2 kHz, 3 kHz, 4kHz, 6 kHz and 8 kHz.

For evaluation, the mean values of the individual frequencies are calculated and the compared with each other.

3.3 Realisation of the study

3.3.1 Course

Patients who met the inclusion criteria for the intervention group and gave their consent to the participation in the study were included in the study and treated according to general osteopathic principles.
Four osteopathic treatments were then performed within a period of four weeks. The interval between the first and the second treatment amounted to three to four days, between the second and the third treatment to six to nine days and between the third and fourth treatment to thirteen to eighteen days.
The initial presentation included the establishment of the case history and a comprehensive osteopathic examination on the basis of which the osteopathic treatment was performed.
The patients of the intervention group underwent osteopathic treatment for a period of approximately four weeks following in-patient drug treatment. New osteopathic examinations and treatments were performed in the other treatment sessions.

At the end of the osteopathic treatment, another tone audiometry was performed the values of which were then used as final condition for comparison with the initial condition.

3.3.2 Initial condition

The values of the follow-up audiogram on discharge from hospital or the last available audiogram before the start of the osteopathic treatments was used as initial condition.

For the control group, the available audiometry values four to ten days after the start of treatment were used as initial condition.

3.3.3 Final condition

At the end of the series of osteopathic treatments another audiometric hearing test was carried out. This value constitutes the final condition. The comparison period for the intervention group was 33.2 days on average (shortest period: 28d and longest period: 43d).

For the control group, the available audiometry data of at least two weeks after the beginning of the comparison period were used. The observation period of the control group amounted to 59.5 days on average, the shortest follow-up amounting to 14 and the longest to 241 days.

3.4 Statistical analysis

The measuring results for the different frequencies were first checked for outlier values in the original records.
The original values were manually copied into an Excel spreadsheet and processed for presentation for the SPSS 13.0 (SPSS Inc., California).
"Repeated analysis of variance" was used for establishing the frequency differences before and after the intervention. These frequency differences were "within-subjects effects" in the analysis. The "Bonferonni correction for multiple comparisons" was applied *post hoc* and the "Huynh-Feldt adjustment for non-sphericity" was done.

4 Study results

Measure	Measure	Mean	Std. Error	95% Confidence Interval	
				lower bound	upper bound
125Hz	1	40,000	6,124	22,998	57,002
	2	32,500	5,123	18,275	46,725
250Hz	1	38,000	7,842	16,227	59,773
	2	36,000	6,964	16,664	55,336
500Hz	1	41,000	7,314	20,692	61,308
	2	37,000	6,042	20,226	53,774
1kHz	1	47,000	5,612	31,417	62,583
	2	41,000	6,403	23,222	58,778
1,5kHz	1	42,500	7,826	20,771	64,229
	2	45,000	9,618	18,297	71,703
2kHz	1	48,000	9,566	21,442	74,558
	2	45,000	10,840	14,904	75,096
3kHz	1	55,000	6,708	36,375	73,625
	2	41,000	10,654	11,421	70,579
4kHz	1	51,000	11,979	17,741	84,259
	2	48,000	11,895	14,973	81,027
6kHz	1	68,000	15,215	25,756	110,244
	2	61,000	16,462	15,294	106,706
8kHz	1	65,000	17,321	16,911	113,089

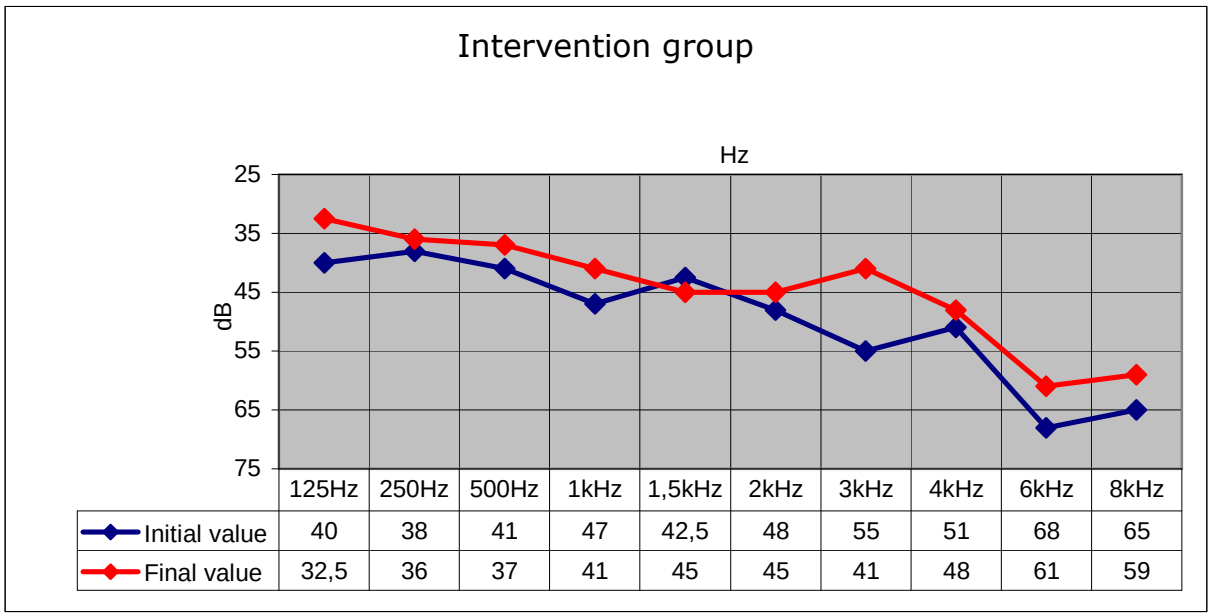
Intervention group: comparison of the mean values in the individual frequencies, initial value (1) and final value (2), standard deviation, lower and upper limit

The table shows the mean values of the intervention group in the frequencies from 125 Hz to 8 kHz. Measurement one shows the mean value, the standard deviation as well as the lower and upper limit of the 95% confidence interval of the initial value. Measurement two shows the final values.

Measure	Measure	Mean	Std. Error	95% Confidence Interval	
				lower bound	upper bound
125Hz	1	36,876	6,788	21,521	52,231
	2	29,444	5,698	16,554	42,334
250Hz	1	39,000	7,102	22,933	55,067
	2	32,000	7,272	15,549	48,451
500Hz	1	40,000	7,491	23,055	56,945
	2	34,000	6,616	19,032	48,968
1kHz	1	35,500	7,433	18,685	52,315
	2	27,500	6,760	12,208	42,792
1,5kHz	1	34,000	7,775	16,413	51,587
	2	27,500	7,500	10,534	44,466
2kHz	1	32,500	8,207	13,934	51,066
	2	27,500	7,500	10,534	44,466
3kHz	1	45,500	8,315	26,690	64,310
	2	42,500	7,426	25,702	59,298
4kHz	1	54,500	7,128	38,376	70,624
	2	53,000	6,675	37,900	68,100
6kHz	1	58,500	6,238	44,388	72,612
	2	59,500	5,080	48,008	70,992
8kHz	1	60,500	4,913	49,386	71,614
	2	60,000	4,944	48,816	71,184

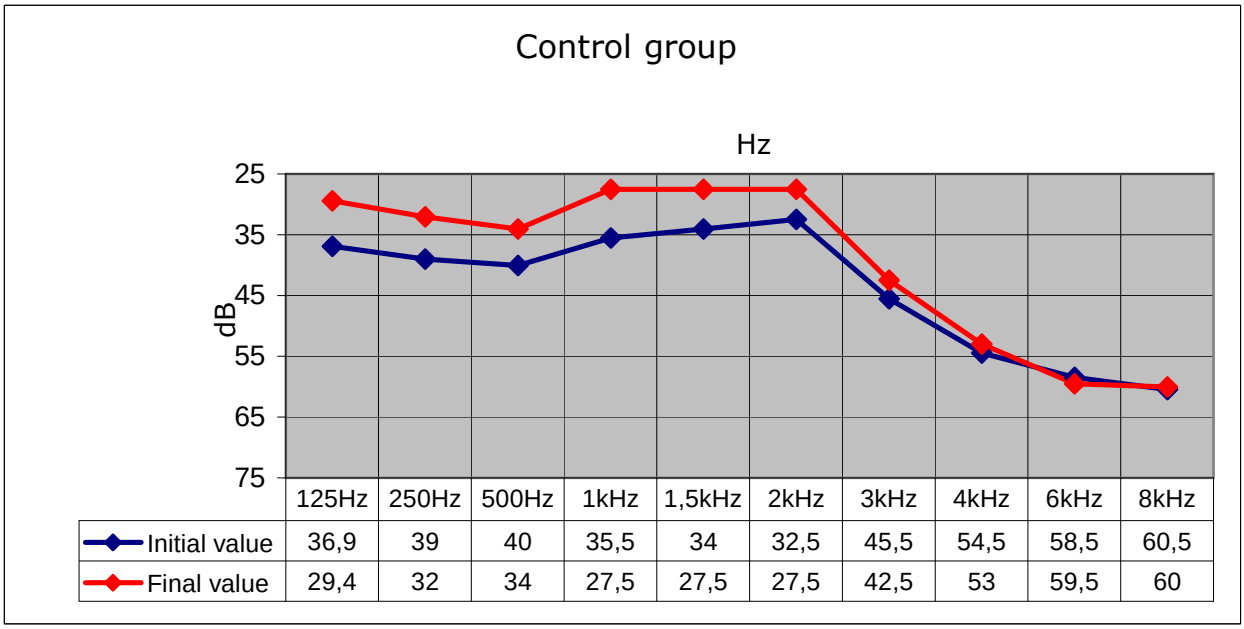
Control group: comparison of the mean values in the individual frequencies, initial value (1) and final value (2), standard deviation, lower and upper limit

The table shows the mean values of the control group in the frequencies from 125 Hz to 8 kHz. Measurement one shows the mean value, the standard deviation as well as the lower and upper limit of the 95% confidence interval of the initial value. Measurement two shows the final values.



	125Hz	250Hz	500Hz	1kHz	1,5kHz	2kHz	3kHz	4kHz	6kHz	8kHz
Initial value	40	38	41	47	42,5	48	55	51	68	65
Final value	32,5	36	37	41	45	45	41	48	61	59

The diagram shows the comparison of the mean values of the hearing threshold of the intervention group before (initial value) and after (final value) the osteopathic treatments.



	125Hz	250Hz	500Hz	1kHz	1,5kHz	2kHz	3kHz	4kHz	6kHz	8kHz
Initial value	36,9	39	40	35,5	34	32,5	45,5	54,5	58,5	60,5
Final value	29,4	32	34	27,5	27,5	27,5	42,5	53	59,5	60

The diagram shows the comparison of the mean values of the hearing threshold of the control group at the beginning (initial value) and at the end (final value) of the period of observation.

5 Discussion

5.1 Problems in patient recruitment

The recruitment of patients turned out to be the biggest problem in the context of this work. Although I have tried to find out already when preparing the draft whether there are enough patients available for the realisation of the study, problems still occurred at the stage of realisation. There would have been enough patients suffering from a sudden hearing loss, but after the in-patient drug therapy only a very small number of patients were left who were then eligible for a participation in the study.
It is possible that a participation of patients failed to take place for organisational reasons. This can happen for example when the patient was not informed in time of the possibility of participating in the study and/or it was not checked on discharge whether the patient meets the inclusion and exclusion criteria.
This is how a very small intervention group of five patients was created in the end.
Since it could be seen already very early that it would not be possible to get enough patients for the intervention and the control group, I had to change my strategy. First of all, I extended my catchment area and included additional physicians for patient recruitment. This step, however, was not enough, so I chose to establish the control group retrospectively. A special thank goes to the Otorhinolaryngologic Department of the hospital LKH Feldkirch and its head, Prim. Dr. Wolfgang Elsässer, and Dr. Rene Summesberger who placed the necessary audiometry data at my disposal. Thanks to that I was then able to form a control group of ten patients. A great drawback, however, consists in the fact that I do not have any further information on these patients' case history apart from the audiometry data.
In my opinion, it is extremely difficult to carry out a scientific study as osteopath in private practice since you are always confronted with difficulties of communication, of the flow of information and organisation.

5.2 Discussion of the methods

The difficulties of patient recruitment also had an effect on the method and I was constrained to change the planned study design.
For the reasons already mentioned above, it was not possible to carry out the prospective separation into groups planned initially – to divide the patients meeting the inclusion and exclusion criteria into intervention and control group by means of a randomisation list. Therefore the two groups are inhomogeneous – differing essentially in size, sex ratio and the follow-up intervals.

It was necessary for organisational reasons to start the osteopathic treatment after the in-patient therapy in hospital. Another reason of the start of the osteopathic treatment after an interval of up to three weeks is the very high rate of spontaneous remission of 30% to 65% of the cases without any treatment (Fayad and De La Cruz, 2003). The delayed start of the osteopathic treatment may improve the presentation of its effect since spontaneous remission and improvement of the symptoms thanks to drug treatment can be excluded and/or have less influence.
The follow-up intervals of the two groups vary clearly. The mean observation period of the intervention group is 33.2 days, whereas with 59.5 days it is nearly twice as high for the control group. This is a fact that clearly limits the comparability of the two groups. This period of time automatically resulted for the control group from the available audiometry data and the difference between the shortest (14d) and the longest (241d) observation period also is very high. On account of the fact that the user observation has already been in progress for a rather long time when I was compelled to change the planned design and to generate the control group retrospectively, I was no longer able to adapt the observation period of the intervention group to that effect.
It might have been better to define the moment for the audiometry identifying the final condition differently. In the present study, the final hearing test was carried out between one and eleven days (on average 5.8d) after the last osteopathic treatment. This took into account a very essential basic idea of osteopathy to an insufficient extent only, that is that the human body needs enough time in order to react to treatment stimuli and to implement the changes initiated. Therefore, in retrospect, a follow-up of the hearing threshold would have been indicated three weeks (ideally four to five weeks) after the end of the osteopathic treatment at the earliest. This assumption is supported by the fact that the hearing threshold of patient PU2 continued to improve after the observation period. This was detected in an audiometry carried out three months later.

5.3 Discussion of the results

When comparing the results of the two groups you can see that there are only minor differences.
The intervention group showed an improvement of the hearing threshold by 5 dB on average throughout the entire hearing range tested. Compared to that, the hearing threshold of the control group improved by 4.4 dB. The difference of 0.6 dB in the intervention group's favour is too small for identifying a trend.
This is particularly clear when visualising that the values of audiometry are presented in steps of 5 dB.

It is noticeable in the control group that improvements mainly take place in the low- and mid-frequency range and constantly amount to 5 dB and more up to 2 kHz. By contrast, only minor improvements are found in the high-frequency range from 3 kHz on.
The intervention group shows improvements in the low- and in the high-frequency range, but also a deterioration at 1.5 kHz.

It is interesting that the hearing threshold recovered equally in both groups at 125 Hz and only shows a small standard deviation here as well. The regeneration potential of the cochlea in the low-frequency (apical) area might be higher than in the higher frequencies that are perceived nearer at the base of the cochlea.
In the high-frequency range, clearly greater hearing benefits are found compared to the control group, but the standard deviation and the spread of the 95% confidence interval are already relatively large in this range. Kessinger and Boneva (2000) report in their case study on greater hearing benefits (15 dB – 35 dB) in the high-frequency range as well.

The question expressed in the beginning whether osteopathic treatments result in a greater improvement of the hearing threshold than drug therapy alone, would have to be answered "no" according to this thesis. The circumstances, however, that made the realisation of this work difficult and complicated it also resulted in an inhomogeneous group constellation that is not comparable in the end. Therefore it is not possible to answer this question based on this study.
For answering the question it would be necessary to do further investigations. When doing this, homogeneous groups of patients and equal conditions for all patients should be sought for. For the time being, the range could be limited to the low frequencies, since there are already

reports on positive effects of manual treatments for hearing impairment in literature (Hülse, 1994 and 2004).

It is true that the results of this work do not show any objectifiable changes in the recovery of the hearing threshold, the osteopathic treatments nevertheless have some very positive aspects for the patients. The symptoms accompanying the sudden loss in hearing such as vertigo, sensation of pressure in the head, sense of fullness in the ear, tinnitus and general feeling of being ill have improved partially or completely according to the patients' subjective statements. And all patients were calmer and more relaxed in dealing with their disease and might be the reason why they found the hearing impairment less irritating.

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7 Annex

7.1 Black Box

The patients were examined and treated according to general osteopathic criteria.

The osteopathic examination showed the striking fact that all patients had dysfunctions in the area of the upper cervical spine. The intensity of these functional disorders varied, but C0 and C1 were not functional in all cases. The first cervical vertebra was translated towards the side of the hearing impairment. C2 and C3 were rotated towards the side of the affected ear in three out of five patients. Several other individual dysfunctions were found in the area of the cervical spine, which I do not mention separately here since they occurred only individually. Palpation findings revealed another common condition at the cervicothoracic transition. This area was very rigid in all patients and allowed only little movement. The seventh cervical vertebra was in extension position and the first thoracic vertebra was in flexion position with the first ribs blocked on both sides.

Musculature also showed a relatively uniform picture characterised by hypertonicity in the craniocervical area as well as of the sternocleidomastoid muscle, the trapezius muscle, the scalenus muslces and the subclavius muscle.

In three patients, the thoracic spine showed an increased kyphosis and restricted mobility. This was accompanied by a poor thoracic mobility, and it has to be mentioned in particular that two of these patients had had a bypass surgery. In the area of the pelvis, sacrum torsion was found in two patients. It was interesting in this context that the patient suffering from the sacrum torsion left/left had the sudden loss of hearing on the left side as well and the patient suffering from the sacrum torsion right/right had the hearing impairment on the right side.

The anamnesis of all patients revealed a more or less serious physical trauma in their past. This includes events such as fall onto the coccyx,

whiplash injury of the cervical spine (two patients), fracture of the zygomatic bone and cerebral contusion. One patient also had had meningitis and another patient had had a dislocation of the shoulder joint twice that had been sanitised surgically. This information from the patients' case histories suggests that the cranial mechanism probably does not work perfectly.
And indeed, all patients showed a functional disorder of the cranial system. The reciprocal tension membrane (RTM) was very tense in all patients. This resulted to a massively limited inherent cranial mobility and a hardly perceptible "Cranial Rhythmic Impulse" (CRI). The sphenobasilar symphysis (SBS) showed the pattern of an extension dysfunction in all patients and in one patient a lateral strain on the right.

No conspicuous dysfunctions were found in the visceral region.

Mainly structural and cranial techniques were used for the treatment.

The aims in the structural area were the improvement of articular mobility, muscular relaxation, improvement of the metabolic condition and calming of the vegetative nerve system in the area of the cervical spine, the shoulder girdle as well as the thoracic spine with the entire thorax.

To this effect, I applied the following techniques: "General Osteopathic Techniques" (GOT), "Muscle Energy Techniques" (MET), "Soft Tissue Techniques", muscular stretching, inhibition techniques, listening techniques and articular mobilisation techniques.

In the cranial area, I used Balanced Membranous Tension techniques (BMT) for relaxing the RTM and indirect techniques for correcting the SBS dysfunctions.

While I looked more intensely in the topic of anatomy of the auditory organ and its connections within the scope of this work, I noticed some facts that are interesting from the osteopathic point of view.

- There are neural connections from the nerve roots C2 and C3 to the central areas of the cranial nerve nuclei (dorsal vestibulocochlear nucleus) (Arvidsson and Pfaller, 1990; Pfaller and Arvidsson, 1988)

- Hack et al (1995) showed direct connections of connective tissue between the rectus capitis posterior minor muscle and the underlying posterior atlantooccipital membrane and this membrane with the spinal dura mater.

- The cerebral membranes stretch through the internal acoustic pore into the external acoustic meatus and the subarachnoidal spaces communicate with the perilymphatic spaces of the inner ear. (Reiss, 2003)

- The facialis, intermedius and vestibulocochlear nerves run together with the labyrinthine artery, that is surrounded by sympathetic plexus, in the internal acoustic meatus. (Fanghänel et al, 2003)

7.2 Patient information

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Sehr geehrte/r Patient/in!

Vorweg einige Informationen zu meiner Person.

Ich bin seit 1995 diplomierter Physiotherapeut und habe mich nach drei Jahren Berufserfahrung im Angestelltenverhältnis in den Krankenhäusern Feldkirch und Hohenems als freiberuflicher Physiotherapeut niedergelassen. Danach habe ich mit der sechsjährigen berufsbegleitenden Ausbildung zum Osteopathen an der Wiener Schule für Osteopathie begonnen und 2001 abgeschlossen. Gegenwärtig besuche ich den Universitätslehrgang M. Sc. Osteopathie an der Donauuniversität in Krems, zu dem auch das Erstellen einer wissenschaftlichen Diplomarbeit gehört.

Für meine Diplomarbeit führe ich eine klinische Studie zum Thema Hörsturz durch.

Ziel der Studie ist es festzustellen, ob osteopathische Behandlungen die Regeneration des Hörvermögens nach einem Hörsturz unterstützen.

Osteopathie ist eine Behandlungsform, die Ihnen möglicherweise dabei helfen kann, Ihr Hörvermögen rascher und besser wiederzuerlangen, als dies bei alleiniger medikamentöser Therapie möglich ist.

Die Durchführung einer solchen Studie ist natürlich nur in Zusammenarbeit mit Ihnen - einem/r betroffenen Patienten/in - möglich.

Sie haben die Möglichkeit, an einer Studie zur Erforschung der Wirksamkeit osteopathischer Behandlungen in der Hörsturztherapie teilzunehmen.

Die Teilnahme an der Studie ist freiwillig.

Die osteopathischen Behandlungen sind für Teilnehmer an der Studie kostenlos.

Während der Teilnahme an der Studie dürfen außer den vom Arzt verordneten Medikamenten und den osteopathischen Behandlungen keine weiteren Therapien durchgeführt werden.

Die Teilnahme an der Studie kann vom Patienten jederzeit abgebrochen werden. Im Falle eines beabsichtigten Austretens aus der Studie ist dies unbedingt 24h vor dem nächsten vereinbarten Termin bekannt zu geben. Gleichzeitig möchte ich Sie ersuchen, wenn Sie sich für eine Teilnahme an der Studie entschlossen haben auch bis zum Ende dabei zu bleiben, da die Durchführung einer Studie einen sehr großen zeitlichen und finanziellen Aufwand darstellt.

Mit Ihrer Teilnahme unterstützen Sie mich bei der Durchführung und möglicherweise auch zukünftige Patienten/innen mit demselben Problem.
Nach Abschluss der medikamentösen Behandlung im Krankenhaus kann mit den osteopatischen Behandlungen begonnen werden. In einem Zeitraum von vier Wochen werden Sie viermal osteopathisch behandelt. Bei der ersten Sitzung wird eine genaue Krankengeschichte erhoben und der gesamte Körper manuell untersucht. Auf Basis der vorgefundenen körperlichen Merkmale wird dann ein Behandlungsplan erstellt und ausgeführt. Die angewandten Techniken stellen keinerlei Risiko für Sie dar. Es können lediglich Reaktionen im Sinne von vorübergehenden funktionellen Beschwerden wie Muskelkater, muskuläre Verspannungen und Müdigkeit auftreten.

7.3 Declaration of consent

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Einverständniserklärung

Name

Vorname

Geburtsdatum

- Hiermit erkläre ich mich einverstanden, an der osteopathischen Studie „Hörsturz" teilzunehmen.
- Ich wurde über den Hintergrund und Zweck der Studie sowie mögliche Nebenwirkungen informiert.
- Ich erkläre mich damit einverstanden, dass die für die Studie relevanten Untersuchungsdaten (z. B. Tonaudiogramme,…) vom behandelnden Arzt/ Institution an Uwe Staffa weitergegeben werden.

Die Daten werden im Sinne des Datenschutzgesetzes streng vertraulich behandelt.
Die Auswertung der Daten in der Studie erfolgt anonymisiert.

Unterschrift:_____ Datum

7.4 Findings sheet

Untersuchungsgruppe Datum

Patient Nr:

Name

Geb. Datum

Hörsturz

Seit wann

Wie angefangen

über Nacht plötzlich

Symptome Hörminderung Seite?

Schwindel

Tinnitus

Übelkeit / Benommenheit

Wattegefühl im Ohr

Therapie Medikamente:

Andere Probleme:

Operationen:

Unfälle/ Stürze/ Verletzungen:

Atmung

häufig erkältet Bronchitis Pneumonie Nasenneben-/Stirnhöhlen

Ernährung

Unverträglichkeiten

Diäten

Verdauung

Ösophagus

Magen Sodbrennen Gastritis

Duodenum

Dünndarm

Dickdarm

Ausscheidung Verstopfung Durchfall

Leber

Pankreas
Milz
Flüssigkeitshaushalt
Nieren
Blase
Uterus/Ovarien
Prostata
Soziales Umfeld:
- Berufliche Tätigkeit
- Streß
- Beziehung
- Familie
- Arbeitsplatz
- Sport

Untersuchungsbefund:
General listening
Local listening
Sotto Hall
Strukturell:
WS HWS

BWS

LWS

Becken/ Sacrum/ Coccygis
Thorax/Rippen
Viszeral

Cranial

Arbeitshypothese

Zielsetzung

Behandlung:

7.5 Audiometric values

7.5.1 Audiometric values of the intervention group

7.5.1.1 Initial values of the intervention group

Patient	125Hzv	250Hzv	500Hzv	1kHzv	1,5kHzv	2kHzv	3kHzv	4kHzv	6kHzv	8kHzv
PU1	55	55	65	65	70	70	60	65	115	105
PU2	50	50	45	45	25	15	-	15	30	15
PU3	35	35	40	45	45	55	70	75	80	75
PU4	20	10	20	30	30	40	30	30	40	35
PU5	40	40	35	50	-	60	60	70	75	95

7.5.1.2 Final values of the intervention group

Patient	125Hzn	250Hzn	500Hzn	1kHzn	1,5kHzn	2kHzn	3kHzn	4kHzn	6kHzn	8kHzn
PU1	45	50	50	65	75	75	65	70	105	105
PU2	40	45	45	30	20	10	5	10	10	10
PU3	30	35	35	40	45	50	55	65	80	70
PU4	15	10	15	30	30	35	30	30	40	35
PU5	-	40	40	40	55	55	50	65	70	75

7.5.2 Audiometric values of the control group

7.5.2.1 Initial values of the control group

Patient	125Hzv	250Hzv	500Hzv	1kHzv	1,5kHzv	2kHzv	3kHzv	4kHzv	6kHzv	8kHzv
PK1	70	65	60	40	30	30	55	65	65	75
PK2	30	40	45	50	55	60	60	65	75	80
PK3	15	10	10	10	15	10	25	35	45	45
PK4	15	20	15	10	15	10	20	40	50	65
PK5	75	80	80	80	80	70	75	70	60	55
PK6	45	50	60	60	60	65	75	85	85	75
PK7	-	50	50	15	10	10	10	25	20	40
PK8	-	30	30	45	45	50	60	65	75	75
PK9	30	30	40	25	20	10	10	20	40	40
PK10	15	15	10	20	10	10	65	75	70	55

7.5.2.2 Final values of the control group

Patient	125Hzn	250Hzn	500Hzn	1kHzn	1,5kHzn	2kHzn	3kHzn	4kHzn	6kHzn	8kHzn
PK1	50	50	45	20	15	20	50	60	65	60
PK2	25	30	40	40	55	60	60	70	70	70
PK3	15	10	10	10	15	15	25	40	55	50
PK4	15	10	15	15	10	10	15	35	40	65
PK5	70	75	75	80	80	75	70	65	55	45
PK6	15	15	15	10	10	15	55	65	70	70
PK7	-	60	55	20	10	10	15	25	45	45
PK8	30	35	35	40	40	45	60	70	80	80
PK9	30	25	35	25	25	15	10	20	35	35
PK10	15	10	15	15	15	10	65	80	80	80

7.6 SPSS Summary table of the results

7.6.1 Univariate testing of the intervention group

Univariate Tests(a)

Source	Measure		Type III Sum of Squares	df	Mean Square	F	Sig.
measure	125Hz	Sphericity Assumed	140,625	1	140,625	45,000	,003
		Greenhouse-Geisser	140,625	1,000	140,625	45,000	,003
		Huynh-Feldt	140,625	1,000	140,625	45,000	,003
		Lower-bound	140,625	1,000	140,625	45,000	,003
	250Hz	Sphericity Assumed	10,000	1	10,000	2,667	,178
		Greenhouse-Geisser	10,000	1,000	10,000	2,667	,178
		Huynh-Feldt	10,000	1,000	10,000	2,667	,178
		Lower-bound	10,000	1,000	10,000	2,667	,178
	500Hz	Sphericity Assumed	40,000	1	40,000	1,455	,294
		Greenhouse-Geisser	40,000	1,000	40,000	1,455	,294
		Huynh-Feldt	40,000	1,000	40,000	1,455	,294
		Lower-bound	40,000	1,000	40,000	1,455	,294
	1kHz	Sphericity Assumed	90,000	1	90,000	4,235	,109
		Greenhouse-Geisser	90,000	1,000	90,000	4,235	,109
		Huynh-Feldt	90,000	1,000	90,000	4,235	,109
		Lower-bound	90,000	1,000	90,000	4,235	,109
	1,5kHz	Sphericity Assumed	15,625	1	15,625	,714	,446
		Greenhouse-Geisser	15,625	1,000	15,625	,714	,446
		Huynh-Feldt	15,625	1,000	15,625	,714	,446
		Lower-bound	15,625	1,000	15,625	,714	,446
	2kHz	Sphericity Assumed	22,500	1	22,500	2,250	,208
		Greenhouse-Geisser	22,500	1,000	22,500	2,250	,208
		Huynh-Feldt	22,500	1,000	22,500	2,250	,208
		Lower-bound	22,500	1,000	22,500	2,250	,208
	3kHz	Sphericity Assumed	490,000	1	490,000	2,096	,221
		Greenhouse-Geisser	490,000	1,000	490,000	2,096	,221
		Huynh-Feldt	490,000	1,000	490,000	2,096	,221
		Lower-bound	490,000	1,000	490,000	2,096	,221
	4kHz	Sphericity Assumed	22,500	1	22,500	1,385	,305
		Greenhouse-Geisser	22,500	1,000	22,500	1,385	,305

	Huynh-Feldt	22,500	1,000	22,500	1,385	,305
	Lower-bound	22,500	1,000	22,500	1,385	,305
6kHz	Sphericity Assumed	122,500	1	122,500	3,500	,135
	Greenhouse-Geisser	122,500	1,000	122,500	3,500	,135
	Huynh-Feldt	122,500	1,000	122,500	3,500	,135
	Lower-bound	122,500	1,000	122,500	3,500	,135
8kHz	Sphericity Assumed	90,000	1	90,000	2,667	,178
	Greenhouse-Geisser	90,000	1,000	90,000	2,667	,178
	Huynh-Feldt	90,000	1,000	90,000	2,667	,178
	Lower-bound	90,000	1,000	90,000	2,667	,178
measure * gruppe 125Hz	Sphericity Assumed	,000	0	.	.	.
	Greenhouse-Geisser	,000	,000	.	.	.
	Huynh-Feldt	,000	,000	.	.	.
	Lower-bound	,000	,000	.	.	.
250Hz	Sphericity Assumed	,000	0	.	.	.
	Greenhouse-Geisser	,000	,000	.	.	.
	Huynh-Feldt	,000	,000	.	.	.
	Lower-bound	,000	,000	.	.	.
500Hz	Sphericity Assumed	,000	0	.	.	.
	Greenhouse-Geisser	,000	,000	.	.	.
	Huynh-Feldt	,000	,000	.	.	.
	Lower-bound	,000	,000	.	.	.
1kHz	Sphericity Assumed	,000	0	.	.	.
	Greenhouse-Geisser	,000	,000	.	.	.
	Huynh-Feldt	,000	,000	.	.	.
	Lower-bound	,000	,000	.	.	.
1,5kHz	Sphericity Assumed	,000	0	.	.	.
	Greenhouse-Geisser	,000	,000	.	.	.
	Huynh-Feldt	,000	,000	.	.	.
	Lower-bound	,000	,000	.	.	.
2kHz	Sphericity Assumed	,000	0	.	.	.
	Greenhouse-Geisser	,000	,000	.	.	.
	Huynh-Feldt	,000	,000	.	.	.
	Lower-bound	,000	,000	.	.	.
3kHz	Sphericity Assumed	,000	0	.	.	.
	Greenhouse-Geisser	,000	,000	.	.	.
	Huynh-Feldt	,000	,000	.	.	.

		Lower-bound	,000	,000	.	.	.
	4kHz	Sphericity Assumed	,000	0	.	.	.
		Greenhouse-Geisser	,000	,000	.	.	.
		Huynh-Feldt	,000	,000	.	.	.
		Lower-bound	,000	,000	.	.	.
	6kHz	Sphericity Assumed	,000	0	.	.	.
		Greenhouse-Geisser	,000	,000	.	.	.
		Huynh-Feldt	,000	,000	.	.	.
		Lower-bound	,000	,000	.	.	.
	8kHz	Sphericity Assumed	,000	0	.	.	.
		Greenhouse-Geisser	,000	,000	.	.	.
		Huynh-Feldt	,000	,000	.	.	.
		Lower-bound	,000	,000	.	.	.
Error(measure)	125Hz	Sphericity Assumed	12,500	4	3,125		
		Greenhouse-Geisser	12,500	4,000	3,125		
		Huynh-Feldt	12,500	4,000	3,125		
		Lower-bound	12,500	4,000	3,125		
	250Hz	Sphericity Assumed	15,000	4	3,750		
		Greenhouse-Geisser	15,000	4,000	3,750		
		Huynh-Feldt	15,000	4,000	3,750		
		Lower-bound	15,000	4,000	3,750		
	500Hz	Sphericity Assumed	110,000	4	27,500		
		Greenhouse-Geisser	110,000	4,000	27,500		
		Huynh-Feldt	110,000	4,000	27,500		
		Lower-bound	110,000	4,000	27,500		
	1kHz	Sphericity Assumed	85,000	4	21,250		
		Greenhouse-Geisser	85,000	4,000	21,250		
		Huynh-Feldt	85,000	4,000	21,250		
		Lower-bound	85,000	4,000	21,250		
	1,5kHz	Sphericity Assumed	87,500	4	21,875		
		Greenhouse-Geisser	87,500	4,000	21,875		
		Huynh-Feldt	87,500	4,000	21,875		
		Lower-bound	87,500	4,000	21,875		
	2kHz	Sphericity Assumed	40,000	4	10,000		
		Greenhouse-Geisser	40,000	4,000	10,000		
		Huynh-Feldt	40,000	4,000	10,000		
		Lower-bound	40,000	4,000	10,000		
	3kHz	Sphericity Assumed	935,000	4	233,750		

		Greenhouse-Geisser	935,000	4,000	233,750		
		Huynh-Feldt	935,000	4,000	233,750		
		Lower-bound	935,000	4,000	233,750		
	4kHz	Sphericity Assumed	65,000	4	16,250		
		Greenhouse-Geisser	65,000	4,000	16,250		
		Huynh-Feldt	65,000	4,000	16,250		
		Lower-bound	65,000	4,000	16,250		
	6kHz	Sphericity Assumed	140,000	4	35,000		
		Greenhouse-Geisser	140,000	4,000	35,000		
		Huynh-Feldt	140,000	4,000	35,000		
		Lower-bound	140,000	4,000	35,000		
	8kHz	Sphericity Assumed	135,000	4	33,750		
		Greenhouse-Geisser	135,000	4,000	33,750		
		Huynh-Feldt	135,000	4,000	33,750		
		Lower-bound	135,000	4,000	33,750		

Intervention group

7.6.2 Univariate testing of the control group

Univariate Tests(a)

Source	Measure		Type III Sum of Squares	df	Mean Square	F	Sig.
measure	125Hz	Sphericity Assumed	140,625	1	140,625	45,000	,003
		Greenhouse-Geisser	140,625	1,000	140,625	45,000	,003
		Huynh-Feldt	140,625	1,000	140,625	45,000	,003
		Lower-bound	140,625	1,000	140,625	45,000	,003
	250Hz	Sphericity Assumed	10,000	1	10,000	2,667	,178
		Greenhouse-Geisser	10,000	1,000	10,000	2,667	,178
		Huynh-Feldt	10,000	1,000	10,000	2,667	,178
		Lower-bound	10,000	1,000	10,000	2,667	,178
	500Hz	Sphericity Assumed	40,000	1	40,000	1,455	,294
		Greenhouse-Geisser	40,000	1,000	40,000	1,455	,294
		Huynh-Feldt	40,000	1,000	40,000	1,455	,294
		Lower-bound	40,000	1,000	40,000	1,455	,294
	1kHz	Sphericity Assumed	90,000	1	90,000	4,235	,109
		Greenhouse-Geisser	90,000	1,000	90,000	4,235	,109
		Huynh-Feldt	90,000	1,000	90,000	4,235	,109
		Lower-bound	90,000	1,000	90,000	4,235	,109
	1,5kHz	Sphericity Assumed	15,625	1	15,625	,714	,446
		Greenhouse-Geisser	15,625	1,000	15,625	,714	,446
		Huynh-Feldt	15,625	1,000	15,625	,714	,446
		Lower-bound	15,625	1,000	15,625	,714	,446
	2kHz	Sphericity Assumed	22,500	1	22,500	2,250	,208
		Greenhouse-Geisser	22,500	1,000	22,500	2,250	,208
		Huynh-Feldt	22,500	1,000	22,500	2,250	,208
		Lower-bound	22,500	1,000	22,500	2,250	,208
	3kHz	Sphericity Assumed	490,000	1	490,000	2,096	,221
		Greenhouse-Geisser	490,000	1,000	490,000	2,096	,221
		Huynh-Feldt	490,000	1,000	490,000	2,096	,221
		Lower-bound	490,000	1,000	490,000	2,096	,221
	4kHz	Sphericity Assumed	22,500	1	22,500	1,385	,305
		Greenhouse-Geisser	22,500	1,000	22,500	1,385	,305
		Huynh-Feldt	22,500	1,000	22,500	1,385	,305
		Lower-bound	22,500	1,000	22,500	1,385	,305
	6kHz	Sphericity Assumed	122,500	1	122,500	3,500	,135

		Greenhouse-Geisser	122,500	1,000	122,500	3,500	,135
		Huynh-Feldt	122,500	1,000	122,500	3,500	,135
		Lower-bound	122,500	1,000	122,500	3,500	,135
	8kHz	Sphericity Assumed	90,000	1	90,000	2,667	,178
		Greenhouse-Geisser	90,000	1,000	90,000	2,667	,178
		Huynh-Feldt	90,000	1,000	90,000	2,667	,178
		Lower-bound	90,000	1,000	90,000	2,667	,178
measure * gruppe	125Hz	Sphericity Assumed	,000	0	.	.	.
		Greenhouse-Geisser	,000	,000	.	.	.
		Huynh-Feldt	,000	,000	.	.	.
		Lower-bound	,000	,000	.	.	.
	250Hz	Sphericity Assumed	,000	0	.	.	.
		Greenhouse-Geisser	,000	,000	.	.	.
		Huynh-Feldt	,000	,000	.	.	.
		Lower-bound	,000	,000	.	.	.
	500Hz	Sphericity Assumed	,000	0	.	.	.
		Greenhouse-Geisser	,000	,000	.	.	.
		Huynh-Feldt	,000	,000	.	.	.
		Lower-bound	,000	,000	.	.	.
	1kHz	Sphericity Assumed	,000	0	.	.	.
		Greenhouse-Geisser	,000	,000	.	.	.
		Huynh-Feldt	,000	,000	.	.	.
		Lower-bound	,000	,000	.	.	.
	1,5kHz	Sphericity Assumed	,000	0	.	.	.
		Greenhouse-Geisser	,000	,000	.	.	.
		Huynh-Feldt	,000	,000	.	.	.
		Lower-bound	,000	,000	.	.	.
	2kHz	Sphericity Assumed	,000	0	.	.	.
		Greenhouse-Geisser	,000	,000	.	.	.
		Huynh-Feldt	,000	,000	.	.	.
		Lower-bound	,000	,000	.	.	.
	3kHz	Sphericity Assumed	,000	0	.	.	.
		Greenhouse-Geisser	,000	,000	.	.	.
		Huynh-Feldt	,000	,000	.	.	.
		Lower-bound	,000	,000	.	.	.
	4kHz	Sphericity Assumed	,000	0	.	.	.

		Greenhouse-Geisser	,000	,000	.	.	.
		Huynh-Feldt	,000	,000	.	.	.
		Lower-bound	,000	,000	.	.	.
	6kHz	Sphericity Assumed	,000	0	.	.	.
		Greenhouse-Geisser	,000	,000	.	.	.
		Huynh-Feldt	,000	,000	.	.	.
		Lower-bound	,000	,000	.	.	.
	8kHz	Sphericity Assumed	,000	0	.	.	.
		Greenhouse-Geisser	,000	,000	.	.	.
		Huynh-Feldt	,000	,000	.	.	.
		Lower-bound	,000	,000	.	.	.
Error(measure)	125Hz	Sphericity Assumed	12,500	4	3,125		
		Greenhouse-Geisser	12,500	4,000	3,125		
		Huynh-Feldt	12,500	4,000	3,125		
		Lower-bound	12,500	4,000	3,125		
	250Hz	Sphericity Assumed	15,000	4	3,750		
		Greenhouse-Geisser	15,000	4,000	3,750		
		Huynh-Feldt	15,000	4,000	3,750		
		Lower-bound	15,000	4,000	3,750		
	500Hz	Sphericity Assumed	110,000	4	27,500		
		Greenhouse-Geisser	110,000	4,000	27,500		
		Huynh-Feldt	110,000	4,000	27,500		
		Lower-bound	110,000	4,000	27,500		
	1kHz	Sphericity Assumed	85,000	4	21,250		
		Greenhouse-Geisser	85,000	4,000	21,250		
		Huynh-Feldt	85,000	4,000	21,250		
		Lower-bound	85,000	4,000	21,250		
	1,5kHz	Sphericity Assumed	87,500	4	21,875		
		Greenhouse-Geisser	87,500	4,000	21,875		
		Huynh-Feldt	87,500	4,000	21,875		
		Lower-bound	87,500	4,000	21,875		
	2kHz	Sphericity Assumed	40,000	4	10,000		
		Greenhouse-Geisser	40,000	4,000	10,000		
		Huynh-Feldt	40,000	4,000	10,000		
		Lower-bound	40,000	4,000	10,000		
	3kHz	Sphericity Assumed	935,000	4	233,750		
		Greenhouse-Geisser	935,000	4,000	233,750		

		Huynh-Feldt	935,000	4,000	233,750		
		Lower-bound	935,000	4,000	233,750		
	4kHz	Sphericity Assumed	65,000	4	16,250		
		Greenhouse-Geisser	65,000	4,000	16,250		
		Huynh-Feldt	65,000	4,000	16,250		
		Lower-bound	65,000	4,000	16,250		
	6kHz	Sphericity Assumed	140,000	4	35,000		
		Greenhouse-Geisser	140,000	4,000	35,000		
		Huynh-Feldt	140,000	4,000	35,000		
		Lower-bound	140,000	4,000	35,000		
	8kHz	Sphericity Assumed	135,000	4	33,750		
		Greenhouse-Geisser	135,000	4,000	33,750		
		Huynh-Feldt	135,000	4,000	33,750		
		Lower-bound	135,000	4,000	33,750		

Control group

7.6.3 Analysis of variance of the initial values

		Sum of Squares	df	Mean Square	F	Sig.
125Hzv	Between Groups	32,531	1	32,531	,086	,773
	Within Groups	4896,875	13	376,683		
	Total	4929,406	14			
250Hzv	Between Groups	3,333	1	3,333	,008	,932
	Within Groups	5770,000	13	443,846		
	Total	5773,333	14			
500Hzv	Between Groups	3,333	1	3,333	,007	,934
	Within Groups	6120,000	13	470,769		
	Total	6123,333	14			
1kHzv	Between Groups	440,833	1	440,833	1,023	,330
	Within Groups	5602,500	13	430,962		
	Total	6043,333	14			
1,5kHzv	Between Groups	240,833	1	240,833	,470	,505
	Within Groups	6665,000	13	512,692		
	Total	6905,833	14			
2kHzv	Between Groups	800,833	1	800,833	1,319	,271
	Within Groups	7892,500	13	607,115		
	Total	8693,333	14			
3kHzv	Between Groups	300,833	1	300,833	,549	,472
	Within Groups	7122,500	13	547,885		
	Total	7423,333	14			
4kHzv	Between Groups	40,833	1	40,833	,071	,794
	Within Groups	7442,500	13	572,500		
	Total	7483,333	14			
6kHzv	Between Groups	300,833	1	300,833	,481	,500
	Within Groups	8132,500	13	625,577		
	Total	8433,333	14			
8kHzv	Between Groups	67,500	1	67,500	,107	,748
	Within Groups	8172,500	13	628,654		
	Total	8240,000	14			

7.6.4 Analysis of variance of the final values

		Sum of Squares	df	Mean Square	F	Sig.
125Hzn	Between Groups	31,130	1	31,130	,117	,737
	Within Groups	3447,222	13	265,171		
	Total	3478,353	14			
250Hzn	Between Groups	53,333	1	53,333	,121	,734
	Within Groups	5730,000	13	440,769		
	Total	5783,333	14			
500Hzn	Between Groups	30,000	1	30,000	,084	,777
	Within Groups	4670,000	13	359,231		
	Total	4700,000	14			
1kHzn	Between Groups	607,500	1	607,500	1,601	,228
	Within Groups	4932,500	13	379,423		
	Total	5540,000	14			
1,5kHzn	Between Groups	1020,833	1	1020,833	1,920	,189
	Within Groups	6912,500	13	531,731		
	Total	7933,333	14			
2kHzn	Between Groups	1020,833	1	1020,833	1,790	,204
	Within Groups	7412,500	13	570,192		
	Total	8433,333	14			
3kHzn	Between Groups	7,500	1	7,500	,013	,909
	Within Groups	7232,500	13	556,346		
	Total	7240,000	14			
4kHzn	Between Groups	83,333	1	83,333	,158	,697
	Within Groups	6840,000	13	526,154		
	Total	6923,333	14			
6kHzn	Between Groups	7,500	1	7,500	,013	,912
	Within Groups	7742,500	13	595,577		
	Total	7750,000	14			
8kHzn	Between Groups	3,333	1	3,333	,006	,941
	Within Groups	7670,000	13	590,000		
	Total	7673,333	14			

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