

Chapter 60

Ménière's Disease and Tinnitus

Michel Paolino and Vénéra Ghulyan-Bedikian

Keypoints

1. Ménière's disease is a clinical syndrome that comprises vertigo, sensorineural hearing loss, subjective tinnitus, and aural fullness.
2. The tinnitus is classically low pitched and evolves with the progression of the disease.
3. The diagnosis of Ménière's disease is based on patient history, a clinical examination, a complete oto-neurological assessment, and a MRI.
4. The differential diagnosis distinguishes Ménière's disease from vestibular schwannoma, microvascular conflict of the VIII cranial nerve, and migraines.
5. Antivertiginous, antihistamines, loop diuretics, antiemetics, and benzodiazepines are effective in managing the acute attacks.
6. The following therapies are recommended for inter-crisis periods:
 - (a) Dietetic recommendations including low-salt diet
 - (b) Medication (antihistaminics, diuretics, osmo-regulators, vasodilators, antiemetics, corticoids, benzodiazepines)
 - (c) Relaxation therapy, tinnitus retraining therapy, sound therapy, etc.
 - (d) Coordinated treatment of temporomandibulars and cervical disorders
 - (e) Intratympanic therapy with gentamycin or steroids
 - (f) Surgical approach by endolymphatic mastoid shunt or endolymphatic sac decompression, vestibular neurectomy, and labyrinthectomy.
7. Some medications as well as transtympanic therapy seem particularly interesting, because they provide improvements of both vertigo and tinnitus while preserving the hearing in the majority of patients who have this treatment.
8. Conservative treatments should be exhausted and surgery reserved for patients with disabling and refractory vertigo, but surgery cannot prevent the progression of the tinnitus.

Keywords Ménière's disease • Tinnitus • Vertigo • Hearing loss • Endolymphatic hydrops • Psychosomatic incidence

Abbreviations

AAO-HNS	American Academy of Otolaryngology – Head and Neck Surgery
ABR	Auditory brainstem response
BPV	Benign paroxysmal positional vertigo
CBT	Cognitive-behavioral therapy
DPOAEs	Distortion product otoacoustic emissions
ECoG	Electrocochleography
MRI	Magnetic resonance imaging
SP	Summating potential

Ménière's Disease: Pathogenesis, Symptoms, and Clinical Manifestations

Ménière's disease represents one of the many causes of tinnitus. Since its first description in 1861, several etiological theories have been proposed to explain the pathogenesis of this disease: endolymphatic hydrops, autoimmune disorders, viral infections, allergic processes,

M. Paolino (✉)
Department of Oto-Neurology, IMERTA,
C.H.P. Clairval, Marseille, France
e-mail: michel.paolino@wanadoo.fr

and activation of neural plasticity. Ménière's disease is a syndrome that comprises of three (or four) symptoms:

- Recurrent episodes of spontaneous vertigo lasting from several minutes to a few hours, which can be followed by residual unsteadiness.
- Fluctuating and slowly progressive sensorineural hearing loss, usually unilateral and initially prevailing at low frequencies.
- Subjective tinnitus and sensation of aural fullness, pressure, or discomfort. The tinnitus is referred to the affected ear and described as a low-frequency “buzz” or “roar,” but as the disease progresses, it sometimes includes a high-pitched component.

Ménière's disease usually starts in only one ear but can evolve into a bilateral form. It is characterized by periods of exacerbation and remission. The crises are frequently severe, incapacitating, unpredictable, and usually accompanied by anxiety, headaches, and autonomic manifestations (nausea, vomiting, diaphoresis, pallor, tachycardia, diarrhea, etc.).

At the beginning, the tinnitus complaint is secondary. Classically, it becomes worse during vertigo attacks but may significantly improve or even disappear afterward. However, with the progression of the disease, the tinnitus can become permanent, persisting between attacks. Its evolution is then unfavorable due to a significant increase in anxiety.

Besides the above-described classic form, some physicians have also included patients with incomplete clinical features as forms of Ménière's disease. In fact, the frequency and the intensity of the crises, the association of symptoms, and their impact on the patient's quality of life can vary from one patient to another. These various clinical forms can be classified into three groups:

- Predominantly cochlear forms where hearing loss and tinnitus are “in the foreground” and the vertigo is either absent or atypical
- Predominantly vestibular forms with typical vertigo crises, which are not necessarily preceded by tinnitus or aural fullness, and the hearing loss does not always affect the low frequencies
- Separate forms with initially typical vertigo crises without cochlear signs.

These symptoms that have similarities with the vertiginous crises of migraine should be distinguished from those of Ménière's disease. Later, the cochlear symptoms usually occur together with a pattern of

vestibular signs. Signs of endolymphatic hydrops sometimes are later added by vestibular symptoms, and the disease evolves into a typical Ménière's disease.

Diagnostic Criteria

The diagnosis is established with patient history, a clinical examination, a complete oto-neurological assessment, and possibly a MRI of the brain.

Patient History

Patient history is very important for correct diagnosis of Ménière's disease because it can provide information about symptoms during acute attacks. Clinical testing is usually only done between acute attacks. On completing the initial history, the onset, duration, frequency and intensity of crises, the association of symptoms, and their impact on the patient's quality of life should be determined. A typical vertigo attack with no associated hearing loss suggests that the disease is possibly Ménière's disease. A single definitive episode of vertigo that occurs together with the other symptoms of Ménière's disease makes the diagnosis probable but an exact diagnosis of Ménière's disease requires two or more definitive episodes of vertigo and hearing loss associated with tinnitus and/or aural fullness [1].

Clinical Examination

Clinical testing is usually only done between acute attacks and can be normal at the earlier stages of the disease. As the disease progresses, it reveals audio-vestibular abnormalities.

The Romberg test¹ shows axial deviation while the Babinski–Weil test² and the Fukuda stepping

¹Romberg test: The patient stands with feet together, eyes open, and hands by the sides. The patient closes their eyes while the examiner observes for a full minute to note occurrence of a fall or axial deviation toward the affected side.

²Babinski–Weil test: the patient walks with eyes closed, ten steps forward and ten steps backward several times; the examiner looks for a deviation from the straight path, bending to the affected side when walking forward and to the other when walking backward.

test³ show drift toward the affected side. However, these tests are not always reliable when the patient has myo-articular and/or orthopedic problems.

Audiometric and Oto-Neurologic Examination

Pure-tone audiometry should be obtained and should show low-frequency sensorineural hearing loss that gets better or disappears after crises. However, as the disease progresses, the hearing loss often reaches high frequencies and can even change to a flat hearing loss.

Speech audiometry should show normal speech intelligibility and can confirm that the hearing loss is indeed of cochlear origin. Tympanometry can rule out middle-ear problems. The Weber-test⁴ should be lateralized toward the healthy or better ear. The Rinne test⁵ should be positive. In patients with Ménière's disease, it usually does not indicate any difference between the auditory thresholds for air and bone conductance.

³Fukuda stepping test: The patient stands with eyes closed, arms outstretched and wearing ear muffs. The patient marches in place 50 steps at the pace of a brisk walk while keeping the eyes closed. The observer looks for any rotation. Rotation of 30° or more is considered a positive test.

⁴Weber test: A test in which the stem of a vibrating tuning fork is placed on the midline of the head to ascertain which ear the sound is heard by bone conduction. The sound will be perceived in the affected ear when a unilateral conductive hearing loss is present or in the unaffected ear when there is a unilateral sensorineural hearing loss. The result of this test is combined with the result of the Rinne test to interpretation of the type of hearing loss. (From Stedman' Electronic Medical Dictionary).

⁵Rinne test: Tests the ability to hear by air conduction with the ability to hear by bone conduction. By placing the tines of a vibrating tuning fork near the pinna, the acoustic stimulus is presented by air conduction; by placing the stem of a vibrating tuning fork on the mastoid process, the acoustic stimulus is presented by bone conduction. In conductive hearing losses, the stimulus is heard louder and longer by bone conduction. In sensorineural hearing losses, the stimulus is heard louder and longer by air conduction. The result of the test is reported for each ear as air conduction and is found to be greater than bone conduction, or vice versa. This information is combined with the result of the Weber tuning fork test in interpreting the type of hearing loss.

Metz-test⁶ shows objective recruitment that is more marked in Ménière's disease compared to the other cochlear pathologies.

The Reflex Decay Test shows that the stapedius reflex is well maintained and can confirm the cochlear origin of the disease.

If the patient accepts, a Glycerol test⁷ may be helpful if the patient's history and tests are inconclusive. Hearing thresholds, particularly at low frequencies, often improves after administration of glycerol to patients with Ménière's disease.

Recording of distortion product otoacoustic emissions (DPOAEs) provides information about the function of the outer hair cells which may be impaired by abnormal pressure (or rather volume [2]) of the endolymphatic fluid. The combination of vestibular-evoked myogenic potentials and DPOAEs with the glycerol test is suggested for early diagnosis of Ménière's disease and for the differential diagnosis in patients presenting a first attack of vertigo with or without hearing loss [3].

Studies have disagreed regarding the value of recording DPOAEs and cochlear microphonics for differential diagnosis in patients with and without hydrops [4]. The auditory brainstem response (ABR) typically does not show abnormalities in agreement with the assumption that the disease is not affecting retrocochlear functions. The diagnostic value of recordings of the summing potential (SP) that is a component of the electrocochleogram (ECoG) has been advocated by some investigators [5, 6] while others, Eggermont 1979, [7] have been critical regarding the value of ECoG in diagnosis of Ménière's disease, in particular for hearing loss less than 50 dB. The large individual variation in the SP is an obstacle in its use as a diagnostic criterion.

⁶Metz test: The test compares the threshold of the acoustic middle-ear reflex and loudness perception.

⁷Glycerol test: Glycerol is administered orally 1.5 ml/kg of body weight dissolved in the equal amount of the physiological saline. A positive result is defined as a threshold improvement of the audiogram of 10 dB or more in at least three frequencies (500, 1,000, or 2,000 Hz). The speech audiometry must show an improvement of 10% of the discrimination. However, the oral glycerol test prohibits food intake before the testing, requires a long examination time, and is associated with side effects such as headache, nausea, and vomiting. The intravenous glycerol test (intravenous injection of 100 ml glycerol (10%) over 30 min) is known to have no such disadvantages.

Vestibular Examination

Nystagmography⁸ can quantify the vestibular abnormalities. Spontaneous horizontal or horizontal-rotatory nystagmus is common in individuals with Ménière's disease. As soon as the crisis starts, nystagmus beating toward the affected side occurs. But the direction of the eye movements is quickly reversed, confirming a unilateral vestibular deficit. The nystagmus is then very intense and occurs even when the eyes are directed toward the affected side (grade III⁹) nystagmus). Typically it diminishes with time. After the crisis, the nystagmus which is sensitized by the suppression of ocular fixation, disappear from direct observation, but can always be reactivated by the Head shaking test¹⁰ or by vibratory stimulation of the mastoid. If high-frequency vibratory stimulation of the mastoid reveals a latent nystagmus, it is an indication of a unilateral vestibular deficit. In patients with Ménière's disease who have a normal caloric test, the direction of the nystagmus triggered by the head shaking and vibratory tests is usually discordant and informs us on the evolution of the disease.

The Dix Hallpike test¹¹ is typically negative in Ménière's disease. However, detailed attention should

be paid to the realization of this test because the spontaneous or latent nystagmus due to the Ménière's disease can be sensitized or revealed in the decubitus position. In addition, the possibility of benign paroxysmal positional vertigo (BPPV) should not be overlooked. BPPV can be either idiopathic or, due to the mechanical disruption and distortion of the utricle and saccule, related to the progression of Ménière's disease.

Magnetic Resonance Imaging

A MRI of the brain with Gadolinium contrast can determine whether the internal auditory canals are open and that the morphology of structures in the posterior fossa is normal.

Differential Diagnosis

An expert clinical judgment is required to distinguish between true Ménière's disease and several other conditions characterized by vertigo, hearing loss, and tinnitus, such as cochlear otosclerosis; bacterial or viral labyrinthitis; temporal bone trauma; V, VII, and VIII neuroma; meningioma; cholesteatoma; and migraine. The complete oto-neurological assessment including an ABR, ECoG, and MRI of the posterior fossa makes it possible to differentiate Ménière's disease from these etiologies.

Distinguishing Ménière's disease from vestibular schwannoma and vascular conflict of the VIII cranial nerve are the most important factors. Kentala and Pyykkö (2000) [8] compared test results from 128 individuals with vestibular schwannoma and 243 with Ménière's disease and found that 38% of patients with small and medium-sized vestibular schwannoma had an association of all the symptoms of a typical Ménière's disease. In 69% of the patients, the attacks lasted from 5 min to 4 h and occurred only once or twice a year. In addition, half of the patients had spontaneous nystagmus and 61% of the patients had caloric asymmetry. Tinnitus in these patients was either mild or intense (in 49 and 12% of cases, respectively).

Vascular conflict of the VIII nerve is characterized by intermittent paroxysms of dizziness and unilateral tinnitus, which can become more frequent over time (see also Chap.40). In a chronic stage, this condition

⁸Nystagmography: The technique of recording nystagmus. Frenzel glasses or videonystagmography mask should be used to eliminate the visual fixation that may suppress the nystagmus.

⁹Grade III nystagmus: Spontaneous nystagmus that occurs when the eyes are directed to the center, right, and left. The intensity of this nystagmus increases when the gaze direction is the same at the quick phase of the nystagmus.

¹⁰Head shaking test: The patient is positioned upright and instrumented with a videonystagmography mask that suppresses the visual fixation and records the eye movements. The examiner grasps the patient's head and moves it briskly approximately 30° to either side in the horizontal plane around the vertical axis. The head shaking for a frequency of about 2 Hz is continued for 20 cycles and then abruptly stopped. A head shaking nystagmus indicates a dynamic imbalance between the ears. It is usually beating toward the "better" ear during about 30 s and can be followed by a second phase of nystagmus that is weaker, decays more slowly, and is directed toward the "bad" ear. The main value of seeing a secondary phase is that one can clearly identify the primary phase, which is sometimes very short.

¹¹Dix Hallpike test: test for eliciting paroxysmal vertigo and nystagmus in which the patient is brought from the sitting to the supine position with the head hanging over the examining table and turned to the right or left (45°); vertigo and nystagmus are elicited when the head is rotated toward the affected ear. Frenzel glasses or videonystagmography mask are used to eliminate the visual fixation that may suppress the nystagmus.

induces persistent unsteadiness [9, 10] often associated with constant tinnitus. Unfortunately, this condition, which has a similar course as Ménière's disease, is often not recognized. In a study by Ryu and coworkers, it was even shown that up to 73% of the patients diagnosed preoperatively as having Ménière's disease were successfully treated for vascular conflict of the vestibular nerve [11].

Patients with Ménière's disease have normal ABR with normal interpeak latency I–III [12].

Ménière's disease also has similarities with migraine (see a recent review by Minor, 2004 [13] and Chap. 38). However, the glycerol test is negative for migraine, the crises of dizziness always start in the early morning, and there is often a family or personal history of migraines. In addition, individuals with migraine-associated dizziness usually have normal hearing, and when a sensorineural hearing loss is present, it rarely progresses, thus, different from individuals with Ménière's disease [14] (see also Chap. 38).

Treatment

There are several different treatments available for Ménière's disease, and the choice of treatment requires careful consideration. Both medical and surgical treatments are in general use, but there is not a consensus regarding the specific treatment and a divergence of different protocols currently in use.

Management of Acute Attacks

During an attack, the treatment is aimed at alleviating the acute symptoms.

Vertigo is often the most disabling symptom of Ménière's disease, and the medical treatment seeks, above all, to control these symptoms. The intravenous injections of *Acetyllecine* used in some countries are effective in alleviating vertigo attacks. The action of *Acetyllecine* is not understood, but studies in animal models suggest that it acts mainly on abnormally hyperpolarized and/or depolarized vestibular neurons by restoring their membrane potential [15]. Administration of *Acetyllecine* does not have any proven effect on tinnitus.

A randomized double-blind clinical study showed that *betahistine dimesylate* 12 mg as well as a fixed combination of *cinnarizine* 20 mg and *dimenhydrinate* 40 mg are highly effective and safe treatment options for Ménière's disease and may be used in both the management of acute episodes and long-term treatment. These drugs, commonly used to treat vestibular disorders, reduce tinnitus in approximately 60% of patients with Ménière's disease [16].

Some physicians prescribe loop diuretics to normalize the balance of fluid volumes in the inner ear. For example, intravenous injections of 40 mg *furosemide* in the morning during 3–5 days are effective treatments but require checking the blood electrolytes. Tinnitus should be watched since furosemide can give tinnitus.

During crises, intravenous injections of 40 ml of 30% *Glucosé-hyper* in the morning and evening for 3 days are also effective treatments. Corticosteroids (such as *Methylprednisolon* 20 mg in intravenous perfusion) are used by some physicians for the management of acute vertigo attacks. Treatment for preventing nausea and vomiting, which can be very intense during a crisis, should also be available. The following antiemetics are often prescribed:

Compazine (per os or suppository) – 5 mg every 12 h as needed.

Meclizine (per os) – dose ranges from 12.5 mg twice a day to 50 mg three times a day.

Métopimazin (per os) – 1 or 2 (15 mg) tablets three times a day.

Métoclopramid – intramuscular or intravenous injections of a 10 mg/2 ml vial three times a day.

Benzodiazepines (for example: *Lorazepam* sublingual tablets, 0.5 mg twice a day) are used to relieve the anxiety accompanying Ménière's attacks.

Therapies for Inter-crisis Periods

The purpose of treatment is to reduce the number of attacks while trying to prevent further hearing loss and damage to the vestibular system. This form of treatment depends on the inter-crisis symptoms, their intensity, and their impact on the patients' quality of life.

Non-invasive Therapies

If the symptoms disappear after the crisis, the patient only needs dietetic recommendations such as to avoid caffeine, alcohol, tobacco, and aspartame, which worsen tinnitus and other Ménière's disease symptoms. A low-salt diet is also important.

To reduce the frequency of vertigo attacks and alleviate the inter-crisis symptoms, *Betahistine* (16 mg three times a day or 24 mg twice a day) is often beneficial [17]. Diuretics (such as *furosemide* 20 mg a day, two times a week with a control of electrolytes), osmoregulators (such as *glycerol* or *mannitol*), or vasodilators (for example, *Buflomedil* 150 mg twice a day) can also be effective in selected patients. However, there is insufficient evidence that this medication has any significant effect on Ménière's disease-related tinnitus [18, 19].

A randomized and controlled clinical study showed the effectiveness of the combination of *diphenidol* (25 mg/d), *acetazolamide* (250 mg/48 h), and *prednisone* (0.35 mg/kg) on the tinnitus, as well as the frequency and duration of vertigo [20].

Corticosteroids are especially helpful in bilateral forms, in particular, if an autoimmune cause is suspected. Desensitizing therapies for allergies have been shown to be effective to relieve the Ménière's disease symptoms, including tinnitus in some patients [21, 22].

Depending on the presence of psychosomatic components, the social–professional impact of tinnitus, and other Ménière's disease symptoms, a patient's regular follow-up by a multidisciplinary team may be beneficial. Relaxation therapy may be beneficial in some patients because of its beneficial effects on unsteadiness, as well as on tinnitus. It should be associated with standard methods of tinnitus management (see Part V – Management of Tinnitus). Balance rehabilitation can improve a patient's balance.

Intratympanic Therapy

When vertigo persists despite optimal medical management, an intratympanic therapy with gentamycin or steroids may be proposed to control the vertigo. The intratympanic administration of low-dose gentamycin provides long-term vertigo control, whilst preserving

hearing and vestibular function in the majority of patients [23]. In addition, it is effective to treat the tinnitus in Ménière's disease [24, 25]. New protocols have been developed to reduce the risk of permanent gentamycin ototoxicity. The one-shot injection protocols present a minimal risk to hearing, whereas repeated or continuous application protocols result in higher gentamycin doses in the cochlea and can cause damage to hearing [2, 26]. In a review of literature, Dodson and Sismanis (2004) [27] suggest that this therapy should mainly be proposed to patients with Ménière's disease who do not have useful hearing. The authors recommend intratympanic therapies with steroids for Ménière's patients with normal hearing, which have some success in controlling vertigo.

Treatments that can control vertigo may not always improve tinnitus in Ménière's patients. A prospective double-blind placebo-controlled trial by Garduno-Anaya et al. (2005) [28] showed relief of tinnitus in 48% of the patients who were treated with intratympanic dexamethasone. It was also shown that the inner-ear perfusion via transtympanic delivery of dexamethasone 4 mg/ml improves hearing, tinnitus, and aural pressure in patients with a cochlear form of Ménière's disease [29]. Nonetheless, Araujo et al. (2005) [30] reported that a prospective randomized placebo-controlled but single-blind trial showed that intratympanic dexamethasone had no significant effect on severe tinnitus compared to placebo.

Surgical Approach

Surgical treatment should be a last resort and is reserved for Ménière's patients who are refractory to medical therapy. Conservative and destructive surgical procedures are used according to the severity of the crises, the degree of serviceable hearing, and the condition of the contralateral ear.

Endolymphatic Sac Surgery

Conservative surgery by endolymphatic mastoid shunt or endolymphatic sac decompression without sac incision is the operation most often practiced. It can lead

to a temporary decrease in vertigo occurrence and intensity, while generally preserving hearing [31]. However, the literature reveals disagreement regarding the effectiveness of this approach in reducing vertigo.

Sectioning of the Vestibular Nerve

Sectioning of the vestibular nerve is effective in controlling vertigo while preserving hearing in most patients. Thus, it is available for patients with serviceable hearing who have failed all other treatments and are especially incapacitated by Ménière's disease. Dandy (1941) [32], described how he treated patients with Ménière's disease by sectioning the eighth cranial nerve. Later, this technique has been refined and now, most typically, only the vestibular nerve is sectioned. Different techniques are in use for sectioning the vestibular nerve, such a retromastoid (retrolabyrinthine) approach to the cerebello pontine angle, and a middle fossa approach has been used as well. Endoscope-assisted, minimally invasive retrosigmoid approach that is now recommended rather than the middle fossa or retrolabyrinthine approaches is simpler, more reliable, and has lower risk of complications [33–35]. Analysis of 18 publications mentioning tinnitus status after vestibular neurectomy in a total of 1,318 patients shows that the tinnitus had worsened after the operation from 0 to 60%, but most of the patients had no change in their tinnitus (17–72%) and others even reported improvements of 6–61% [36]. Thus, vestibular neurectomy does not consistently worsen tinnitus, but the risk is present.

For patients with unilateral Ménière's disease and total deafness, labyrinthectomy can be undertaken as a last resort.

The procedures that control the episodic vertigo by destroying the vestibular function in the affected ear should be reserved for patients who have handicapping vertigo, which persists in spite of conservative treatments. Typically, the balance improves significantly after these procedures, thanks to compensatory and substitutive mechanisms. The ability to compensate for loss of vestibular input decreases with age, and for people over the age of 50 years, the compensation takes a long time and is rarely complete. These operations, however, cannot prevent the progression of hyperacusis or tinnitus.

Microvascular Decompression

Cranial nerve roots in contact with a blood vessel have been associated with specific diseases such as hemifacial spasm, trigeminal neuralgia, glossopharyngeal neuralgia, and geniculate neuralgia. Also, blood vessels in close contact with the root of the vestibular nerve have been associated with a specific disorder such as a specific vestibular disorder [disabling positional vertigo (DPV)] [37, 38], and blood vessels in contact with the auditory nerve have been associated with some special forms of tinnitus [10]. Microvascular decompression operations (MVD) for DPV have shown beneficial effect in about 85% of patients [39] (see Chap. 40). MVD operations for tinnitus are effective in giving relief of tinnitus in some patients with this condition [10] (see Chap. 84).

The fact that vascular loops have been reported to be in contact with the vestibular nerve in patients with Ménière's disease does not mean that contact with a blood vessel is associated with symptoms. Studies have shown that vascular loops in contact with cranial nerve roots occur frequently without giving specific symptoms from the respective cranial nerve [40] (see Chap. 40).

Other Forms of Treatment

Applying Air Puffs to the Inner Ear

It has been shown that applying air pressure to the inner ear can relieve some of the symptoms of Ménière's disease [41, 42]. This was first realized by placing individuals with Ménière's disease in a pressure chamber. These findings have later been explored, and a practical device that a person can wear was developed (the Meniett, now marketed by Medtronic, Inc.). This device provides a series of air puffs to the sealed ear canal. Using this device requires that ventilation tubes (PE tubes) are inserted in the eardrum to make it possible for the air puffs to reach the middle-ear cavity. The Meniett device is now in use for management Ménière's disease.

The efficacy of such treatment was studied by Odkvist et al. (2000) [43] in a prospective randomized placebo-controlled, multicenter clinical trial. The study had

56 participants with active Ménière's disease, age 20–65 years, with a hearing loss of 20–65 dB PTA. Thirty-one participants completed 2 weeks using the Meniett device and 25 patients completed the 2 weeks with the placebo device. A grommet (PE tube) was inserted in the eardrum on the affected side 2 weeks before the study began. The active group experienced significant improvement concerning the frequency and intensity of vertigo, dizziness, aural pressure, and tinnitus, assessed using a visual analogue scale (VAS). The placebo group experienced no difference from the normal course of their disease. Pure-tone threshold improved at the frequencies 500 and 1,000 Hz after active treatment, but there were no improvement of hearing after placebo treatment. Boudewyns et al. reported a significant decrease in the median number of vertigo spells without any improvement in hearing status, tinnitus and functional level, or self-perceived dizziness handicap [44].

In another study, Densert and Sass (2001) [45] found beneficial effect on the symptoms in 37 individuals with Ménière's disease, 31 of whom had failed to respond to medical treatment in a 2-year follow-up; 19 were free from vertigo spells; 15 had a significantly fewer vertigo spells; and 3 did not respond to pressure treatment. These three individuals later had treatment with gentamicin injections, one of these three became deaf in the affected ear. None of the patients' conditions when treated with air puffs became worse [45]. All participants in the study reported improvement in functionality of at least two levels, according to the AAO-HNS functionality scale.

References

1. Beasley NJ, NS Jones (1996) Ménière's disease: evolution of a definition. *J Laryngol Otol.* 110.12: 1107–1113.
2. Salt AN, RM Gill, SK Plontke (2008) Dependence of hearing changes on the dose of intratympanically applied gentamicin: a meta-analysis using mathematical simulations of clinical drug delivery protocols. *Laryngoscope.* 118.10: 1793–1800.
3. Magliulo G, G Cianfrone, M Gagliardi et al (2004) Vestibular evoked myogenic potentials and distortion-product otoacoustic emissions combined with glycerol testing in endolymphatic hydrops: their value in early diagnosis. *Ann Otol Rhinol Laryngol.* 113.12: 1000–1005.
4. Fetterman BL (2001) Distortion-product otoacoustic emissions and cochlear microphonics: relationships in patients with and without endolymphatic hydrops. *Laryngoscope.* 111.6: 946–954.
5. Rotter A, S Weikert, J Hensel et al (2008) Low-frequency distortion product otoacoustic emission test compared to ECoG in diagnosing endolymphatic hydrops. *Eur Arch Otorhinolaryngol.* 265.6: 643–649.
6. Ferraro JA, JD Durrant (2006) Electrocochleography in the evaluation of patients with Ménière's disease/endolymphatic hydrops. *J Am Acad Audiol.* 17.1: 45–68.
7. Eggermont JJ (1979) Summating potentials in Ménière's disease. *Arch Otorhinolaryngol* 222: 63–75.
8. Kentala E, M Havia, I Pyykkö (2001) Short-lasting drop attacks in Ménière's disease. *Otolaryngol Head Neck Surg.* 124.5: 526.
9. Schwaber MK, JW Hall (1992) Cochleovestibular nerve compression syndrome. I. Clinical features and audiovestibular findings. *Laryngoscope.* 102.9: 1020–1029.
10. Møller MB, AR Møller, PJ Jannetta et al (1993) Microvascular decompression of the eighth nerve in patients with disabling positional vertigo: selection criteria and operative results in 207 patients. *Acta Neurochir (Wien).* 125.1–4: 75–82.
11. Ryu H, S Yamamoto, K Sugiyama et al (1998) Neurovascular compression syndrome of the eighth cranial nerve. What are the most reliable diagnostic signs. *Acta Neurochir (Wien).* 140.12: 1279–1286.
12. Møller MB (1988) Controversy in Ménière's disease: results of microvascular decompression of the eighth nerve. *Am J Otol.* 9.1: 60–63.
13. Minor LB, DA Schessel, JP Carey (2004) Ménière's disease. *Curr Opin Neurol* 17: 9–16.
14. Battista RA (2004) Audiometric findings of patients with migraine-associated dizziness. *Otol Neurotol.* 25.6: 987–992.
15. Vibert N, PP Vidal (2001) In vitro effects of acetyl-dl-leucine (tanganil) on central vestibular neurons and vestibulo-ocular networks of the guinea-pig. *Eur J Neurosci.* 13.4: 735–748.
16. Novotný M, R Kostrica (2002) Fixed combination of cinnarizine and dimenhydrinate versus betahistine dimesylate in the treatment of Ménière's disease: a randomized, double-blind, parallel group clinical study. *Int Tinnitus J.* 8.2: 115–123.
17. Ganança MM, HH Caovilla, FF Ganança (2009) Comparable efficacy and tolerability between twice daily and three times daily betahistine for Ménière's disease. *Acta Otolaryngol.* 129.5: 487–492.
18. Simpson JJ, WE Davies (1999) Recent advances in the pharmacological treatment of tinnitus. *Trends Pharmacol Sci.* 20.1: 12–18.
19. Thirlwall AS, S Kundu (2006) Diuretics for Ménière's disease or syndrome. *Cochrane Database Syst Rev.* 19.3: CD003599.
20. Morales-Luckie E, A Cornejo-Suarez, MA Zaragoza-Contreras et al (2005) Oral administration of prednisone to control refractory vertigo in Ménière's disease: a pilot study. *Otol Neurotol.* 26.5: 1022–1026.
21. Derebery MJ (2000) Allergic management of Ménière's disease: an outcome study. *Otolaryngol Head Neck Surg.* 122.2: 174–182.
22. Thai-Van H, MJ Bounaix, B Fraysse (2001) Ménière's disease: pathophysiology and treatment. *Drugs.* 61.8: 1089–1102.
23. Suryanarayanan R, JA Cook (2004) Long-term results of gentamicin inner ear perfusion in Ménière's disease. *J Laryngol Otol.* 118.7: 489–495.

24. Eklund S, I Pyykkö, H Aalto et al (1999) Effect of intratympanic gentamicin on hearing and tinnitus in Ménière's disease. *Am J Otol.* 20.3: 350–356.
25. Diamond C, O'Connell DA, Hornig JD et al (2003) Systematic review of intratympanic gentamicin in Ménière's disease. *J Otolaryngol.* 32.6: 351.361.
26. De Beer L, R Stokroos, H Kingma (2007) Intratympanic gentamicin therapy for intractable Ménière's disease. *Acta Otolaryngol.* 127.6: 605–612.
27. Dodson KM, A Sismanis (2004) Intratympanic perfusion for the treatment of tinnitus. *Otolaryngol Clin North Am.* 37.5: 991–1000.
28. Garduño-Anaya MA, H Couthino De Toledo, R Hinojosa-González et al (2005) Dexamethasone inner ear perfusion by intratympanic injection in unilateral Ménière's disease: a two-year prospective, placebo-controlled, double-blind, randomized trial. *Otolaryngol Head Neck Surg.* 133.2: 285–294.
29. Light JP, H Silverstein (2004) Transtympanic perfusion: indications and limitations. *Curr Opin Otolaryngol Head Neck Surg.* 12.5: 378–383.
30. Araújo MF, CA Oliveira, FM Bahmad (2005) Intratympanic dexamethasone injections as a treatment for severe, disabling tinnitus: does it work? *Arch Otolaryngol Head Neck Surg.* 131.2: 113–117.
31. Brinson GM, DA Chen, MA Arriaga (2007) Endolymphatic mastoid shunt versus endolymphatic sac decompression for Ménière's disease. *Otolaryngol Head Neck Surg.* 136.3: 415–421.
32. Dandy WE (1941) Surgical treatment of Ménière's disease. *Surg. Gynecol. Obstet.* 72: 421–425.
33. Magnan J, G Bremond, A Chays et al (1991) Vestibular neurectomy by retrosigmoid approach: technique, indications, and results. *Am J Otol.* 12.2: 101–104.
34. Miyazaki H, A Deveze, J Magnan (2005) Neuro-otologic surgery through minimally invasive retrosigmoid approach: endoscope assisted microvascular decompression, vestibular neurectomy, and tumor removal. *Laryngoscope.* 115.9: 1612–1617.
35. Li CS, JT Lai (2008) Evaluation of retrosigmoid vestibular neurectomy for intractable vertigo in Ménière's disease: an interdisciplinary review. *Acta Neurochir (Wien).* 150.7: 655–661.
36. Baguley DM, P Axon, IM Winter et al (2002) The effect of vestibular nerve section upon tinnitus. *Clin Otolaryngol Allied Sci.* 27.4: 219–226.
37. Møller MB, AR Møller, PJ Jannetta et al (1986) Diagnosis and surgical treatment of disabling positional vertigo. *J. Neurosurg.* 64: 21–28.
38. Jannetta PJ, MB Møller and AR Møller (1984) Disabling positional vertigo. *New Engl. J. Med.* 310.26: 1700–1705.
39. Møller MB, AR Møller, PJ Jannetta et al (1993) Microvascular decompression of the eighth nerve in patients with disabling positional vertigo: Selection criteria and operative results in 207 patients. *Acta Neurochir (Wien).* 125: 75–82.
40. Sunderland S (1948) Microvascular relations and anomalies at the base of the brain. *J. Neurol. Neurosurg Psychiatry.* 11: 243–257.
41. Ingelstedt S, A Ivarsson and O Tjernström (1976) Immediate relief of symptoms during acute attacks of Ménière's disease, using a pressure chamber. *Acta Otolaryngol.* 85: 368–378.
42. Densert B, O Densert (1982) Overpressure in treatment of Ménière's disease. *Laryngoscope* 92: 1285–1292.
43. Odkvist LM, S Arlinger, E Billermark et al (2000) Effects of middle ear pressure changes on clinical symptoms in patients with Ménière's disease – a clinical multicentre placebo-controlled study. *Acta Otolaryngol Suppl.* 543: 99–101.
44. Boudewyns AN, FL Wuyts, M Hoppenbrouwers et al (2005) Meniett therapy: rescue treatment in severe drug-resistant Ménière's disease? *Acta Otolaryngol.* 125.12: 1283–1289.
45. Densert B and K Sass (2001) Control of symptoms in patients with Ménière's disease using middle ear pressure applications: Two years follow-up. *Acta Otolaryng (Stockh.).* 121: 616–621.