BMJ Best Practice

Meniere's disease

Straight to the point of care

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Summary

Meniere's disease (MD) is an episodic auditory and vestibular disease characterised by sudden onset of vertigo, hearing loss, tinnitus, and sensation of fullness in the affected ear. Earlier in the disease process, all symptoms may not be present.

The cause is unknown, but results in an over-production or impaired absorption of endolymph in the inner ear.

Diagnosis is made on clinical history and detailed audiological tests; other investigations may be required to exclude other causes.

Dietary changes and diuretics may control symptoms in early stages of the disease; specific medical therapies for vertigo control can be tried if required.

If symptoms persist despite maximal medical therapy, several surgical interventions are available.

Definition

Meniere's disease or Meniere syndrome is an auditory disease characterised by an episodic sudden onset of vertigo, low-frequency hearing loss (in the early stages of the disorder), low-frequency roaring tinnitus, and sensation of fullness in the affected ear.[1] Usually the terms are used interchangeably, but MD is commonly used if it is idiopathic and Meniere syndrome if it is secondary to a number of known inner-ear disorders.[2] It is also called endolymphatic hydrops, because of the described pathological state observed on post-mortem histological sectioning.[3] [4] It is unclear whether this is a cause of symptoms or a result of the pathological process.

THEORY

Epidemiology

The true incidence and prevalence of meniere's disease (MD) is not known. In a study from Rochester, Minnesota, the incidence was calculated as 15.30 per 100,000 people, with a prevalence of 218 per 100,000 population.[5] Data from South Korea report the incidence of MD in 2017 between 78.03 and 158.80 people per 100,000, in men and women respectively.[6]

MD is primarily a disease of adulthood, although several cases have been reported in children. Onset usually occurs in the fourth decade.[3] MD seems to have a higher prevalence in white people, and around 50% of patients have a family history of MD.[7] [8] MD is slightly more common in females, with a 2.17:1 female-to-male ratio.[6]

Incidence of bilateral disease varies in the literature between 2% and 73%.[9] [10] Other studies report that bilateral disease may occur in up to 50% of patients with MD during their lifetime.[3] [7] [11] More than 10% of people with apparent unilateral disease may be found to have bilateral disease on testing.[12]

Aetiology

The underlying cause of meniere's disease (MD) remains unknown. Some authors, however, believe that in up to 55% of cases, a specific aetiological agent can be identified.[13] [14] Among these aetiological agents are allergic responses (especially to food), congenital or acquired syphilis, Lyme disease, hypothyroidism, stenosis of the internal auditory canal, and acoustic or physical trauma.[13] Viral infection and immune-mediated mechanisms affecting the absorption of endolymph have also been implicated.[15] Hereditary factors are thought to play a role in the development of MD.[15] A multi-factorial inheritance may be the best model, leading to endolymphatic malabsorption and subsequent hydrops.[15]

Pathophysiology

Endolymphatic hydrops is thought to be due to over-production or impaired absorption of endolymph. This may occur as a result of one or a combination of the proposed aetiological agents. Some histopathological studies of the temporal bones suggest that, although endolymphatic hydrops is a histological marker for MD, it is not directly responsible for its symptoms.[16] However, studies from 2010 demonstrate through magnetic resonance imaging the central role of endolymphatic hydrops in the pathology of MD.[17]

During the acute attack the excessive endolymphatic fluid pressure causes distension and rupture of Reissner's membrane. This results in the release of potassium-rich endolymph into the perilymphatic space and causes injury to the sensory and neural elements of the inner ear, which manifests clinically as sudden hearing loss, tinnitus, and vertigo. Between attacks, Reissner's membrane may reattach itself, the chemical balance is restored, and symptoms remit.[3] However, some researchers are questioning this theory because membrane ruptures were found post-mortem in temporal bones with no history of vertigo.[16]

Immune-mediated mechanisms have long been implicated in the pathophysiology of MD. This has been supported by the presence of increased levels of immune complexes and the presence of auto-antibodies to structures of the inner ear in patients with MD. Lymphocytes and immunoglobulins have also been found in the endolymphatic sac.[18]

Case history

Case history #1

A 40-year-old woman presents with a 1-year history of recurrent episodes of vertigo. The vertigo spells are described as a sensation of the room spinning that lasts from 20 minutes to a few hours and may be associated with nausea and vomiting. The spells are incapacitating and are accompanied by dizziness, vertigo, and disequilibrium, which may last for days. No loss of consciousness is reported. The patient also reports aural fullness, tinnitus, and hearing loss in the right ear that is more pronounced around the time of her vertigo spells. Physical examination of the head and neck is normal. A horizontal nystagmus is noted. She is unable to maintain her position during Romberg's testing or the Fukuda stepping test. She turns towards the right side and is unable to walk tandem. Her cerebellar function tests are normal.

Other presentations

Patients may present with any combination of hearing loss, tinnitus, vertigo, or aural fullness.

Approach

Risk factors

Meniere's disease (MD) usually presents in middle-aged people, with fluctuating auditory and vestibular symptoms. A family history of MD is present in up to 50% of patients. Patients with associated autoimmune disorders may have an autoimmune inner-ear disorder. These patients usually present with bilateral symptoms.[20]

History

Classic MD has the triad of vertigo, hearing loss, and tinnitus. Vertigo is unprovoked, sudden in onset, spinning in nature, and often incapacitating. It lasts minutes to hours and may be associated with nausea and vomiting.[4] Hearing loss is usually worse during acute attacks, especially in early stages of the disease. As the disease progresses, hearing loss increases in severity and may become constant. Tinnitus is described as roaring in nature and may be severe. Aural fullness is a sensation of pressure and fullness in the ear or ear discomfort and may also be present during the episode.

An atypical presentation of MD is fluctuating hearing loss and tinnitus without vertigo. This is usually referred to as cochlear hydrops, and up to 40% of patients will eventually develop vertigo.[22]

Some patients with MD complain of drop attacks, which are described as sudden loss of balance without loss of consciousness or other autonomic or neurological symptoms. The incidence varies between studies and ranges between 3% to 10% of patients.[9] [23] One study reported an unusually high incidence of 72%.[24] They are more common in end-stage disease and in the older population.[23]

Bilateral disease may be present in around 30% to 50% of patients.[3] [7] [11]

Physical examination

Head and neck examination in patients with MD is usually normal. Horizontal and/or rotatory nystagmus that can be suppressed by visual fixation may be present. A positive Romberg's test (inability to stand with feet together and eyes closed) and an inability to walk tandem (heel-to-toe) in a straight line is often present. In the Fukuda stepping test (also known as the Unterberger test) the patient is asked to march in place with eyes closed, and may be unable to maintain position and will turn towards the affected side.

Audiology

Complete audiological evaluation is important for the diagnosis of MD and should be done in any patient presenting with hearing loss, tinnitus, vertigo, or aural fullness. Complete audiological evaluation includes pure-tone air and bone conduction with appropriate use of masking, speech audiometry, tympanometry, and oto-acoustic emissions. Hearing loss in patients with MD is typically sensorineural in nature and mainly in the low frequencies, although other configurations of hearing loss may be present. Usually, low-frequency hearing loss is present in the early stages of MD and during or before attacks. As the disease progresses, middle and high frequencies are affected. There may be a disproportionate drop in word score recognition in comparison with pure-tone findings. Serial audiological evaluation might show fluctuation in the hearing, which at times is helpful in making the diagnosis in patients with MD.

Once the diagnosis of MD is suspected, electrocochleography (ECochG) may be helpful. ECochG is a technique for recording the electrical events of the cochlea. The clinical application is confined to the stimulus-related cochlear potentials and usually includes measurement of the entire nerve or compound

action potential of the auditory nerve. An ECochG consists of a cochlear microphonic and measures the cochlear summating potential and the action potential, independently or in combination. Broad-band clicks or tonal stimuli are used to evoke the components of interest.[25] [26] [27] [28] [29] ECochG should not be used when the pure-tone average for frequencies 1000-4000 Hz reaches or exceeds 50 dB HL. It does have value in the early stages of the disease when symptoms are present but audiometry is normal.[30] Once hearing is affected, the best serial test is pure-tone audiometry in the affected ear or ears. However, it is the author's opinion that the test should be performed at low frequencies down to 125 Hz.

Vestibular testing using electronystagmography or videonystagmography is performed routinely in patients presenting with vertigo, dizziness, or loss of balance. Rotatory chair or vestibular-evoked myogenic potentials are not available in all centres, but may be helpful in the diagnosis of MD. Vestibular testing may not be possible during or shortly after acute attacks.

Other investigations

Any patients with asymmetry of hearing should have magnetic resonance imaging (MRI) with gadolinium to exclude a retro-cochlear cause of hearing loss, such as acoustic neuroma.

3-D MRI protocols have been developed to delineate endolymphatic hydrops, but they are still in the investigational stages.[31] MRI, after both intravenous and intratympanic injection of gadolinium, is being studied.[32] [33] [34]

Patients with MD who have bilateral symptoms and do not respond to conventional therapy should be tested for autoimmune disorders. This may include anti-nuclear antibody, anti-neutrophil cytoplasmic antibody, and rheumatoid factor.[35]

Patients with an acute or recent decrease in hearing should be assessed for hypothyroidism, Lyme disease, and syphilis.

History and exam

Key diagnostic factors

presence of risk factors (common)

- Risk factors include positive family history, recent viral illness, and autoimmune disorders.

vertigo (common)

- Recurrent episodes of vertigo, described as a spinning sensation lasting minutes to hours. Usually associated with nausea and vomiting. Attacks tend to cluster in groups.

hearing loss (common)

- Usually fluctuating and worsens during or around the vertigo spells in initial stages.
- May become constant in later stages. Usually unilateral in the affected ear.

tinnitus (common)

- Usually described as roaring tinnitus. Usually unilateral in the affected ear.

aural fullness (common)

- Occurs in the affected ear.
- May increase prior to an attack.

drop attacks (uncommon)

- Sudden loss of balance without loss of consciousness or other autonomic or neurological symptoms.
- More common in older people but can occur at any age.
- Tend to occur in late stages.

Other diagnostic factors

positive Romberg's test (common)

- Swaying or falling when asked to stand with feet together and eyes closed.

Fukuda's stepping test (common)

- Turning towards the affected side when asked to march in place with eyes closed.

bilateral symptoms (uncommon)

- Autoimmune inner-ear disease is suspected in patients with bilateral meniere's disease or history of other autoimmune systemic disorders.

nystagmus (uncommon)

- Horizontal and/or rotatory nystagmus that can be suppressed by visual fixation.
- Seen in acute attacks.

tandem walk (uncommon)

- Inability to walk (heel-to-toe) in a straight line.

Risk factors

Weak

recent viral infection

- Viral infection with haematogenous spread, or direct spread through the round window membrane as a result of middle-ear infection or at the time of an upper respiratory tract infection, has been implicated in meniere's disease (MD).[15]
- It is thought that viral infections cause inflammation in the inner ear and subsequently trigger a reactive immune response in the vicinity of the endolymphatic sac, causing damage to its function.
- Evidence of viral aetiology is supported by demonstration of the presence of human cytomegalovirus by polymerase chain reaction in the endolymphatic sac tissues; antiviral immunoglobulin E in the sera of patients with MD; elevated anti-herpes simplex virus immunoglobulin G (IgG) in the perilymph of patients with MD; and higher titres of IgG against adenovirus and varicella zoster in patients when compared with controls.[19]

genetic predisposition

- There have been several reports in the literature about a genetic predisposition in meniere's disease (MD). Hereditary factors have been found to play a role in 10% to 50% of cases.[15]
- The most commonly reported mode of inheritance is autosomal dominant pattern, but X-linked inheritance has also been reported.
- Higher frequency of histocompatibility antigen (HLA-DR, -DQ, and -DP) has been reported in patients with MD compared with controls.[15]

autoimmune disease

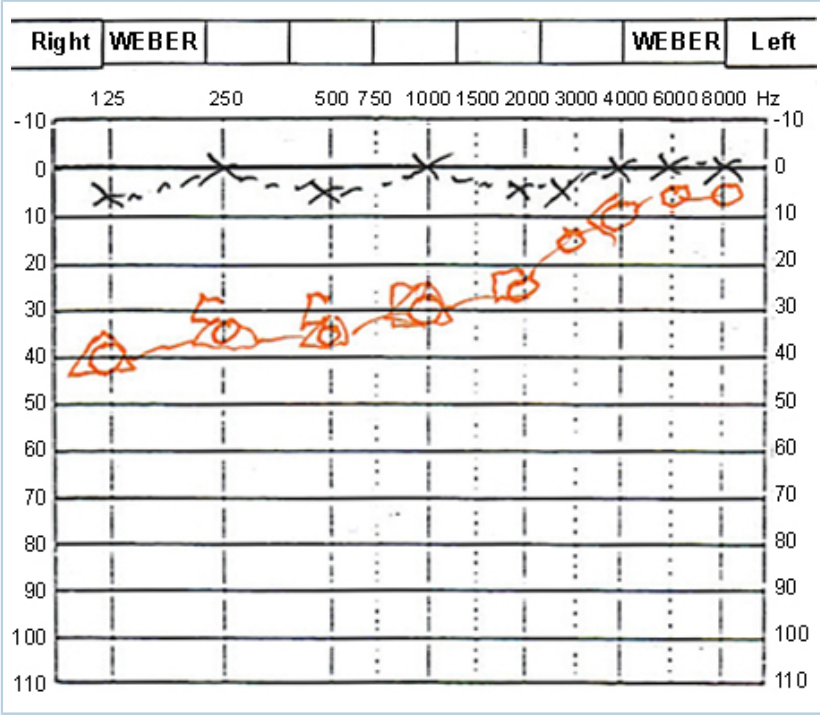
- Patients with associated autoimmune disorders such as vasculitis, rheumatoid arthritis, and lupus may have an autoimmune inner-ear disorder.[20] This is especially likely when meniere's disease is bilateral at initial presentation.

increasing age

- Several studies indicate that prevalence and incidence rates increase with age, up to an age threshold of approximately 80 years.[6] [21] meniere's disease is reported almost exclusively in adults, with peak onset occurring between aged 40 to 50 years.[8] [21]

Investigations

1st test to order

Test	Result
pure-tone air and bone conduction with masking • Pure-tone audiometry is the basic measurement of hearing sensitivity and the integrity of the entire auditory receptive pathway. Air-conduction thresholds are measured under headphones or with insert earphones. Bone measurements attempt to by-pass the outer and middle ear and test the function of the cochlea and the auditory nerve. • In MD (as well as in other cochlear disorders), air and bone conduction are equal, indicating that the underlying pathology is in the cochlea or auditory nerve, not the outer and the middle ear.  *Pure-tone audiometry typical of Meniere's disease* *From the collection of Maurice H. Miller, PhD*	**unilateral sensorineural hearing loss; usually low-frequency hearing loss is present in early stages of meniere's disease (MD) and during or before attacks; as disease progresses, middle and high frequencies are affected**
speech audiometry • Speech recognition threshold (SRT) measures the threshold (50% correct response) for a series of simple, everyday bisyllabic words such as keyboard, football, and pavement. • Discrepancies between SRT and pure-tone averages can suggest pseudohypacusis (non-organic hearing loss) or a severe problem in word recognition ability (discrimination). Such discrepancies are not characteristic of meniere's disease (MD). • Word recognition score (WRS) measures the percentage of correct monosyllabic words, which are heavily loaded with high-frequency consonantal material (at increasing levels of intensity), above the SRT. • WRS at multiple levels above the SRT is used to access the rollover phenomenon. The roll-over ratio is calculated by subtracting the lowest speech discrimination score of phonetically balanced (PB)	**no discrepancies on SRT, absence of positive roll-over index**

Test	Result
words (PB min) from the highest score (PB max) and dividing this figure by the PB max. A ratio ≥0.45 indicates a retro-cochlear lesion. MD patients should not show a positive roll-over index.[36] • Serial audiometry is helpful in making the diagnosis as well as a means of follow-up on the degree of hearing loss.	
tympanometry/immittance/stapedial reflex levels • Immittance evaluation including tympanometry stapedial reflex measurements (both ipsilateral and contralateral) and measurement of acoustic reflex decay.	**normal tympanogram; elicitation of acoustic reflex <60 dB patient threshold; no abnormal reflex decay**
oto-acoustic emissions (OAE) • OAE are an electrophysiological measure of outer hair cell dysfunction. • Although measurable OAE is absent in the frequency range affected by meniere's disease (MD), 18% of hydropic ears had unexpectedly present emissions even when the pure-tone thresholds were ≥50 dB.[37] • Sounds produced by the cochlea are measured with a sensitive microphone in the ear canal, using a brief stimulus such as a click (transient-evoked OAE) or 2 stimulus tones of different frequency (distortion product OAE). • These findings are not specific to MD but also apply to other cochlear disorders. OAE may also be affected by middle-ear disease.	**absence of measurable OAE in frequency range affected by MD**

Other tests to consider

Test	Result
electrocochleography • Measures electrical potentials that are derived from the hair cells in the cochlea and the auditory nerve. • Should be repeated during the quiescent stage of the disease.	**abnormally large summating potential amplitude relative to the action potential amplitude**
electronystagmography • Records eye movements and responses to ocular and vestibular stimuli. • A unilateral decreased vestibular response in the affected ear suggests peripheral aetiology, such as vestibular neuronitis.[38] • Caloric response diminishes with increased disease duration, and a canal paresis of 35% to 50% is commonly observed in the affected ear.[9] • Great care should be exercised in performing vestibular testing when the patient is having an acute attack of meniere's disease (MD). Some vestibular evaluation procedures may precipitate an acute attack and should be avoided if not necessary for diagnosis.	**abnormal in MD; unilateral decreased vestibular response in the affected ear is common**
rotary chair test • Sinusoidal harmonic acceleration or rotating chair testing involves a variety of measurements of nystagmus on a patient who is rotated from side to side during the procedure in a computer-controlled chair.	**decreased gain, abnormal phase, and asymmetry in the response**
vestibular-evoked myogenic potential (VEMP) • VEMP uses an intense, brief auditory stimulus to assess the saccule ipsilateral to the stimulus.	**increased amplitude in early disease; attenuated or absent in later stages**
MRI of internal auditory canals • Any patient with unilateral, sudden, or asymmetrical sensorineural hearing loss should have MRI with gadolinium to exclude a retro-cochlear cause of hearing loss (such as acoustic neuroma).	**normal in meniere's disease**
thyroid function tests • Elevated thyroid-stimulating hormone and low thyroxine if hypothyroidism is cause of hearing loss.	**normal in meniere's disease**
Lyme disease and syphilis serology • Positive titres suggest these conditions are the cause of acute or recent deterioration in hearing.	**normal in meniere's disease**
anti-nuclear antibody • High sensitivity, but low specificity for systemic lupus erythematosus.[35]	**negative in most cases; positive titre in the presence of associated autoimmune pathology**
anti-neutrophil cytoplasmic antibody • Associated with vasculitis.	**negative in most cases; positive titre in the presence of associated autoimmune pathology**
rheumatoid factor • Sensitive, but non-specific for rheumatoid arthritis.[35]	**negative in most cases; positive titre in the presence of associated autoimmune pathology**

Emerging tests

Test	Result
3-dimensional MRI • 3-D MRI protocols have been developed to delineate endolymphatic hydrops, but they are still in the investigational stages. MRI, after both intravenous and intratympanic injection of gadolinium, is being studied.[32] [33] [34]	**perilymphatic space surrounding the endolymph is either small or unable to be visualised**

Differentials

Condition	Differentiating signs / symptoms	Differentiating tests
Acoustic neuroma	• Small acoustic tumours typically present as unilateral high-frequency hearing loss with difficulty hearing on the telephone on affected ear. • Acoustic neuroma should be ruled out in any unilateral sensorineural hearing and therefore in patients with meniere's disease.[39]	• There is reduced word recognition score to an inordinate degree when compared with pure-tone air and bone conduction testing (phonemic regression), roll-over phenomenon, absent or elevated acoustic reflexes, abnormal findings on stapedial reflex decay, and abnormal auditory brainstem response. • Hearing tests may be within normal limits in patients with small acoustic neuromas. • MRI with gadolinium contrast will show a tumour involving the acoustic nerve.
Vestibular migraine (also called migraine-associated dizziness and migraine-associated vertigo)	• Incidence of migraine is significantly greater in populations of meniere's disease (MD) patients, and incidence of complaints of dizziness and MD is greater in migraine populations, than the incidence of either in the general unselected population.[40] • Symptoms and clinical test findings produced by both disorders overlap, and both conditions can co-exist in the same patient. • A very short (<15 minutes) or prolonged (>24 hours) duration of vertigo suggests migraine, and visual auras are more likely. Hearing loss is usually mild and stable over time.	• Investigations are variable and non-specific. Diagnosis is made on clinical history.[40]
Vestibular neuronitis	• Neural degeneration or viral infection of the eighth nerve can produce acute or chronic vertigo, nausea, and vomiting. There is no hearing loss, tinnitus, or aural fullness. • Occurs in epidemics and is most common in people between 40 and 50 years	• Electronystagmography (particularly using bi-thermal caloric testing) often shows unilateral weakness on the affected side, but may be normal.

Condition	Differentiating signs / symptoms	Differentiating tests
	of age. Frequently, patients have had a recent viral infection. • Attacks are of precipitous onset, often occurring at night. Severe rotational vertigo lasts 12 to 36 hours with decreasing disequilibrium for the next 4 to 5 days.	
Viral labyrinthitis	• Similar presentation to vestibular neuronitis but accompanied by hearing loss and tinnitus.[41]	• Patients show various degrees of hearing loss on complete audiological evaluation.
Benign paroxysmal positional vertigo (BPPV)	• Patients typically present with episodic vertigo lasting in the range of a few seconds to a minute elicited by certain head movements. These movements include lying flat with the neck extended and turned towards the affected ear, neck extension, and bending over. Unlike attacks caused by meniere's disease (MD), the vertigo spells are not associated with hearing loss, tinnitus, or aural fullness. The vertigo could recur over a period of weeks to months and may resolve spontaneously. Patients usually report a history of trauma or vestibular neuritis. It is important to note that BPPV and MD have been reported to co-exist in the same patient.[42]	• Hallpike's test will show rotatory nystagmus on the affected side. This is performed by starting in the sitting position then bringing to a supine position with the head turned 45° towards one side with 20° neck extension. Patients with BPPV usually demonstrate a short-lasting torsional nystagmus in this position.[43]
Vertebrobasilar insufficiency	• Cerebrovascular disease is more common in older people. The vertigo might be secondary to ischaemia of the labyrinth, brain stem, or both, because they are all supplied by the vertebrobasilar circulation.[44] • Vertigo spells usually last for several minutes, and are accompanied by nausea, vomiting, and severe imbalance. Associated	• Carotid duplex ultrasound may show changes of atherosclerosis, which implies changes in the cerebral circulation. • CT head may show evidence of previous cerebral infarction. • Magnetic resonance angiography of vessels of neck, base of skull, and circle of Willis may be abnormal.

Condition	Differentiating signs / symptoms	Differentiating tests
	symptoms may include visual blurring or black-outs, diplopia, drop attacks, weakness and numbness of the extremities, and headache.[44]	

Criteria

Diagnostic criteria for Menière's disease: Classification Committee of the Bárány Society, Japan Society for Equilibrium Research, European Academy of Otology and Neurotology (EAONO), Equilibrium Committee of the American Academy of Otolaryngology-Head and Neck Surgery (AAO-HNS), Korean Balance Society[45]

Definite meniere's disease (MD)

- Two or more spontaneous episodes of vertigo, each lasting 20 minutes to 12 hours
- Audiometrically documented low- to medium-frequency sensorineural hearing loss in one ear, defining the affected ear on at least one occasion before, during, or after one of the episodes of vertigo
- Fluctuating aural symptoms (hearing, tinnitus, or fullness) in the affected ear
- Not better accounted for by another vestibular diagnosis.

Probable MD

- Two or more episodes of vertigo or dizziness, each lasting 20 minutes to 24 hours
- Fluctuating aural symptoms (hearing, tinnitus, or fullness) in the affected ear
- Not better accounted for by another vestibular diagnosis.

Ten-point scale for the clinical diagnosis of MD[12]

Based on clinical history. One point awarded to each of the following. The closer the score is to 10, the more likely the patient is to have MD.

- Rotational vertigo
- Attacks of vertigo lasting >10 minutes
- Rotational vertigo associated with 1 or more of hearing loss, tinnitus, or aural pressure
- Sensorineural hearing loss
- Fluctuating hearing loss
- Hearing loss or fluctuation associated with vertigo, tinnitus, or aural pressure
- Peripheral tinnitus lasting >5 minutes
- Tinnitus fluctuating or changing with 1 or more of the following: vertigo, hearing loss, or aural pressure
- Aural pressure/fullness lasting >5 minutes
- Aural pressure fluctuating or changing with vertigo, hearing loss, or tinnitus.

Approach

There is no cure for meniere's disease (MD). The goals of treatment are vertigo control, prevention of further deterioration in hearing whenever possible, amelioration of tinnitus, and balance control. Treatment options, however, do not appear to influence hearing results or the natural history of MD.[46]

Endolymphatic hydrops has been implicated in the pathophysiology or pathogenesis of MD and, therefore, the management of patients with MD has traditionally been targeted towards decreasing endolymphatic pressure. This has been questioned by a study suggesting that such measures aiming at reduction in hydrops would be unlikely to control the disease. Some histopathological studies of the temporal bones suggest that, although endolymphatic hydrops is a histological marker for MD, it is not directly responsible for its symptoms.[16] However, studies from 2010 demonstrate through magnetic resonance imaging the central role of endolymphatic hydrops in the pathology of MD.[17] It is important to note that MD presents a research controversy in evaluating the efficacy of different therapies.[47] [48] [49]

Dietary changes and lifestyle modification

All patients should be educated on dietary changes and lifestyle modification. Patients should be advised to restrict salt intake to 1500 to 2300 mg/day, as this is thought to prevent sodium-related water retention and re-distribution into the endolymphatic system.[21] [50] Although there are no randomised controlled trials (RCTs) to document the benefits of low-salt diet on the treatment of MD, patients often report exacerbation of their symptoms or even precipitation of an attack after a salty meal.[50] [51]

Limiting caffeine intake, reducing alcohol consumption, ceasing smoking, and managing stress are also advisable, as these may trigger an attack. However, there is no evidence from RCTs to support or refute the restriction of salt, caffeine, or alcohol intake in patients with MD.[51] [52]

Such dietary changes may be the only necessary treatment required in the early stages of the disease.

Medical therapy to decrease endolymphatic pressure

Diuretics are believed to reduce the volume of the endolymph and may be offered for maintenance therapy.[21] [53] The most commonly used diuretics in the treatment of MD are thiazides-with or without potassium-sparing diuretics (e.g., hydrochlorothiazide/triamterene)-and acetazolamide.[21] Thiazide diuretics are thought to act on the sodium/potassium adenosine triphosphatase levels in the stria vascularis in cochlear tissues and to have an effect on the maintenance of endolymph homeostasis.[54] Acetazolamide is thought to act on carbonic anhydrase in dark cells and in the stria vascularis.[50] If the patient remains symptom-free for 6 months, diuretics may be slowly tapered and re-started if required. If there is no response, the patient should be changed to the alternative diuretic. These medicines should not be used in patients with a known or suspected reaction to sulfonamides.

The evidence of the efficacy of diuretics on MD is controversial, and direct evidence of its efficacy on disease progression is lacking in the literature.[2] However, diuretics are still considered by many physicians to be first-line treatment in patients with MD.

Symptomatic treatments

Vertigo

Symptoms of individual and acute vertigo spells can be treated with vestibular suppressants and anti-emetics. However, much of the effect is from the sedative action of these drugs. The literature lacks RCTs

assessing the effects of these medicines for acute attacks of MD. Commonly used treatments include antihistamines (e.g., meclozine, dimenhydrinate, promethazine), benzodiazepines (e.g., diazepam), and phenothiazines (e.g., prochlorperazine).[55] Diazepam should only be used in acute attacks.[21] It should be prescribed at low doses where possible, and long-term prescription avoided due to the risk of dependency. Anticholinergics (e.g., hyoscine and atropine) are not commonly prescribed due to their significant side-effect profile.[21]

Betahistine is used in some countries to reduce the frequency and severity of the vertigo attacks in patients with MD. However, one Cochrane review did not find enough evidence to show its efficacy in patients with MD and one 2016 RCT found no significant differences in the mean attack rate compared with placebo.[56] [57]

Corticosteroids, whether used orally or as intratympanic injections, may be used to treat acute attacks of vertigo, especially when accompanied by acute hearing loss and tinnitus. They are widely used because of their anti-inflammatory properties, although no RCTs are available to assess their efficacy in MD.[58]

Tinnitus

Patients with severe, intractable tinnitus can receive relief with a number of modalities, such as tinnitus maskers, tinnitus retraining therapy (TRT), various forms of sound-based therapies such as neuromonics phase-shift tinnitus reduction, amplifications, medicine, and biofeedback. Tinnitus questionnaires are helpful in evaluating the severity of the problem and in documenting the effects of various treatment modalities.[59] [60] [61]

Tinnitus maskers (white noise generators) are devices similar to hearing aids that fit behind or in the ear. They produce an external sound that distracts the patient from the internal tinnitus noise.

TRT is counselling accompanied by white noise generators. TRT is a favoured treatment, but may it take up to 18 months before full benefits are achieved.[62] [63]

Amplification (hearing aids) may help in masking the tinnitus and achieving residual inhibition.

Biofeedback techniques attempt to decrease the anxiety that is associated with tinnitus. This can be achieved through relaxation techniques, hypnosis, and cognitive behavioural therapy.

Neuromonics uses a customised neural stimulus combined with specific music, delivered through a coordinated programme to interact with, interrupt, and desensitise tinnitus disturbance for long-term benefit.[64] [65] [66]

Medicines such as antidepressants (e.g., amitriptyline) and benzodiazepines (e.g., alprazolam) may help patients with intractable tinnitus, but are associated with adverse effects.[67] They should only be used if the above techniques are unsuccessful and debilitating tinnitus persists.

Hearing loss

Sudden hearing loss is treated with corticosteroids (either orally or intratympanically).

Amplification using fully digital hearing aids with variable digitally adjustable circuitry should be evaluated. The traditional view that amplification does not work for patients with MD is not based on experience with modern amplification.

New forms of directional microphones, digital signal processing circuitry, and wireless technology can provide significant benefits in helping MD patients to hear better in environments with competing noise.[68]

Assistive listening devices are a form of amplification for those with situational difficulties in hearing, and who are not yet ready or willing to use personal hearing aids.

Intensive high-quality audiological counselling is needed for patients with MD in the adjustment to, and acceptance of, amplification.

Intratympanic therapy

In intratympanic therapy, medicines are injected into the middle ear and are then absorbed through the round window into the fluid system of the inner ear. This allows targeting of the inner-ear system without exposing the body to the systemic adverse effects of the medicine in use.

Two agents can be used intratympanically in patients with MD, depending on the presenting symptoms. Intratympanic corticosteroids are more commonly used in patients with MD presenting with sudden onset of hearing loss.[69] Intratympanic gentamicin (an aminoglycoside antibiotic) injections are helpful in the treatment of intractable vertigo.

Intratympanic corticosteroid injections (e.g., dexamethasone or methylprednisolone) are used in patients with MD where systemic corticosteroids are contra-indicated, or in patients who do not respond to oral corticosteroids. One double-blind, RCT found no significant difference between intratympanic gentamicin and intratympanic methylprednisolone at controlling attacks of vertigo in patients with refractory, unilateral MD.[70] The reported success rate of intratympanic corticosteroids in other studies has been variable.[58] [71] [72] [73]

When injected into the middle ear, gentamicin preferentially destroys the vestibular labyrinth. This results in chemical labyrinthectomy and is an alternative to surgical labyrinthectomy in patients with intractable vertigo. Hearing loss can be minimised by meticulously titrating the dose of gentamicin to vertigo control, stopping therapy at the earliest signs of hearing loss, and following up closely with repeated hearing tests. This approach has been found to result in complete (81.7%) and effective (96.3%) vertigo control.[74] One meta-analysis on gentamicin injections found complete vertigo control in about 75% of the patients and complete or substantial control in about 93%.[75] Hearing level and word recognition did not deteriorate with treatment. None of the trials were double-blind or had a blinded, prospective control, and therefore the level of evidence was insufficient.[75] Overall hearing loss, as a complication of gentamicin injection, has been found in 25% of patients, with a range of 13.1% to 34.7%. In a prospective, double-blind, randomised, placebo-controlled clinical trial, intratympanic gentamicin treatment was found to reduce the score of vertigo severity and perceived aural fullness in the treatment group.[76] Evidence suggests that intratympanic gentamicin injections improve vertigo symptoms, are well tolerated, and have a low incidence of severe hearing loss.[21] However, two systematic reviews in 2023 found that evidence for the use of intratympanic gentamicin and corticosteroid injections in the treatment of patients with MD is uncertain.[77] [78]

Meniett device

The Meniett device is a handheld device that delivers intermittent pressure pulses through the ear canal and is self-administered 3 times per day. A tympanostomy tube is placed in the tympanic membrane and should be kept patent throughout the treatment. It is thought that the pressure treatment induces

longitudinal movement of endolymph and improves the hydropic condition. Evidence for the Meniett device for use in MD appears to be mixed. Initial RCTs have shown that the use of the Meniett device significantly reduced vertigo frequency in two-thirds of the patients and that the improvement was maintained long term.[79] Furthermore, no serious adverse effects have been reported in the trials.[80] In contrast, systematic reviews assessing the effectiveness of positive pressure therapy devices (including the Meniett device or similar) have failed to show any benefit of these devices in improving the symptoms of MD.[81] [82] The American Academy of Otolaryngology 2020 guidelines recommend against the use of positive pressure therapy in patients with MD.[21]

Surgical therapy

The surgical management of patients with MD has changed as a result of the introduction of less invasive procedures, including intratympanic therapy and the Meniett device.

Surgical approaches are used in patients with intractable vertigo who are refractory to medical therapy. The choice between these procedures depends on the severity of the vertiginous spells, degree of serviceable hearing, age and physical condition of the patient, condition of the opposite ear, and the patient's choice.

Surgical procedures are divided into non-destructive procedures that reverse the pathophysiology of hydrops and preserve hearing, such as endolymphatic sac surgery (ELS), and destructive procedures that abolish the vestibular response either by destroying the inner ear (as in labyrinthectomy) or by cutting the vestibular nerve (as in vestibular neurectomy).

ELS is a procedure that consists of decompression of the endolymphatic sac from the overlying bone and drainage of its endolymph. Its role in MD is controversial, with studies that show 90% resolution of vertigo, and others that demonstrate it is no more effective than placebo, or that there is insufficient evidence of the beneficial effect of ELS in MD.[7] [83] [84] [85][86] [87] [88] A recent systematic literature search and meta-analysis revealed a paucity of studies on this surgical procedure, indicating ELS may be a beneficial treatment for patients with MD.[88] However, further studies are needed to attain a better understanding of the efficacy of ELS for treating MD.

Labyrinthectomy results in loss of residual hearing and therefore is indicated in patients who have no serviceable hearing. Vestibular nerve section is aimed at preserving residual hearing and is therefore a choice in patients with serviceable hearing.

Vestibular and balance rehabilitation therapy

Vestibular and balance rehabilitation therapy is recommended for patients who have problems with balance.[89] [90] Originally, patients considered for vestibular therapy were the ones who had relief from vertigo by destructive surgery or intratympanic gentamicin injections but who complained of persistent disequilibrium. It has been reported that patients whose vertigo is controlled by medical therapy or intratympanic corticosteroid injections and who complain of disequilibrium may benefit from vestibular therapy.[91] Vestibular therapy should not be recommended to patients with MD experiencing acute vertigo attacks.[21]

Treatment algorithm overview

Please note that formulations/routes and doses may differ between drug names and brands, drug formularies, or locations. Treatment recommendations are specific to patient groups: see disclaimer

Acute (summary)		
all patients		
	1st	dietary changes and lifestyle modification
	adjunct	diuretic
symptomatic vertigo	plus	vestibular suppressant, anti-emetic, or corticosteroid
	adjunct	intratympanic injection
symptomatic tinnitus	plus	non-pharmaceutical therapy
	adjunct	antidepressant or benzodiazepine
sudden hearing loss	plus	corticosteroid

Ongoing (summary)		
persistent hearing loss		
	1st	amplification (hearing aid) or assistive listening device
	plus	intensive high-quality audiological counselling
failure of medical and intratympanic therapies; hearing adequate		
	1st	endolymphatic sac surgery
	1st	vestibular nerve section
failure of medical and intratympanic therapies; hearing severely impaired		
	1st	labyrinthectomy

Treatment algorithm

Please note that formulations/routes and doses may differ between drug names and brands, drug formularies, or locations. Treatment recommendations are specific to patient groups: see disclaimer

Acute

all patients

1st **dietary changes and lifestyle modification**

» All patients should be educated on dietary changes and lifestyle modification. Patients should be advised to restrict salt intake to 1500 to 2300 mg/day, as this is thought to prevent sodium-related water retention and re-distribution into the endolymphatic system.[21] [50] Although there are no randomised controlled trials to document the benefits of low-salt diet on the treatment of meniere's disease (MD), patients often report exacerbation of their symptoms or even precipitation of an attack after a salty meal.[50] [51]

» Limiting caffeine intake, reducing alcohol consumption, ceasing smoking, and managing stress are also advisable, as these may trigger an attack. However, there is no evidence from randomised controlled trials to support or refute the restriction of salt, caffeine, or alcohol intake in patients with MD.[51] [52]

» Such dietary changes may be the only necessary treatment required in the early stages of the disease.

adjunct **diuretic**

Treatment recommended for SOME patients in selected patient group

Primary options

» triamterene/hydrochlorothiazide: 37.5 mg (triamterene)/25 mg (hydrochlorothiazide) orally once daily
» acetazolamide: 250 mg orally (regular-release) twice daily

» Diuretics are believed to reduce the volume of the endolymph and may be offered for maintenance therapy.[21] [53]

» The most commonly used diuretics in the treatment of MD are thiazides with or without potassium-sparing diuretics (e.g., hydrochlorothiazide/triamterene) and acetazolamide.[21]

Acute

» If the patient remains symptom-free for 6 months, diuretics may be slowly tapered and re-started if required. If there is no response, the patient should be changed to the alternative diuretic. These medicines should not be used in patients with a known or suspected reaction to sulfonamides.

» The evidence of the efficacy of diuretics on MD is controversial, and direct evidence of its efficacy on disease progression is lacking in the literature.[2] However, diuretics are still considered by many physicians to be first-line treatment in all patients with MD.

symptomatic vertigo **plus** **vestibular suppressant, anti-emetic, or corticosteroid**

Treatment recommended for ALL patients in selected patient group

Primary options

» meclozine: 12.5 to 25 mg orally every 6 hours when required

OR

» dimenhydrinate: 50 mg orally every 4-6 hours when required

OR

» promethazine: 12.5 to 25 mg orally/rectally every 4-6 hours when required

Secondary options

» diazepam: 2-10 mg orally every 4-6 hours when required

OR

» prochlorperazine: 5-10 mg orally every 6-8 hours when required

OR

» prochlorperazine rectal: 25 mg rectally twice daily when required

Tertiary options

» prednisolone: 20 mg orally three times daily for 2-3 weeks, then gradually taper

» Symptoms of individual and acute vertigo spells can be treated with vestibular

Acute

suppressants and anti-emetics. However, much of the effect is from the sedative action of these drugs.

» The literature lacks randomised controlled trials assessing the effects of these medicines for acute attacks of MD.

» Commonly used treatments include antihistamines (e.g., meclozine, dimenhydrinate, promethazine), benzodiazepines (e.g., diazepam), and phenothiazines (e.g., prochlorperazine).[55] Diazepam should only be used in acute attacks.[21] It should be prescribed at low doses where possible, and long-term prescription avoided due to the risk of dependency. Prochlorperazine is a second-line treatment for patients with refractory nausea.

» Oral corticosteroids may be used to treat acute attacks of vertigo, especially when accompanied by acute hearing loss and tinnitus. They are widely used because of their anti-inflammatory properties, although no randomised controlled trials are available to assess their efficacy in MD.[58] The dose, indications, and duration of corticosteroids used vary in the literature. Oral corticosteroids have very well-known adverse effects but are often tolerated at such a dose and for short durations.

adjunct **intratympanic injection**

Treatment recommended for SOME patients in selected patient group

Primary options

» dexamethasone sodium phosphate: consult specialist for guidance on intratympanic dose

OR

» methylprednisolone sodium succinate: consult specialist for guidance on intratympanic dose

OR

» gentamicin: consult specialist for guidance on intratympanic dose

» Intratympanic corticosteroids: there is wide variation in the dose used, frequency of administration, and method of application in the literature. Tympanic membrane perforation and infection can occur.

Acute

» Intratympanic gentamicin: gentamicin (an aminoglycoside antibiotic) preferentially destroys the vestibular labyrinth when injected into the middle ear, resulting in chemical labyrinthectomy. In one prospective, double-blind, randomised, placebo-controlled clinical trial, intratympanic gentamicin treatment was found to reduce the score of vertigo severity and perceived aural fullness in the treatment group.[76] Evidence suggests that intratympanic gentamicin injections improve vertigo symptoms, are well tolerated, and have a low incidence of severe hearing loss.[21] However, two systematic reviews in 2023 found that the efficacy of intratympanic gentamicin and corticosteroid injections in the treatment of patients with MD is unclear due to the uncertainty of available evidence.[77] [78]

symptomatic tinnitus **plus** **non-pharmaceutical therapy**

Treatment recommended for ALL patients in selected patient group

» Patients with severe, intractable tinnitus can receive relief with a number of modalities, such as tinnitus maskers, tinnitus retraining therapy (TRT), hearing aids, medication, biofeedback, and neuromonics.[65]

» Tinnitus maskers (white noise generators) are devices similar to hearing aids that fit behind the ear. They produce a quiet external sound that distracts the patient from the internal tinnitus noise. In one form of tinnitus masking, the level of the masker is increased until the patient's own tinnitus is rendered inaudible. In TRT the masker remains audible along with the patient's tinnitus, and the patient learns to adjust to the audible masking level along with his or her own tinnitus. Thus, both remain audible in a graduated situation where the patient learns to tolerate his or her own tinnitus while accepting the audible tinnitus masker.

» TRT is counselling accompanied by white noise generators. TRT is a favoured treatment, but it may take up to 18 months before full benefits are achieved.[62] [63]

» Neuromonics uses a customised neural stimulus combined with specific music, delivered through a coordinated programme to interact with, interrupt, and desensitise tinnitus disturbance for long-term benefit.[64] [65] [66]

» Hearing aids may help in masking the tinnitus.

Acute

» Biofeedback techniques attempt to decrease the anxiety that is associated with the tinnitus. This can be achieved through relaxation techniques, hypnosis, and cognitive behavioural therapy.

adjunct **antidepressant or benzodiazepine**

Treatment recommended for SOME patients in selected patient group

Primary options

» amitriptyline: 25-75 mg/day orally given in 1-3 divided doses

OR

» alprazolam: 0.25 to 0.5 mg orally (immediate-release) three times daily

» Medicines such as antidepressants (e.g. amitriptyline) and benzodiazepines (e.g. alprazolam) may help patients with intractable tinnitus, but are associated with adverse effects.[67] They should only be used if non-pharmaceutical treatments are unsuccessful and debilitating tinnitus persists.

sudden hearing loss **plus** **corticosteroid**

Treatment recommended for ALL patients in selected patient group

Primary options

» prednisolone: 20 mg orally three times daily for 2-3 weeks, then gradually taper

Secondary options

» dexamethasone sodium phosphate: consult specialist for guidance on intratympanic dose

OR

» methylprednisolone sodium succinate: consult specialist for guidance on intratympanic dose

» The dose, indications, and duration of oral corticosteroids used vary in the literature. Oral corticosteroids have very well known adverse effects but are often tolerated at such a dose and for short durations.

» Intratympanic corticosteroids are used in patients with MD where systemic corticosteroids are contra-indicated, or in patients who do not respond to oral corticosteroids. There is a

MANAGEMENT

Acute

wide variation in the dose used, frequency of administration, and method of application in the literature. Tympanic membrane perforation and infection can occur.

Ongoing

persistent hearing loss

1st **amplification (hearing aid) or assistive listening device**

» Because hearing varies dramatically in these patients, access to audiology experts for re-programming of the hearing aid(s) is essential. Use of instruments incorporating algorithms to improve word recognition in noisy listening environments is also essential.

» Amplification using fully digital hearing aids with variable digitally adjustable circuitry should be evaluated. The traditional view that amplification does not work for patients with MD is not based on experience with modern amplification.

» New forms of directional microphones, digital signal processing circuitry, and wireless technology can provide significant benefits in helping MD patients to hear better in environments with competing noise.[68]

» Assistive listening devices are a form of amplification for those with situational difficulties in hearing, and who are not yet ready or willing to use personal hearing aids.

plus **intensive high-quality audiological counselling**

Treatment recommended for ALL patients in selected patient group

» Intensive high quality audiological counselling is needed for patients with MD in the adjustment to, and acceptance of, amplification.

failure of medical and intratympanic therapies; hearing adequate

1st **endolymphatic sac surgery**

» A non-destructive procedure that consists of decompression of the endolymphatic sac from the overlying bone and drainage of its endolymph. This maintains the vestibular neuroepithelium and its innervation.

» Decreases endolymphatic pressure and addresses both cochlear and vestibular dysfunctions.

» Its role in MD is controversial, with studies that show 90% resolution of vertigo, and others that demonstrate that it is no more effective than placebo, or that there is insufficient evidence of the beneficial effect of endolymphatic sac

Ongoing

surgery in MD.[7] [83] [84] [85] [86] [87] [88] A 2023 systematic literature search and meta-analysis revealed a paucity of studies on this surgical procedure, indicating ELS may be a beneficial treatment for patients with MD.[88] However, further studies are needed to attain a better understanding of the efficacy of ELS for treating MD.

» Endolymphatic sac surgery carries a risk of hearing loss in up to 2% of patients.[92] Other potential complications of this procedure include bleeding from the sigmoid sinus and cerebrospinal fluid leak.

1st **vestibular nerve section**

» In this procedure, the vestibular portion of the eighth cranial nerve (CN VIII) is selectively cut and its cochlear portion is left intact; thus, this is potentially a hearing conservation approach. This prevents the vestibular afferent stimuli from reaching the brain.

» This does not alter the pathophysiology of MD, but provides relief from vertigo, its most disturbing symptom. It should be avoided in bilateral MD, otherwise oscillopsia (perception of bouncing of the visual field with walking) and permanent imbalance may occur.

» Central compensation after vestibular nerve section is crucial for post-operative recovery of balance. Central nervous system disease such as cerebellar dysfunction, multiple sclerosis, physiological ageing, and poor medical condition are relative contra-indications for vestibular nerve section.

» Vertigo control rates are up to 90% with vestibular nerve section.[93] [94] Persistent or recurrent vertigo after vestibular nerve section can be treated by intratympanic gentamicin.

» Potential complications are uncommon and include hearing loss, facial nerve paralysis, cerebrospinal fluid leak, and headache.[93]

failure of medical and intratympanic therapies; hearing severely impaired

1st **labyrinthectomy**

» Involves surgical removal of the inner ear's neuroepithelium in an attempt to eliminate vertigo.

» Hearing loss is inevitable with this procedure and it should only be used in patients with no

Ongoing

serviceable hearing. Avoided in patients with bilateral disease, as bilateral loss of vestibular input to the brain may result in oscillopsia (perception of bouncing of the visual field with walking) and permanent imbalance.

» Central compensation after labyrinthectomy is important for balance recovery, and vestibular rehabilitation therapy after surgery may help speed the recovery. Central nervous system disease, advanced age, and a variety of significant medical conditions can prevent central compensation after surgery and these patients are, therefore, not good candidates for labyrinthectomy.

» Vertigo control rates up to 97% after have been reported.[95]

» Complications from labyrinthectomy include facial nerve injury (2%) and cerebrospinal fluid leak in 3%.[96]

Patient discussions

All patients should be educated on dietary changes and lifestyle modification. Patients with meniere's disease (MD) should be advised to restrict salt intake to 1500 to 2300 mg/day. Limiting caffeine intake, reducing alcohol consumption, ceasing smoking, and managing stress are also advisable, as these may trigger an attack. However, there is no evidence from randomised controlled trials to support or refute the restriction of salt, caffeine, or alcohol intake in patients with MD.[51]

Monitoring

Monitoring

Meniere's disease is considered a chronic disease whose activity might wax and wane over time. Patients are usually followed up for a long period of time with an otologist and an audiologist. Regular hearing tests are obtained to monitor hearing acuity and to provide appropriate amplification as needed. Patients' symptoms are monitored and treated accordingly. Dietary restrictions should be routinely reinforced, and medical or surgical therapy should be provided when needed.

Complications

Complications	Timeframe	Likelihood
falls	**variable**	**medium**
Patients who complain of imbalance and unsteadiness are at higher risk of falls. Vestibular dysfunction is an important cause of falls, where around 80% of patients who had unexplained falls were found to have symptoms of vestibular system involvement and 41% to have vertigo.[98] Patients with drop attacks are also predisposed to fall during attacks.[9]		
profound hearing loss	**variable**	**low**
Hearing loss is one of the manifestations of the disease. The incidence of bilateral, severe to profound hearing loss is estimated at 1% to 6%.[97]		

Prognosis

Most patients start with hearing loss and tinnitus. Patients may or may not then develop the complete clinical profile of meniere's disease (MD).[3]

Symptoms tend to get worse over time regardless of medical intervention. MD goes into periods of remission that are variable in duration and frequency.[3] Disproportionately greater hearing loss in low frequencies during early stages is often accompanied by disproportionately greater loss in speech comprehension than would be anticipated by degree of sensitivity. The progression of hearing loss over time is unpredictable for the individual patient.

Diagnostic guidelines

United Kingdom

Hearing loss in adults: assessment and management (https://www.nice.org.uk/guidance/ng98)

Published by: National Institute for Health and Care Excellence **Last published:** 2023

International

Diagnostic criteria for Menière's disease (http://www.jvr-web.org/ICVD.html)

Published by: Bárány Society, Japan Society for Equilibrium Research, European Academy of Otology and Neurotology, American Academy of Otolaryngology-Head and Neck Surgery, Korean Balance Society **Last published:** 2015

North America

Clinical practice guideline: Ménière's disease (https://www.entnet.org/content/clinical-practice-guidelines)

Published by: American Academy of Otolaryngology - Head and Neck Surgery Foundation **Last published:** 2020

Clinical practice guideline: sudden hearing loss (update) (https://www.entnet.org/content/clinical-practice-guidelines)

Published by: American Academy of Otolaryngology - Head and Neck Surgery Foundation **Last published:** 2019

ACR appropriateness criteria: hearing loss and/or vertigo (https://www.acr.org/Clinical-Resources/ACR-Appropriateness-Criteria)

Published by: American College of Radiology **Last published:** 2018

Treatment guidelines

North America

Clinical practice guideline: Ménière's disease (https://www.entnet.org/content/clinical-practice-guidelines)

Published by: American Academy of Otolaryngology - Head and Neck Surgery Foundation **Last published:** 2020

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Images

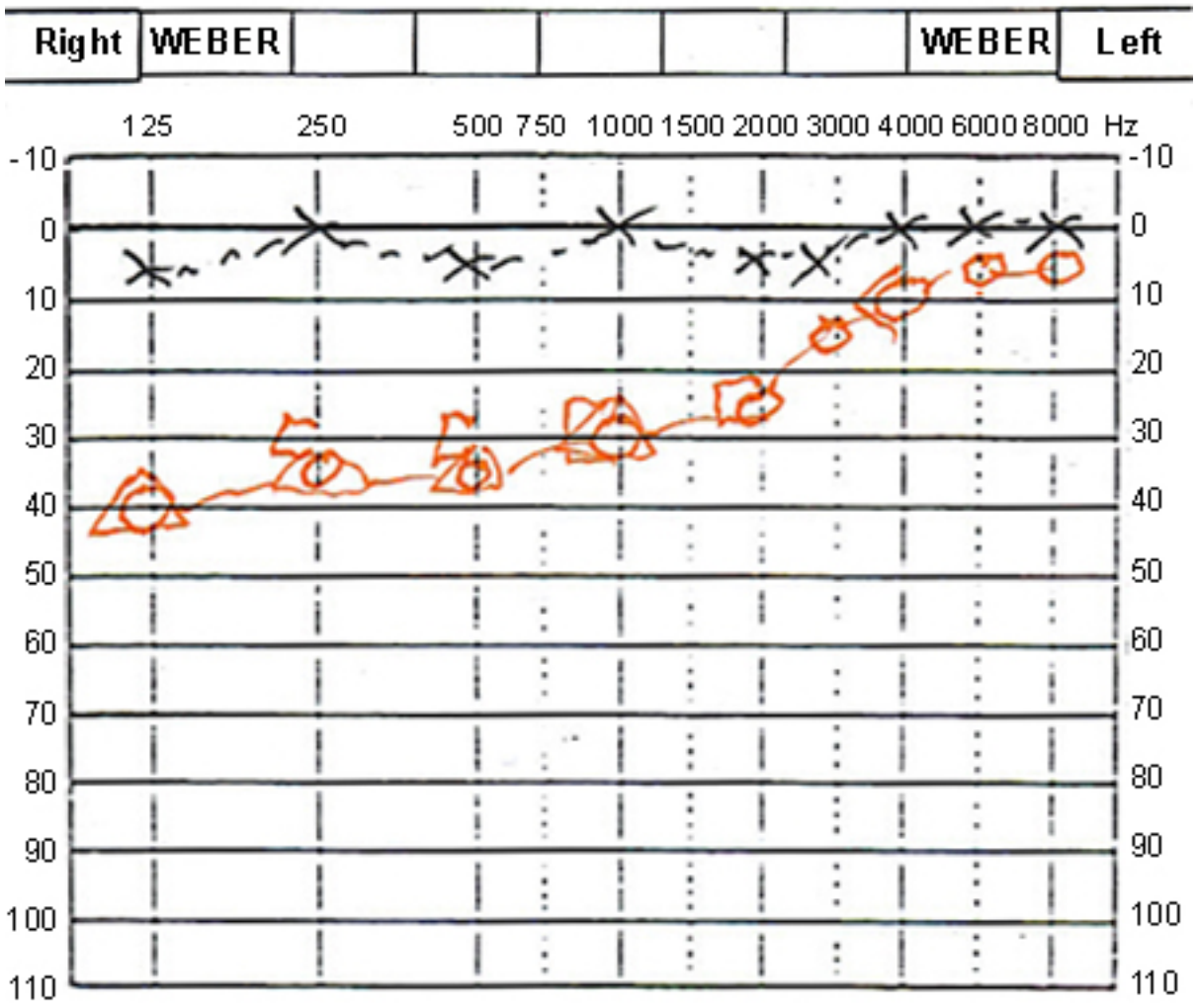

Figure 1: Pure-tone audiometry typical of Meniere's disease

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Figure 1 – BMJ Best Practice Numeral Style

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Contact us

+ 44 (0) 207 111 1105
support@bmj.com

BMJ
BMA House
Tavistock Square
London
WC1H 9JR
UK

Contributors:

// Authors:

Soha N. Ghossaini, MD, FACS

Otology-Neurotology
Ear Nose and Throat Associates of New York, New York, NY
DISCLOSURES: SNG declares that she has no competing interests.

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// Peer Reviewers:

Steven D. Rauch, MD

Associate Professor of Otology and Laryngology
Harvard Medical School, Boston, MA
DISCLOSURES: SDR declares that he has no competing interests.

Christopher J. Linstrom, MD

Professor
Otolaryngology/Head and Neck Surgery, The New York Eye and Ear Infirmary, Surgeon Director, New York, NY
DISCLOSURES: CJL declares that he has no competing interests.

Peter Rea, MA, BM BCh, FRCS(ORL-HNS)

Consultant ENT Surgeon
Leicester Royal Infirmary, Leicester, UK
DISCLOSURES: PR declares that he has no competing interests.

Doris Eva Bamiou, MD, MSc, PhD

Clinical Senior Lecturer & Consultant in Audiovestibular Medicine
Ear Institute, University College London, London, UK
DISCLOSURES: DEB declares that she has no competing interests.