Diagnosis and Management of Ménière’s Disease

In 1861, Prosper Ménière described a syndrome characterised by deafness, tinnitus and episodic vertigo that still bears his name.1 It remains one of the most challenging diseases that otolaryngologists have to manage. Its aetiology and pathogenesis are elusive and its management controversial. To date no single treatment has been shown to alter the underlying pathology or affect the clinical progression of the disease.

In 1938 the underlying consistent pathological finding of endolymphatic hydrops was described but the explanation for this phenomenon still eludes us.2

There are various treatment options available and many patients do gain symptomatic relief from what can be a disabling disease.

The incidence of Ménière’s disease is thought to be around 15 per 100 000 per year with a prevalence of 100 per 100 000 in the UK.3 The incidence between sexes is approximately equal and age of onset varies between the third and sixth decades. Most patients present in the fourth decade and it is rare for the disease to develop after the age of 60 years. Long-term follow-up would suggest the disease is bilateral in up to 50% of cases.

Diagnosis
Ménière’s disease is probably the most common diagnosis given to dizzy patients before being seen in an Otolaryngology clinic. This can be problematic as it can be difficult to dissuade the patient from his original diagnosis and often this has an adverse effect on subsequent management. Patients in the current climate have often already gathered information about their presumed diagnosis on the internet and it can be difficult to counsel them about other possible causes for their dizziness.

Classical Ménière’s disease can be diagnosed on clinical grounds with simple audiometric tests as an adjunct. The diagnosis can often take time and the patient should ideally be seen in a dedicated ‘vertigo’ clinic by a consultant with a special interest in the management of such problems.

Clinical features
The disease characteristically consists of episodic vertigo with associated tinnitus and reduced hearing on the affected side. This may be preceded by fullness in the affected ear up to 20 minutes prior to an attack. The vertigo typically lasts ‘minutes to hours’ which in itself is a clue to the diagnosis. It is not precipitated by head movement and does not cause the patient to lose consciousness. The hearing loss is typically low frequency and may not be noticed by the patient in the initial stages of the disease. It will progress to a permanent sensori-neural hearing loss over time with repeated attacks. This loss will continue to fluctuate as the disease relapses and remits.

The vertigo is often so disabling that the other specific symptoms of tinnitus and hearing loss must be specifically asked for in the history as the patient often dismisses them as insignificant.

To try to introduce some uniformity to the diagnosis and management of Ménière’s disease the American Academy of Otolaryngology–Head and Neck Surgery has issued guidelines for evaluating treatment. They are based on control of vertigo, disability and effects on hearing. The most recent version was revised in 1995. It sets out clear definitions of what can and cannot be diagnosed as Ménière’s disease as well as being a tool to assess the functional impairment of the patient. In this way it can be used to quantify the effects of treatment on the course of the disease.

Clinically three stages are recognised:4

Stage I: In the early stages the predominant symptom is disabling vertigo. This is usually rotatory and there is associated nausea and vomiting. The episode may be preceded by aural fullness/presence sensation in the affected ear. The episode typically lasts 20 minutes to several hours. Between attacks the hearing reverts to normal in the initial stages of the disease.

Stage II: As the disease advances the hearing loss becomes more established although it continues to fluctuate. The deafness is sensori-neural and has a predilection for the lower frequencies. The vertiginous episodes reach a maximum and then begin to abate. The pattern of relapse and remission is extremely variable and no two patients will experience the same course of the disease.

Stage III: The hearing loss becomes progressively more severe and the prime disability becomes deafness. In long-term studies 50% of patients will have bilateral disease so many patients will lose all serviceable hearing in both ears. The vertigo diminishes although a residual unsteadiness may remain.

It is important to recognise the change from vestibular over activity to sensory loss, as this will affect what treatment remains appropriate for the patient.

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Differential diagnoses and investigations
Classical Ménière's disease can be diagnosed on clinical grounds with the addition of some simple audiometric tests. However, it is not always easy to elicit an accurate history from these patients, as it is difficult for them to describe their symptoms. A detailed history must be sought, as this will give the main clues to the other possible causes for the vertigo. The vertigo is often so disabling and all-consuming that the patient will not mention associated symptoms unless specifically questioned.

Pure tone audiometry will be normal in the initial stages of the disease unless the patient is acutely symptomatic in which case a low frequency loss may be documented. The audiogram does deteriorate during the course of the disease but fluctuations in thresholds are commonly seen.

There are two other investigations that may help with the diagnosis:

Glycerol Dehydration Test
This measures the audiometric response to an oral dose of glycerol. This lowers plasma osmolality and improves pure tone thresholds and speech discrimination exclusively in Ménière's disease. The test is used less frequently now but may be an indicator of potential response to saccus surgery.

Electrocochleography
This gives a characteristic waveform in the presence of endolymphatic hydrops but may be negative in the early or late stages of the disease. It can also be technically difficult to obtain good diagnostic tracings that are reproducible.

The differential diagnoses are many:
• Benign paroxysmal positional vertigo
• Ménière's disease
• Secondary endolymphatic hydrops
• Vestibular neuronitis
• Head injury
• Infective labyrinthitis
• Labyrinthine fistula
• Vertebrobasilar insufficiency
• Otological surgery
• Cerebellopontine angle tumours or vascular lesions

Many of these have their own distinguishing features in the history that enables the clinician to differentiate the numerous aetiologies.

Any patient with a unilateral nature to his symptoms must have a MRI scan to exclude the possibility of a space-occupying lesion. The scan will also reassure the majority of patients that their vertigo is not due to a life-threatening disorder.

Management
No treatment has been shown to alter the long-term course of the disease. This could be due to one of a number of reasons:
• The disease's unknown aetiology
• The spontaneous remission rate of 60-70%
• The relapsing and remitting nature of the disease
• The significant placebo effect of many of the treatments

Despite this, masterly inactivity is not an option and we are obliged to attempt to control our patients' often disabling, symptoms.

Psychological
Management begins with a detailed explanation of the condition to the patient and an outline of the expected course of the disease. This is therapeutic in itself. The most debilitating factor that a clinician has to address is the 'fear' of dizziness that many of these patients have which prevents them from leading a normal daily life. Those who allow the vertigo to dominate their daily activities are those who are more likely to require surgery and have a poorer outcome in the long term.

The acute episode
There are a number of drugs that are capable of sedating the vestibulo-brainstem axis. These remain the mainstay of treating an acute episode of vertigo. Drugs such as prochlorperazine, promethazine, cinnaron and diazepam have all been used to good effect. The drawback is that they cannot be used in the long term due to their sedating effects and their risk of extra-pyramidal side-effects, especially in the elderly.

Maintenance therapy
This consists of dietary advice such as the avoidance of salt and the restriction of vasoactive substances such as caffeine. The former is based on the evidence that in animals, salt restriction is shown to alter Na+K+-ATPase activity in the stria vascularis. Whether this occurs in humans is debatable. The latter is based on the positive association of migraine with Ménière's disease and that restriction of vasoactive substances may confer some additional benefit.

Drug therapy consists of the use of thiazide diuretics. Clinical evidence for the benefit of this class of drug is sparse but they are shown to have a definite placebo effect.

In animal models betahistine produces vasodilatation of the stria vascularis and the spiral ligament and reduces endolymphatic pressure. Clinically it has been shown to confer benefit in terms of vertigo and hearing thresholds.

An alternative conservative management strategy is using the Meniett device. This device applies micropressure pulses to the inner ear via a tympanostomy tube. A small study of 10 patients showed that all reported a significant improvement in their vertigo. There were no adverse effects reported and there was a statistically significant improvement in hearing across the group. Other small studies have reproduced these findings. Further work will need to be done with larger numbers of patients but these preliminary results are encouraging.

Vestibular ablation
Systemic aminoglycosides are known to be toxic to the neuroepithelium of the inner ear. In the past, systemic administration of streptomycin was used to perform a chemical labyrinthectomy. Unfortunately the side-effect profile of almost certain associated cochlear toxicity, ataxia and oscillopsia was unacceptable.

More recently much work has been done on the instillation of intratympanic Gentamicin. Many studies have been carried out on the outcomes of intratympanic Gentamicin. Administration varies from low dose to high dose, continuous infusion to single injection treatment. Method of administration also varies from intratympanic needles to the use of a MicroWick to deliver the drug. Most studies show a significant improvement in vertigo – up to 90% in some studies.
The main side-effect is cochlear damage with a significant hearing loss occurring in up to 30% of patients. Most researchers report that the degree of hearing loss does not seem to correlate with the dose of Gentamicin used. A separate study showed a coincidental improvement in tinnitus in 28% of patients.1

**Surgical Treatment**

Since 70% of patients will enter a period of sustained remission this leaves at least 25% of patients who will still have significant episodes of vertigo. This is the group in which surgery must be considered. The decision to operate and which procedure to use will depend on the individual surgeon's experience.

Patient factors must also be considered such as the effect on lifestyle, employment, hearing levels, severity of the vertigo, presence of bilateral disease, physiological age and general health.

Procedures can be divided into hearing preservation and those destructive to hearing.

Non-destructive procedures include:
- Insertion of tympanostomy tubes
- Endolymphatic sac surgery
- Vestibular nerve section
- Sacculotomy
- Ultrasound treatment
- Cryosurgical treatment
- Cervical sympathectomy

The latter four procedures are now rarely undertaken.

Destructive procedures include:
- Labyrinthectomy
- Cochleosaccusotomy
- Vestibulocochlear neurectomy
- Translabyrinthine vestibular neurectomy

The simplest procedure is to simply insert a tympanostomy tube into the affected ear. There is no explanation for its efficacy other than its placebo effect.

Endolymphatic sac surgery remains controversial. First described in 1927, the precise mechanism by which relief of symptoms occurs is still unknown. The success rate remains at around 80% and has been reproducible in many centres. Hearing may also improve in up to 20% of patients in the early stages of the disease.

Vestibular nerve section does not alter the underlying pathophysiology of the disease it merely blocks abnormal vestibular signals from reaching the brain. Its reported success rate is up to 95%. It remains a major neuro-otological procedure with the associated risks of a posterior fossa craniotomy. It may be used as a primary intervention or after failed endolymphatic sac surgery.

Labyrinthectomy is used in those patients with little or no serviceable hearing. It is the most reliable method of preventing vertigo. The procedure leads to total permanent deafness. The dilemma remains that if followed up for long enough 50% of patients will develop hydrops in the other ear. This could result in bilateral total deafness. The argument remains that using non-destructive interventions should preserve any hearing however poor. It remains that whilst vertigo can be controlled in the long term, the deafness is the most disabling feature.

**Cochlear Implantation**

Bilateral end-stage Menière’s disease remains an indication for cochlear implantation due to the disabling effects of the profound sensori-neural hearing loss that results from the natural progression of the disease.

**Conclusion**

Menière’s disease remains controversial in every aspect. The aetiology and pathogenesis are unclear, the diagnosis is often difficult and inaccurate and the behaviour of the disease unpredictable. The efficacy of many of our current treatments is difficult to explain and control of vertigo is often at the expense of serviceable hearing.

However, most patients’ symptoms can be controlled to a satisfactory level and we should not let our lack of understanding detract from our aim to provide symptomatic relief from this disabling condition.

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References


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A Review of the Pathophysiology of Ménière’s Disease

The attacks of vertigo caused by Ménière’s disease are characteristic causing a feeling of rotation usually in the plane of the lateral semicircular canal. During the attack, there is a feeling of nausea associated with vomiting and sometimes diarrhoea. The shorter attacks last a few minutes whereas more violent attacks can last several hours. The attacks tend to occur in clusters during several weeks followed by variable periods of remission. At the onset of the disease the attacks are more severe but as the disease progresses the vertigo tends to become less distressing. Typically there is warning of an impending attack as the patient senses one or a combination of the following: a feeling of aural fullness, increased tinnitus, and/or a change in hearing in the affected ear. During the attack, the tinnitus may alter its pitch and the feeling of aural fullness may gradually lessen. The hearing does not change noticeably although after the hearing may improve.

Ménière’s disease can be classified into three main stages on the basis of the audiogram. In early (stage one) Ménière’s disease the audiogram is normal between vertigo attacks. In stage two, the audiogram shows a sensory loss which fluctuates especially in the lower audiometric frequencies. Eventually a ‘burnt out’ or stage three is reached when the audiogram shows a fixed sensory loss of at least 60dBHL and the attacks of vertigo cease.

What is the relationship of endolymphatic hydrops to Ménière’s Disease and the attacks of vertigo?

In 1938, Halipike and Cairns in England and Yamakawa in Japan published temporal bone studies showing endolymphatic hydrops (ELH) and fibrosis of the endolymphatic sac (ELS). Subsequently it has been shown that ELH is inconsistently present in early cases of Ménière’s disease and that some ELH in the apical region of the cochlea is encountered commonly at autopsy of non-Ménière’s ears. Although ELH can be experimentally induced in guinea pigs, these animals do not suffer attacks of vertigo.

How is endolymph produced and absorbed?

The original theory of endolymph circulation was a ‘longitudinal theory’ with endolymph secreted by the stria vascularis and then absorbed in the ELS. Dysfunction of the ELS was widely thought to be a contributing cause of ELH. Studies in guinea pigs showed that ELH occurred consistently after removal of the ELS but higher mammals, such as a monkey rarely develop any ELH after removal of the ELS even when examined several years after the surgery.

Next, a radial theory of endolymph circulation was proposed and the ELS was considered a vestigial structure. This theory proposed that the endolymph was both secreted and absorbed by the stria vascularis. This theory began to waver when the detailed histology of the ELS showed complex activity and studies showed that foreign materials were transported into the sac for removal. Subsequently a combination of the longitudinal theory and the radial theory was proposed. It was still widely believed that endolymph circulated and the endolymph fluid was frequently replenished.

Finally the issue has been settled due to the work of Salt and colleagues. Using dye iontophoresis into the cochlear duct, they have shown that endolymph is essentially a ‘biological puddle’. Their studies clearly show in guinea pigs, that no flow of endolymph fluid occurs. The water component remains static while the stria vascularis continually replenishes the ionic composition. Longitudinal flow only occurs if excess fluid is injected into the cochlea duct.

What is the role of the endolymphatic sac?

The endolymphatic sac can no longer be considered vestigial as research over the last decade has revealed its complexities. The ELS is the only part of the inner ear that continues to develop after birth. The human ELS forms a single lumen, as in guinea pigs up to one year of age, however it rapidly evolves canicular structures and assumes the adult shape by three or four years of age. The term ‘sac’ is a misnomer as the structure has no empty centre but has a ‘sponge-like’ structure. The ELS is the only part of the inner ear which has a macrophage role. Cells with both secretory and absorptive functions have been identified. But the most fascinating discovery has been the intermittent presence of glycoprotein within the ELS. Glycoprotein is an intensely hydrophilic substance and it can ‘ suck out’ the contents of cells but the cells of the ELS have an ability to withstand this substance. The secretion of glycoprotein by the ELS seems to be related to longitudinal flow and may only be needed on special occasions when the endolymph volume needs adjustment. It is not known what triggers the secretion of glycoprotein but natriureptides have been identified in the cochlea and pressure receptors are seen on the intrinsic surface of the round window membrane. In animals with experimentally induced ELH, ‘ dark cells’ proliferate in the stria vascularis. Whether these cells actually produce endolymph or whether these cells are trying to remove the excess endolymph is unknown.

What is the relationship of an abnormal summing potentials recorded using electrocochleography and Ménière’s disease?

In 1977, Gibson and co-workers were the first to report that electrocochleography showed large negative summing potential (SP) in ears affected by endolymphatic hydrops. Originally this was shown by comparing the ratio of the SP to the action potential (AP). An SP/AP ratio of over 35% was considered significant. Gibson et al believed that the SP was increased abnormally but this theory was disputed by Eggermont, who believed that the SP had not increased but had remained unaffected while the AP had decreased. Subsequent studies using tone burst recordings rather than clicks finally settled the dispute showing that the SP definitely was increased. Studies which bias the basilar membrane using low frequency tones support the hypothesis that the increased SP is caused by ELH.
shifting the basilar membrane towards the scala vestibuli (Figure 1). Increased tone burst SP can be recorded reliably using transystympnic electrode placement and an increased 1kHz SP appears to be the most diagnostic frequency (Figure 2). Electrocochleographic changes before, during and after attacks of vertigo suggest that there is an increase in endolymph volume prior to attacks of vertigo, a gradual decline in SP during the attack of vertigo, and a variable increase after each attack. There are no clear changes in the size of the compound action potential (AP) during attacks of vertigo.

What causes the attacks of vertigo which occur with Ménière’s disease? Various theories have been postulated to account for the vertigo. The purpose of this review is to examine the evidence for each theory in respect to the pathophysiology, which has been discussed.

The rupture theory
Schuknecht and his colleagues proposed this theory after discovering histological evidence of Reissner’s membrane ruptures. This hypothesis envisages that an increase in the volume of endolymph leads to ruptures of Reissner’s membrane allowing the potassium-rich endolymph to mix with the sodium-rich perilymph which bathes the afferent nerve fibres. The potassium paralyses the nerves causing the attack of vertigo until electrolyte pumps within the inner ear can reconstitute the correct electrolyte levels while the ruptured membrane rapidly repairs itself.

The rupture theory remains a favourite for clinicians but serious flaws are evident:
1. Ruptures have only been shown in advanced cases of Ménière’s disease. Furthermore these ruptures only appear to occur in the cochlea which is distant to the labyrinthine sites which cause vertigo.
2. Experimentally even small ruptures lead to some loss of hair cells. After repeated severe attacks in early Ménière’s some evidence of hair cell damage should be evident.
3. Ruptures would lead to loss of the endolymph potential and should cause total deafness. The hearing is usually worse prior to the attacks and remains the same during the attacks or may even improve in rare cases (Lermoyez syndrome).
4. Electrocochleography during the attacks would suggest that there is no loss of neural activity but that the ELH is gradually decreasing.

The chemical theory
Although it may be conceded that ruptures are an unlikely cause of the vertigo, the concept of potassium intoxication of the perilymph remains in various chemical theories. This theory suggests that transitory endolymph leakages occur because of mechanical stresses on the tight junctions. Other theories suggest that there is a sudden failure of the pumps within the cochlea. The main objection remains that potassium intoxication should cause increased hearing loss unless it is totally confined within the pars superior (labyrinthine portion) and that this theory does not comply with the electrocochleographic findings.

The drainage theory
This theory suggests that periods of enhanced longitudinal endolymph flow relate to the attacks of vertigo. The concept is that there is a normal physiologic mechanism which can detect an increase in endolymph volume and initiate longitudinal flow towards the endolymphatic sac so the excess fluid can be absorbed. It is proposed that Ménière’s disease occurs when the mechanism becomes damaged allowing endolymphatic hydrops to build up in the ear before triggering longitudinal flow. This concept is that there is a critical endolymph volume or pressure that initiates endolymph flow and that in Ménière’s ears excessive endolymph has accumulated prior to the attack.

When the mechanism is triggered the endolymph flows longitudinally through to the sac rather like sand passing through an hour glass. Movement of endolymph is the cause of the vertigo. The severity of the vertigo is less than that of benign paroxysmal positional vertigo but more prolonged. In stage three Ménière’s disease, the ELS has lost its function, perhaps due to the repeated glycoprotein production, and there is permanent ELH, as seen in guinea pigs after removal of the endolymphatic sac, but the attacks of vertigo cease or become trivial.

In conclusion
This review highlights some of the more recent discoveries and questions some of the longstanding concepts of the pathophysiology of Ménière’s disease. It is clear that until the pathophysiology of Ménière’s disease is understood, there can be little rational progress in the treatment of this disabling disorder.
References


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Living with Ménière’s Disease – What Helps?

In October 2000 a three year study of factors influencing quality of life in Ménière’s disease was initiated with the joint support of the Ménière Society (a support group for people with Ménière’s disease) and the UK Economic and Social Research Council. The three symptoms of Ménière’s disease – vertigo, hearing loss and tinnitus – are known to have the potential to impact on quality of life but there has been surprisingly little research into their combined impact in Ménière’s disease. The aim of this study was firstly to assess what factors were associated with a better or worse quality of life. This would allow us to identify the characteristics of sufferers that might make them more vulnerable to the impact of Ménière’s disease, and therefore in greater need of support. Secondly, we hoped to identify factors that could lead to improving or worsening quality of life over time.

The study
The findings reported here are based on two different measures of quality of life. The first was the widely used SF-36, which assesses objective functioning (e.g. whether the respondent can walk a mile) and subjective functioning (e.g. whether the respondent feels that they can carry out their normal roles). The second was a new scale we constructed that assesses whether respondents feel that they are moving towards or away from the goals that are important to them (e.g. at work or at home). The factors that we thought might influence quality of life included: a) Illness factors (severity of symptoms of vertigo, tinnitus, hearing loss and fullness in the ear, duration of symptoms); b) Situational factors (e.g. age, gender and occupational status); c) Personal factors (optimism, self-esteem and a feeling of control over the illness); and d) Support factors (support provided by family, health professionals and the Ménière Society). All these factors were assessed by a set of validated questionnaires, which were administered to 1000 randomly selected members of the society. Follow-up questionnaires were sent 10 months later to those who were willing to complete them.

A total of 550 people (211 men, 296 women) completed the initial set of questionnaires and 370 people completed the follow-up questionnaires. The average age of the initial sample was 60 with a range of 21 to 86 years, and 90% had been diagnosed with Ménière’s disease by an ENT specialist. The average duration of symptoms was 13 years.

The findings
The impact of Ménière’s disease on the eight aspects of quality of life assessed by the SF-36 is shown in Figure 1, with mean scores for a healthy and chronic illness population for comparison. The Ménière’s disease and chronic illness samples had very similar scores for physical functioning, physical and mental health, social functioning, pain and energy. However, Ménière’s disease appears to have a greater impact than other types of illness on the ability to perform one’s role, or tasks associated with one’s role, both physically and emotionally (see scores for R(E) and R(P)). This may be because the unpredictable and uncontrollable nature of the vertigo results in greater interference with lifestyle - sufferers feel they have to avoid many activities in case they provoke or experience a sudden attack, even if their symptoms would not currently prevent them from undertaking these activities.
The factors associated with a better quality of life are shown in Box 1 (a more detailed analysis is reported elsewhere). All symptoms contributed to a worse quality of life, but severity of vertigo had the greatest impact. Unsurprisingly, those with greater resources (socioeconomic, support from family and health professionals, personal confidence and optimism) had a better quality of life, indicating that additional support should be directed to those who do not.

At follow-up, mean scores on the SF-36 scales assessing functioning had not changed. Despite this, some adjustment to illness seemed to have occurred, since mean scores on the scale assessing goal-oriented quality of life had changed from a perception of moving away from personal goals to a perception of moving (slowly) towards personal goals. Box 2 shows the factors that predicted improving or worsening quality of life over ten months. Severity of symptoms at baseline had no effect on whether quality of life improved or worsened, but several psychosocial predictors were identified. Greater personal resilience and social support led to a better quality of life (as has been observed in other types of chronic illness). Our findings also suggested that membership of the Ménière's society may be beneficial, since quality of life improves following a longer period of membership (but was unrelated to duration of the illness). However, there was evidence that those who made a great deal of use of society resources (e.g. reading the society magazine from cover to cover, writing letters to the society, joining local groups) actually experienced a deterioration in their quality of life. Only a small minority of members did make great use of society resources, and their excessive focus on the illness may reflect personal difficulties in finding better coping methods.

**Conclusions**

It was encouraging to find that over time most respondents felt that they were more able to progress towards the goals that were important to them, even though the impact of the disease on their lifestyle had not reduced. This finding suggests that people with Ménière's disease might be supported in the process of adjustment by assistance in setting goals that they feel they can realistically work towards. Our findings suggest that those who may be particularly in need of support (e.g. from health professionals or from the Ménière's Society) are those who have worse symptoms, less good social and economic circumstances, lower personal confidence and optimism, and less family support.

**References**