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Navigating the challenges of Meniere disease

By Richard L. Pullen, Jr., EdD, MSN, RN, CMSRN

CHRISTY KRAMES

AN ESTIMATED 10 to 150 per 100,000 persons have Meniere disease (MD).[1] Various organizations put the number at around 615,000 people in the United States.[2-4] It's characterized by ear pressure or fullness, tinnitus, fluctuating hearing loss, and vertigo. Although usually unilateral, it can affect both ears and its underlying etiology is unknown.[1-4]

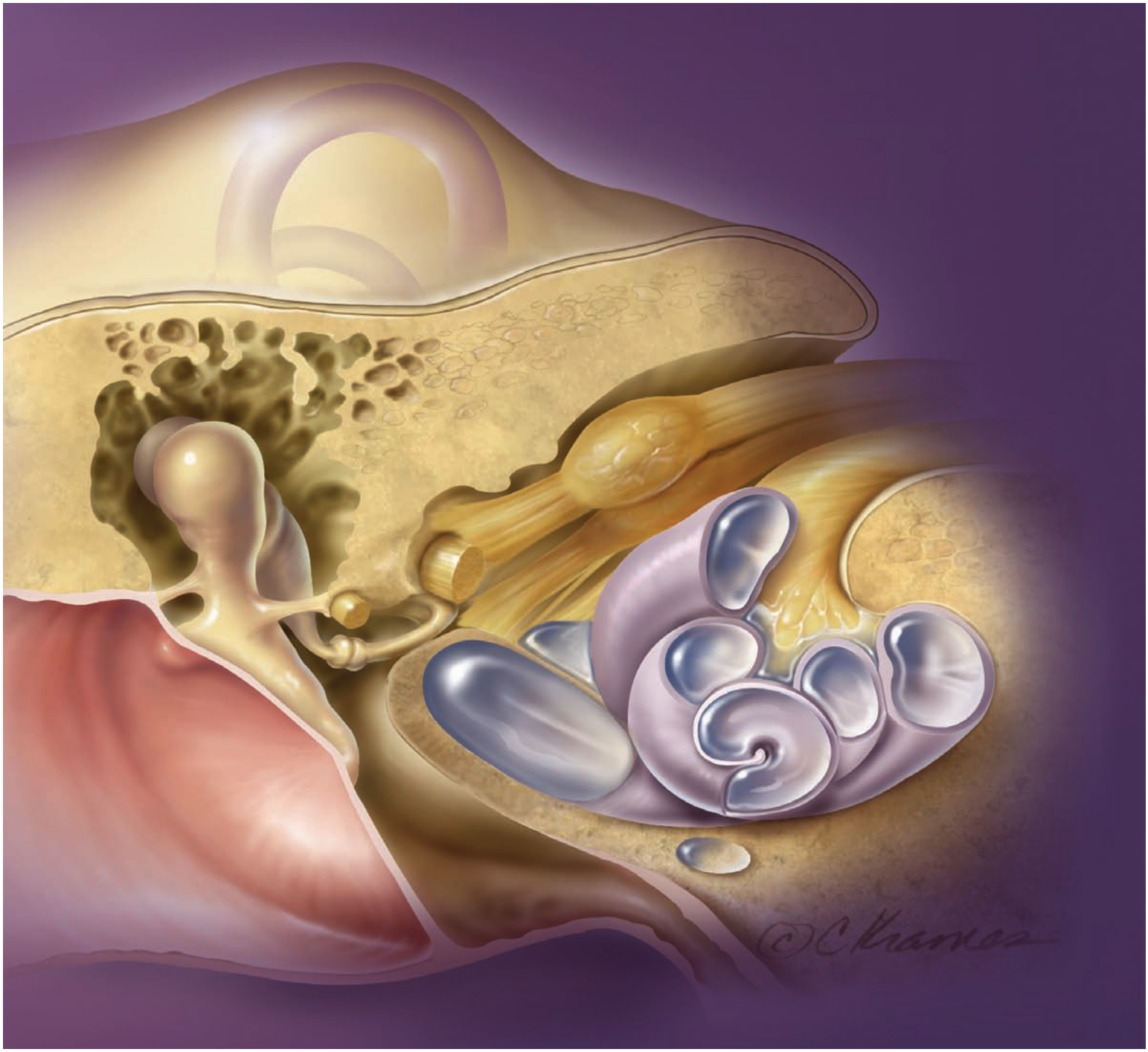
To help the patient with MD achieve an optimal quality of life, nurses need an understanding of MD's pathophysiology and treatment approaches. This disease's hearing and balance symptoms, especially vertigo, can cause significant morbidity. Managing the frequency and intensity of vertigo may be a challenge for some patients. A case study presented here describes a patient with MD who received intratympanic gentamicin (ITG) to manage intractable vertigo.

Pathophysiology

MD is a progressive disorder that leads to an accumulation of endolymph within the inner ear. (See *Looking inside the ear.*) It's not clear why excess fluid builds up in the endolymphatic spaces of the inner ear. Several theories include vascular disorders, viral infections, immunologic mechanisms, and a genetic predisposition.[2-4]

Hearing and balance symptoms occur when the normal volume of endolymph is interrupted.[5] Endolymph is stored and absorbed in the endolymphatic sac, which is believed to play an important role in inner ear immune responses.[6]

Overproduction, limited absorption, and obstructed flow of endolymph cause the endolymphatic sac to expand, leading to signs and symptoms of MD. Sometimes a distended endolymphatic sac is called *endolymphatic hydrops*, meaning increased



pressure within the endolymphatic system and dilatation of the endolymphatic sac.[5,7]

Another fluid in the inner ear is perilymph, an extracellular fluid with a high sodium and a low potassium concentration; it's entirely separate from endolymph. A thin membrane separates the perilymphatic and endolymphatic chambers. Endolymphatic sac distension increases tension on this membrane, causing a feeling of pressure or fullness, tinnitus, and a fluctuating sensorineural hearing loss in the affected ear.[5,7]

As endolymph continues to accumulate, the thin membrane separating the perilymph and endolymph rupture, and the two fluids mix. Because these are chemically different, the mixture causes an increase in the vestibular firing rate leading to vertigo.[5,8,9]

Clinical manifestations

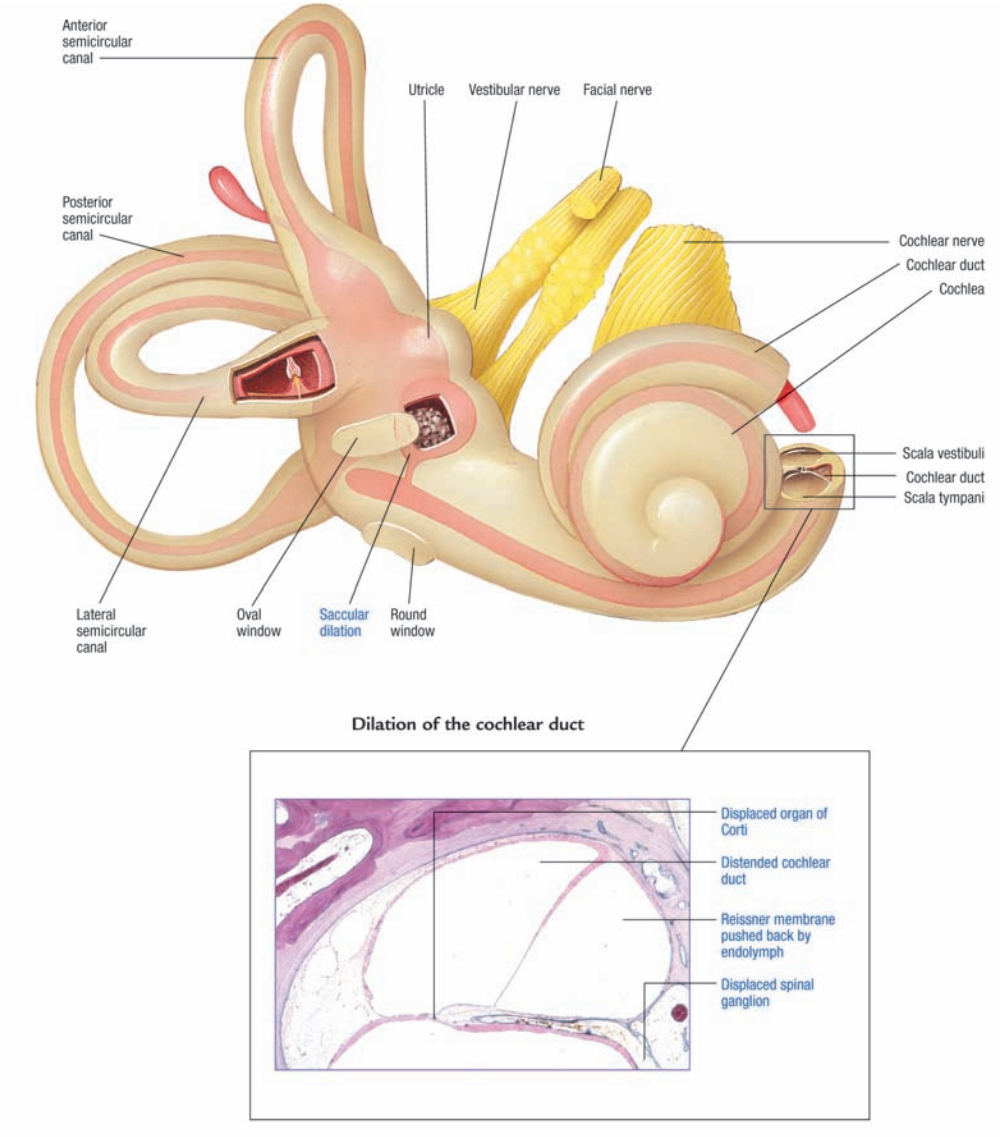
The classic symptoms of MD may present serially or simultaneously and include:

- ear pressure or fullness
- tinnitus, which may be described as a humming, ringing, whistling, and hissing sound of various pitches and tones
- fluctuating hearing loss that affects the lower frequencies, sometimes called low-tone deafness.[1]
- vertigo.[10]

Vertigo is the most distressing, incapacitating, and debilitating symptom of MD.[5,7,11] Significant balance and disequilibrium symptoms are also often present in MD.[7] Vertigo is usually rotational, which means the person feels as if the room is spinning around him or her. It's often accompanied by nausea and vomiting.[12]

Looking inside the ear

MD may result from overproduction or decreased absorption of endolymph—the fluid contained in the labyrinth of the ear. Accumulated endolymph dilates the saccule and cochlear duct. Dilation of the endolymphatic system occurs and the Reissner membrane often tears. When rupture of the membrane causes endolymph to escape into the perilymph, this increases the vestibular firing rate, leading to mild to severe vertigo.



Source: Anatomical Chart Company. *Atlas of Pathophysiology.* 3rd ed. Philadelphia, PA: Wolters Kluwer Health/Lippincott Williams and Wilkins: 2010:424-425.

Assessment and diagnosis

A focused health history and physical assessment should include current medications, comorbidities, and assessment of hearing and neurologic function.[12]

MD can't be diagnosed with one specific test. The clinical diagnosis in most patients is based upon the history, neurotologic evaluation, and clinical response to medical management.[1]

MD is diagnosed when a person has at least two spontaneous episodes of vertigo, sensorineural hearing loss that's confirmed through audiometric studies, and tinnitus. Audiometry should be performed in all patients with suspected MD to determine hearing deficits. Patients with MD often have a low-tone hearing deficit. Patients may also have problems with speech discrimination; this is, they may have trouble distinguishing similar sounds. For example, they may not be able to distinguish the words *sit* and *fit*, and the words *day* and *bay*.[10,12]

Electronystagmography (ENG) evaluates the vestibular system to determine if balance problems originate from the brain (central nervous system) or inner ear (peripheral nervous system). The ENG may also be performed using enhanced technology called *video electronystagmography* (VNG), which uses video cameras to record eye movements. ENG and VNG record and quantify nystagmus whether it's spontaneous or induced. *Nystagmus* is a rhythmic oscillation of the eyes, analogous to a tremor in other parts of the body. Many specialists use these techniques for assessing vestibular and ocular mobility.[13]

A part of the ENG is a caloric test, which involves introducing cool and warm water into the ear canal.[7] The water increases the flow of endolymph to induce nystagmus and vertigo.

Nystagmus has multiple etiologies including disorders of the labyrinth and the cerebellar system. The movement of nystagmus may occur in one or more planes. (See *Understanding the plane of the movements.*) It's the plane of movements, not the direction of gaze, that defines the movement of nystagmus.[14]

Electrocochleography (ECOG) may be used to measure ear pressures. Specifically, it detects distension of the basilar membrane in the inner ear that leads to endolymphatic hydrops. Ear pressures are measured and translated into a waveform for analysis.[7] The ECOG helps diagnose MD by focusing on the amount of hydrops in the endolymphatic sac.[5,12]

Auditory brainstem response (ABR) testing measures the electrical activity of the brain related to hearing and balance.[5,12]

The symptoms of MD may imitate other disease processes. Imaging studies such as magnetic resonance imaging can be used to rule out acoustic neuroma, stroke, multiple sclerosis, or Arnold Chiari malformation. The patient's health history and lab values should be evaluated to rule out the presence of infections or metabolic imbalances.[1,5]

Treatment strategies

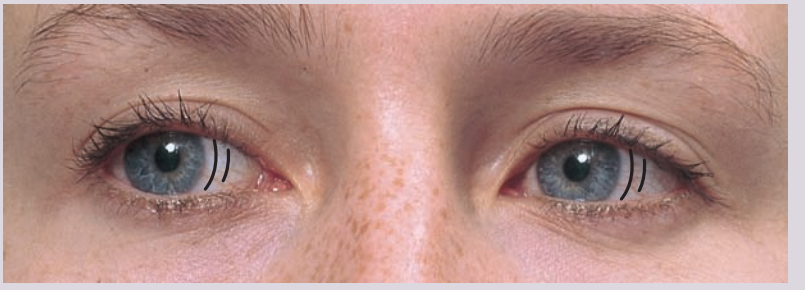
The main goals of treatment in MD are to improve and stabilize fluid volume in the endolymphatic sac, preserve hearing, control vertigo, and help patients optimize their quality of life.[7,12,15,16]

The goal of first- and second-line treatment is to improve fluid balance in the endolymphatic sac.[15] The dearth of randomized controlled trials makes it difficult to determine the best treatment for MD.[1]

- **Stress reduction.** Stress triggers an exacerbation of hearing and balance symptoms in MD. Stress reduction techniques include meditation with deep breathing, yoga, and exercise.[5,15]
- **Nutrition.** A restricted daily sodium intake of 1,000 to 2,000 mg decreases edema and lowers pressure in the endolymphatic sac to reduce the risk of membrane rupture. Sodium restriction is commonly recommended as part of initial therapy, although data supporting its efficacy aren't available. Patients should also avoid other substances that increase endolymphatic sac pressure such as alcohol, nicotine, caffeine, and foods high in sugar. Because they're vasoconstrictors, caffeine and nicotine can affect the flow within the labyrinthine system. Alcohol can shift fluids and electrolytes inside the inner ear. Hypoglycemia, which may be a trigger, should also be avoided.[1,5,15]

Understanding the plane of the movements

The movement of nystagmus may occur in one or more planes (that is, horizontal, vertical, or rotary). It's the plane of the movements, not the direction of the gaze, that defines this variable. Horizontal nystagmus is shown below.



Source: Bickley LS, Szilagyi PG. Bates' *Guide to Physical Examination and History Taking.* 11th ed. Philadelphia, PA: Wolters Kluwer Health/Lippincott Williams and Wilkins; 2013:756.

• **Diuretics.** Hydrochlorothiazide with triamterene (prescribed most commonly), acetazolamide, spironolactone, and furosemide are diuretics used to help decrease excessive endolymphatic fluid volume. Monitor patients' serum electrolytes, especially sodium and potassium levels, and advise patients to increase their intake of fluids, especially water, to avoid dehydration.[5,15] For many patients, restricting sodium intake and instituting diuretic therapy are sufficient to control hearing and balance symptoms without any further interventions.[5,15] Aggressive treatments such as corticosteroid therapy and positive pressure therapy are initiated when the patient experiences worsening hearing or an increase in the frequency and intensity of vertigo or both.[15]

• **Corticosteroids.** These medications are used to reduce edema in the endolymphatic sac. Short-term oral corticosteroids may be prescribed or, to avoid systemic adverse reactions, intratympanic corticosteroids may be infused into the affected ear. Monitor sodium, potassium, and blood glucose levels and white blood cell counts and assess for fluid retention along with changes in mental status. Adverse reactions are usually limited with short-term use.[5,15]

• **Positive pressure therapy.** For this therapy, a positive pressure therapy device is placed in the external ear to generate a sequence of low-pressure (micropressure) pulses. Fluid exchange in the inner ear may be improved by applying positive pressure to the middle ear. These pulses are transmitted to the vestibular system of the inner ear to reduce endolymphatic pressure. A tympanostomy tube is inserted to deliver positive pressure.[1,5,15]

When vertigo is severe and reduces the quality of the patient's life, an endolymphatic sac decompression or shunting or both may be considered.[5,15] This approach is called *nondestructive* because it doesn't destroy the auditory and vestibular systems. Efficacy in trials indicates that the endolymphatic sac decompression or shunting is 75% to 80% effective in reducing or terminating vertigo.[1]

Destructive treatments include ITG, vestibular neurectomy, and labyrinthectomy, which may be considered to control vertigo when all other approaches have failed. With these aggressive approaches, the vestibular system is chemically or surgically destroyed in an attempt to control vertigo.[5,8,15,17,18]

Patients with MD may be helped by using hearing aids, engaging in vestibular rehabilitation, and performing exercises. Exercise activities can improve balance and retrain the central nervous system to compensate for the disequilibrium of MD.[1]

Vertigo's impact

Patients may experience vertigo that's mild to severe. For example, patients may experience a slight whirling sensation or violent episodes that make them feel as if the room is spinning around them (rotational vertigo) and they can't get off the floor. Spinning sensations can vary in speed. When the "spin" starts, vomiting is likely to follow. Less frequently, patients have the sensation of spinning around the room. Patients may experience an increase in tinnitus, hearing loss, fatigue, nausea, pallor, diaphoresis, headache, and a feeling of uneasiness before a vertiginous event.[5,9,16,19]

Nystagmus may begin and move rapidly toward the affected ear in MD.[5] Some patients feel the rapid jerky movement of the eyes. During the recovery phase, nystagmus may move away from the affected ear. The direction of nystagmus can vary.[5]

During violent vertigo, the patient instinctively lowers the head and limits head movement. Any attempt at moving the head may cause it to drop forward or to bob from one side to the other because the balance system is under attack. Any head movement will increase the spin, which leads to more vomiting and imbalance. Because the patient is totally incapacitated, he or she should be positioned in a safe place, such as in bed or on the floor.[5,9,16,19] Cover the patient with a blanket and provide a pillow for warmth and comfort.

The acute phase of violent vertigo may last 1 or 2 hours.[5,7,11,12] During the recovery phase, the patient will be exhausted and may sleep for hours.

After the vertigo has subsided, the patient feels very weak and off balance. Standing isn't safe and the head must be kept low to minimize pressure in the endolymphatic sac that's causing vertigo. Raising and moving the head causes vertigo to worsen. The patient may have the urge to void as recovery continues, but sitting on a commode or standing to void isn't possible because the head would be in a high position. If necessary, the patient can void while on the floor on hands and knees with a pan placed into position.[5,9,16,19]

Soon after, when it's safe and strength returns, the patient will try to stand. The gait is unsteady at first. The patient may use his or her hands to hold onto the wall to avoid falling and instinctively keep the head from moving too much.[5,9,16,19]

Most weakness during the recovery phase stems from the neurologic impact of the vertiginous event. This is similar to the postictal stage of a grand mal seizure or following a migraine headache. Antivertigo medications also contribute to the weakness. Gait unsteadiness may continue for a day

or two. Encourage the patient to walk as much as possible to retrain the vestibular system.[5,7,9,11,12]

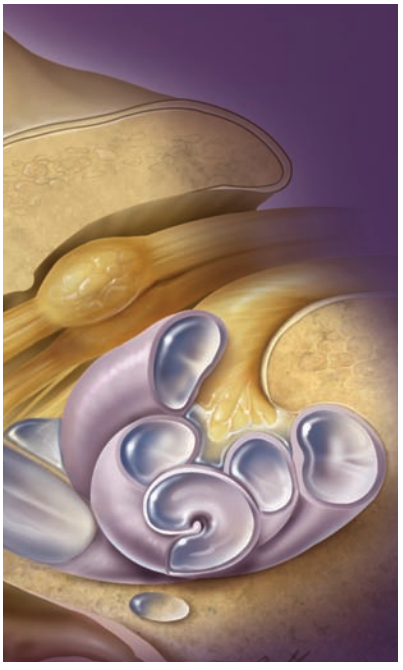
Vertigo is an incapacitating and frightening event for the patient. Medications are needed to decrease the vestibular response to avert, reduce, or terminate vertigo and manage nausea and vomiting. Examples of rescue medications include transdermal scopolamine, meclizine, diazepam, and promethazine. Patients with MD must keep these medications with them at all times.[5,9,10,15,20]

Patient teaching

Patients who have vertigo may feel anxious because they don't know when another episode will occur. The frequency and intensity of vertigo impacts activities of daily living and home life, work, and driving. Other activities such as swimming alone or working on a roof should be avoided.

To prevent or manage vertigo, teach the patient to:[9,15,19,20]

- Always wear a medical-alert bracelet that says: "Meniere disease: Vertigo."
- Always carry antivertigo (rescue) medications.
- When vertigo begins, stop any hazardous activities and get to a safe place.
- Take medications as soon as you have warning (prodromal) signs such as a feeling of uneasiness, an increase in ear pressure, tinnitus, hearing loss, nausea, and diaphoresis. Prodromal signs often provide you with sufficient time to get to a safe place. For example, when driving home from work, if vertigo begins then you must pull the car over to the side of the road, turn the car off, and call someone to let them know where you are if you can.
- Lie down with your head elevated slightly on a small pillow. Look straight ahead with your eyes open and fixed on a stationary object. Closing your eyes tends to increase the spin in vertigo.



In MD, excess fluid builds up in the endolymphatic spaces of the inner ear.

- Don't eat or drink anything because this is likely to increase vomiting and creates a risk of aspiration.
- Stay as calm as you can, and reassure those around you.
- You'll also feel your eyeballs "jumping" (nystagmus) when the vertigo is severe and violent. Keep your eyes open.
- Rest or sleep as much as needed after the vertigo is mostly over. Don't try to get out of bed until you feel well, and then keep your head still.
- When you awake, try sipping some water or other fluids. You'll be very weak, and you may have a headache or blurred vision. When you fixate on objects, they may appear to be slowly gliding in one direction. This is a normal response while your balance system is recovering. Stay calm and patient.
- When you're ready to get out of bed, slowly raise your head. Slowly slide your legs off the bed and place your feet on the floor. Slowly attempt to stand while slowly raising your head. If you're strong enough and you have no vertigo, begin to walk slowly. Your gait will be very unsteady. Walk slowly. Each time you walk, your gait will improve.[5,9,16,19]

Case study

Mr. W, age 58, was diagnosed with MD in August 2004, when he developed an acute onset of pressure in his left ear. He said it felt as if he were on an airplane. Pressure and hearing loss persisted in that ear over the next 2 weeks. Audiometry and ECOG were abnormal in the left ear. A sensorineural hearing loss in the left ear was evaluated and his eye, ear, nose, and throat (EENT) specialist diagnosed MD. His hearing was normal in his right ear. The patient was advised to eat a low-sodium diet to target the abnormal fluid and ion homeostasis in the inner ear.

Pressure and hearing in the left ear worsened, and the patient also experienced tinnitus in the left ear that he described as variable sounds such as hissing, a low-pitched hum, and low- and high-pitched ringing. Hydrochlorothiazide and triamterene, a diuretic, was prescribed to reduce fluid volume in the endolymphatic system. The patient was encouraged to increase water intake to avoid dehydration and electrolyte imbalances from diuretic therapy. Hearing, pressure, and tinnitus in the left ear failed to improve during the next month.

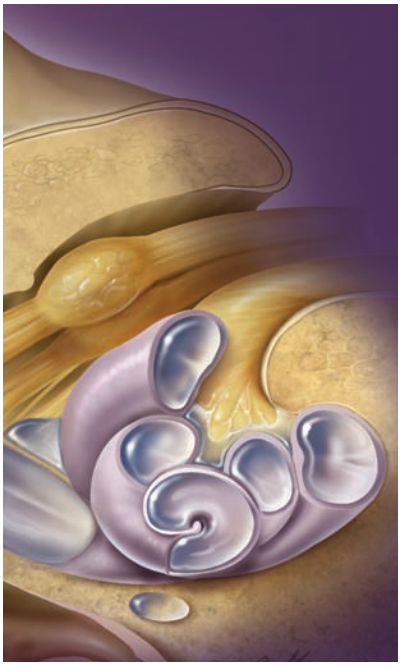
In November 2004, the patient had his first episode of vertigo while at the gym. He suddenly felt weak and the hearing in his left ear seemed to fade. Within seconds he experienced a whirling sensation with imbalance. He instinctively lowered his head and kept it still and quickly made it to the locker room. The room was now spinning clockwise.

He was on his knees with the head in contact with the floor. As the rate of spinning increased, he became nauseated and began to vomit. He was frightened, especially since this was his first experience with vertigo. The event was also frightening to bystanders at the gym.

He was taken by EMS to the ED, where he told them about his diagnosis of MD. An I.V. infusion of lactated Ringer solution was initiated. I.V. diazepam and promethazine were administered to suppress vestibular response and terminate the vertigo. I.V. dexamethasone was administered to suppress the immune system and to reduce inflammation in the endolymphatic system.

The patient had nystagmus toward his affected ear. The vertigo slowly ended over the next 3 hours. He was exhausted the next day, and he had some balance problems for several days. He said, "I had to retrain my legs to go in the right direction."

Mr. W had several additional episodes of violent and mild vertigo during the next month. He was placed on prednisone for 1 week. Then his EENT administered intratympanic dexamethasone into his left ear. However, corticosteroid therapy didn't effectively control the vertigo.



Vertigo is the most distressing, incapacitating, and debilitating symptom of MD.

In February 2005, Mr. W successfully underwent an endolymphatic sac decompression with shunting to reduce the severity of the vertigo. After that, he had no episodes of vertigo for nearly 8 years. However, violent and incapacitating vertigo suddenly returned in February 2013, with several more violent and mild episodes during the next 2 months. Mr. W said, "During this time, I felt as if I was always having vertigo, recovering from vertigo, or being anxious about when the next event was going to happen."

In April 2013, on the advice of his EENT, Mr. W decided to try ITG in his left ear to control the vertigo after bilateral audiometry and caloric testing was performed. (See *More about ITG*.) Mr. W's vestibular and hearing systems in the right ear were normal.

The patient understood the chance of ending the violent vertigo with treatment was 90%. He received five ITG treatments in his left ear over the course of 15 months. He still has some hearing in the left ear, and continues to experience near-constant tinnitus and pressure, but the ITG ended his violent and frequent vertigo. After a year-long period without vertigo, occasional mild episodes of vertigo returned.

The patient has good quality of life, although he has "imbalance issues" secondary to the ITG he describes as a mildly unsteady gait. He also has slightly blurred vision, especially when he's moving. The patient continues to have declining hearing in the left ear, which is consistent with MD. Hearing in his right ear continues to be normal. He averages one episode of mild vertigo every 2 to 4 weeks. This is acceptable to him. However, if vertigo increases in severity, he and his EENT may consider additional ITG or possibly vestibular neurectomy.

More about ITG[1,8,17,18]

Gentamicin is an aminoglycoside antibiotic that can be toxic to the kidneys and the vestibular and cochlear nerves. (This is why peak and trough levels are determined when a patient receives the medication for an infection.) The toxic effects on the vestibular nerves, however, are a benefit in the management of vertigo in MD. Gentamicin is delivered into the middle ear space by injection through a cannula to treat MD. It then destroys the vestibular nerves and hair cells in the semicircular cells. According to research studies, the procedure is 90% effective in reducing or terminating vertigo in MD while limiting hearing loss.

Sometimes the procedure is called a *chemical neurectomy*. ITG is the most commonly used destructive procedure to treat vertigo in MD. Tinnitus and ear pressure aren't improved by this procedure.

Patients may temporarily experience some imbalance issues after the procedure. The balance system in the unaffected ear must compensate for the loss of balance in the affected ear.

A final spin

MD is a challenging disorder that requires an astute interprofessional healthcare team to manage the patient's signs and symptoms. Safety is a concern for patients with MD,

especially when the patient experiences vertigo. Vertigo may occur anywhere and anytime. Nurses should listen to patients' fears about vertigo, teach them to manage their vertigo, and encourage them. Understanding the pathophysiology and management of MD will help nurses and the interprofessional healthcare team improve patients' quality of life. ■

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Navigating the challenges of Meniere disease

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