VESTIBULAR MIGRAINE COLLECTION

Your guide to understanding and coping with Vestibular Migraine.

ABOUT VESTIBULAR MIGRAINE

Migraine, a disorder usually associated with headache, is extremely common and can cause several vestibular symptoms. Approximately 40% of migraine patients have some accompanying vestibular syndrome involving disruption in their balance and/or dizziness at one time or another. A large portion of people with migraine often have no accompanying pain, their predominant symptom instead being vertigo. Migraine and its variants must be addressed in the clinical setting by a combination of medical management and comprehensive testing and rehabilitation techniques that offer the most complete and lasting benefit to the patient.

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Vestibular Migraine

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Migraine is one of the most debilitating chronic disorders in the United States. It is almost as prevalent as hypertension (high blood pressure) and is more common than asthma and diabetes mellitus. More importantly, migraine strikes people during what are expected to be their most productive years: between ages 20 and 40 for most women, with a slightly higher age range for men.

Despite better diagnostic capabilities and efforts to improve public awareness and education, it is estimated that approximately 50% of migraineurs go undiagnosed or mismanaged to this day. Many self-treat, or are treated inappropriately for sinus or other non-migrainous types of headache. [1]

Often described as "sick headache," migraine is typically characterized by unilateral onset of head pain, severe progressive intensity of pain, throbbing or pounding, and interference with the person's routine activities. Accompanying symptoms of photophobia (sensitivity to light) or phonosensitivity (intolerance to noise), as well as nausea and/or vomiting, are common, and often leads to the inability to perform daily tasks.

MIGRAINE AND VESTIBULAR DYSFUNCTION

Approximately 40% of migraine patients have some accompanying vestibular syndrome involving disruption in their balance and/or dizziness at one time or another. This may be prior to, during, after, or totally independent of their migraine event. Some interesting parallels exist between migraine and non-migrainous vestibular dysfunction. Many of the food and environmental triggers for migraineurs (see box on page 2) are the same as those for patients with non-migrainous vestibular dysfunction. Hormonal fluctuations, foods, and weather changes (barometric-pressure variations) often exacerbate both conditions. Finally, diet modifications and certain medications used in migraine management may ameliorate or prevent the vestibular component of the migraine. [2,3] Interestingly enough, some of the analgesic medications for the pain do not resolve the dizziness and medications for the dizziness often

TIPS FOR MIGRAINE

Note: Some of the triggers below may also apply to other types of vestibular dysfunction.

Food Triggers

- Aged or ripened cheeses (examples: Cheddar, Gruyère, Emmenthaler, Stilton, Brie, Gouda, Romano, Parmesan, feta, bleu, Camembert)
- Foods containing large amounts of monosodium glutamate (MSG). Processed foods must list MSG as an ingredient, but they may only say, "flavor enhancer 621."
- Smoked, cured, or processed meats such as bacon, sausage, ham, salami, pepperoni, pickled herring, bologna, chicken livers, and hot dogs
- Food prepared with meat tenderizer, soy sauce, vinegar (except white vinegar), or yeast extract; and food that has been fermented, pickled, or marinated
- Pea pods and pods of broad beans such as
- lima and navy beans
- Onions, olives, pickles
- Alcohol (especially red wine, port, sherry, scotch, gin, and bourbon)
- Sour cream, yogurt, buttermilk
- Hot fresh bread, raised coffee cake, doughnuts
- Excessive aspartame (artificial sweetener)
- Chocolate, cocoa, carob
- Nuts, peanut butter
- Certain fruits, including figs, avocados,
- raisins, red plums, passion fruit, papaya,
- banana, and citrus fruit
- Excessive tea, coffee, cola

Other Triggers

- Hormonal fluctuations
- Barometric-pressure variations
- Sleep disturbance
- Stress
- Medications

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Parts of this listing are adapted from Ronald J. Tusa, MD, PhD, "Diagnosis and Management of Neuro-otologic Disorders Due to Migraine," chap. 12 in Vestibular Rehabilitation, ed. Susan J. Herdman, PhD, PT (Philadelphia: F.A. Davis Co., 1994).

do not resolve the painful headache.

The clinical presentation of vestibular symptoms that often correlate with migraine3 includes—but is not limited to—dizziness; motion intolerance with respect to head, eyes, and/or body; spontaneous vertigo attacks (often accompanied by nausea and vomiting); diminished eye focus with photosensitivity; sound sensitivity and tinnitus; balance loss and ataxia; cervicalgia (neck pain) with associated muscle spasms in the upper cervical spine musculature; confusion with altered cognition; spatial disorientation; and anxiety/panic. [4]

While migraine is often associated with benign recurrent vertigo of adults

or paroxysmal vertigo of childhood, [5,6,7] some migraine patients also present with true benign paroxysmal positional vertigo (BPPV) even after the migraine headache event has ceased. This is thought to be caused by a combination of vascular events along with an alteration of neural activity associated with the migraine event. [8,9] It is believed that these changes more commonly affect the utricle and/or the superior portion of the vestibular nerve and anterior vestibular artery, rather than the saccule and the inferior portion of the vestibular nerve and posterior vestibular artery. [10,11] This may explain why results within the normal range are often obtained with vestibular-evoked myogenic potentials (VEMP) testing of migraine patients in the absence of true BPPV. Similarly normal findings have been reported in cases of migraine in the apparent absence of inferior vestibular neuritis, leading to the belief that if inflammation is in fact present as a result of the migraine, and is a cause for utricular BPPV, the local inflammation of the peripheral blood vessels and/or cranial nerve branches is more prevalent in those supplying the utricle rather than the saccule. However, VEMP also now can be helpful in differentiating the clinical presentation of migraine vs Meniere's syndrome or BPPV. Usually following a migraine event, the VEMP intensity measures are commonly hyperresponsive, whereas with Meniere's exacerbation the affected ear intensity response is hyporesponsive, and with BPPV the affected ear latency response is typically prolonged.

RECOGNITION OF MIGRAINE SYNDROMES

Most people associate migraine with severe head pain and a period of incapacitation. However, a large portion of people with migraine often have no accompanying pain, their predominant symptom instead being vertigo (a spinning sensation) or dizziness/ disequilibrium (balance loss), mental confusion, disorientation, dysarthria, visual distortion or altered visual clarity, or extremity paresis. This presentation may result in a visit to the emergency room and extensive laboratory, imaging, and other diagnostic evaluations—often with normal results, which lead to increased confusion and anxiety on the part of the patient. In addition, anti-emetic (anti-vomiting) medications are often given, which may have sedative side effects associated with increased postural instability and increased fall risks.

50% OF MIGRAINEURS GO UNDIAGNOSED.

Clinicians are faced with the task of attempting to apply objective clinical testing methods to determine the etiology (cause) of a patient's symptoms so as to optimize treatment. Often, a combination of etiologies exists, which can complicate or confuse the diagnostic process.

Physicians should be using the International Headache Society's International Classification of Headache Disorders (2nd edition) in order to better diagnose patients with primary headache disorders. These criteria, used

by neurologists and other headache specialists, are readily available in almost every library, either online or in print.

Migraine headaches (with or without aura), tension-type headache, cluster headache, paroxysmal hemicrania, and chronic daily headache constitute the vast majority of primary headache disorders. Variants of migraine, such as post traumatic headache from concussive injury, exertional migraine and benign orgasmic headaches, are becoming more frequently recognized. These variant presentations may also develop vestibular syndromes that are often more persistent and debilitating than the original headache.

MECHANISMS OF MIGRAINE

The emergence of new technologies, such as functional/dynamic imaging studies, has shown promise in documenting the evol functional/dynamic imaging studies, has shown promise in documenting the evolution of the migraine processes. As a result, a better understanding of the vascular and neural processes of migraine has been developed.

The consensus is that the types of headache outlined above—especially migraine/vascular types—are related to a mixed pathophysiology, with cerebral spreading depression of Leão (a spontaneous spreading of an electrical charge along the cortex) followed by activation of pain receptors located in the brainstem, not far from the vestibular apparatus. The release of neurotransmitters then leads to the dilation of blood vessels near the scalp and other structures outside of the brain substance.

Migraine is also thought to be an inherited disorder giving rise to a "vulnerability" to an abnormal discharge of neurons (different from that seen in epilepsy) that preferentially affects brainstem regions and is triggered by a chemical event.[9]

The vascular theory has been long accepted (and is perhaps better understood), which may make it difficult for some practitioners to accept the neural components and associated vestibular manifestations.

The exact mechanisms of migraine are still not completely understood. But since migraine pathophysiology has been shown to be not solely vascular, and is now thought to be a combination of altered vascular and neural processes, migraine-related vestibulopathy is easier to accept and to treat. [12]

EVALUATION AND TESTING

Migraine and its variants must be addressed in the clinical setting by a combination of medical management and comprehensive testing and rehabilitation techniques that offer the most complete and lasting benefit to the patient.

Traditionally, patients with recurrent vertigo associated with migraine

are seen in consultation by neurologists. Otolaryngologists and internists are now becoming more familiar with this condition, but there remains a huge gap between those who care for migraine patients (with or without associated vertigo) and those who have remained "old school"—that is, not recognizing the peripheral and central vestibular components of migraine.

Patients with migraine associated vertigo (MAV) are often seen by audiologists and vestibular rehabilitation therapists for evaluation and treatment. These paramedical specialists are frequently needed to help the primary care doctor make a diagnosis of MAV.

TREATMENT INCLUDES A COMBINATION OF MEDICATIONS, VESTIBULAR REHABILITATION, AND LIFESTYLE MODIFICATIONS.

After an initial, thorough subjective history is obtained, including a recitation of ongoing symptoms and disruption of activities of daily living, a battery of tests is typically performed, to determine a plan of care for optimized therapy. There are a large number of methods available for testing patients with MAV, and an optimal testing protocol is yet to be determined for this population. Some combination of computerized audiological and vestibular-function tests is typically employed, including positional testing with video-oculography; oculomotor and VOR (vestibulo-ocular reflex) assessments with gaze stability and/or dynamic visual acuity testing; horizontal canal testing with vENG (video electronystagmography), with calorics or rotational chair testing (preferred); audiogram and ABR (auditory brainstem response test); functional balance and gait assessments with CDP (computerized dynamic posturography); and VEMP.

In our clinic, a review of results obtained from such tests with MAV patients reveals a combination of findings that are attributable to both central processes and peripheral vestibular functions.

An important component of the evaluation is reliable documentation of the degree of limitation of daily functional capacities. A number of questionnaires and inventories have been employed for this purpose, including the Jacobsen Dizziness Inventory, Dynamic Gait Index, Activities-Specific Balance Confidence Scale, Timed Up and Go test, and others. [7,13]

TREATMENT

The methodology believed to have the highest efficacy in the management of migraine dizziness is a combination of medications, vestibular rehabilitation, and lifestyle modifications that include limitation of the risk factors associated with migraine (those related to diet, sleep, stress, exercise, and environmental factors).

MEDICATION

Medications may be prescribed to prevent migraines or to stop a migraine that has lready started. Drugs used to prevent frequent migraine attacks include beta-blockers, tricyclic antidepressants, calcium channel blockers, and certain anticonvulsant medications (Depakote and Topamax). Over the last several years, venlafaxine (Effexor XR) has become one of the favored preventative drug treatments for patients with migraine related vertigo. Drugs commonly used to stop migraine are aspirin, ibuprofen, isometheptene mucate, and the triptans, such as Imitrex and Relpax. Some of these medications work by blocking the action of serotonin (a neurotransmitter that causes large blood vessels to contract) or prostaglandins (a family of chemicals stimulated by estrogen that cause blood vessels to expand and contract). [14] Generally the differentiation of whether to use a daily preventive vs an abortive type (taken to stop the already started migraine event) is the frequency and severity of the events. This is best determined by the patient's discussion of options with the treating Neurologist.

VESTIBULAR REHABILITATION

The benefits of vestibular rehabilitation are well documented to reduce symptoms and restore function for vestibular-related disorders. [7,13] With MAV, it is often helpful for the patient to have started the prescribed medications prior to beginning the vestibular rehabilitation course. This may allow for better tolerance to the exercise regimen without exacerbating the symptoms. The intensity of the rehabilitation course in gradually increased to the patient's abilities, yet still at a low enough level so as to not initiate another migraine event.

For patients who have alterations in oculomotor functions and VOR deficits giving rise to visual perceptual dysfunction, a concentrated rehabilitation program consisting of VOR and gaze-stability exercises that emphasize visual acuity is effective. Various eye tracking devices are commercially available which allow the examiner to monitor not only the ability of the patient to visually track objects, but also allow the "method" of eye tracking employed by the patient to be evaluated. Spatial awareness may be altered, and exercises emphasizing proprioception and visual perception are helpful. Isolating visual fields incrementally during visual tracking exercises may be helpful in stabilizing alterations in positional sense. Vestibulo-visual interaction exercises also improve eye tracking abilities. It has become evident that velocity specific exercises are most effective. The velocity of the exercises needs to be matched to the measured velocity deficits on test results. Performing visual retraining exercises at random speeds rather than at specific velocities may be less effective. In cases where BPPV exists, performing canalith repositioning maneuvers is effective, and followed with home habituation exercises.

Postural instability and gait alterations respond to balance and gait-training tasks and exercises, employing both static and dynamic type balance exercises. Dual tasking and exercises that combine hand-eye coordination,

balance maintenance, and gaze stability are effective as well, and can be combined with general conditioning exercises to the extent tolerated by the patient's general health. Performing exercises on various surface textures and variable stabilities also is recommended.

In patients with cervicalgia and cervical muscle spasms that limit range of motion, treatment may also include modalities and manual mobilization and stretching of the upper cervical segments, in order to diminish the muscle spasms and guarding and restore normal mobility to the neck. As an adjunct to therapy, greater occipital nerve block (GON) injections are often helpful in reducing symptoms and restoring motion. Some treating MD's now use Botox for these injections for more lasting effect.

LIFESTYLE MODIFICATIONS

A consistent effort by the patient to adhere to necessary lifestyle modifications (including avoiding the migraine triggers mentioned above), medication usage as prescribed, and specific tasks and exercises performed independently at home are critical to the success of the overall rehabilitation program. Such adherence is essential for effective reduction of the symptoms and limitations of function caused by migraine associated vertigo (MAV). [7]

VESTIBULAR TEST RESULTS COMMONLY OBSERVED IN MIGRAINE ASSOCIATED VERTIGO (MAV) PATIENTS

During video-oculography, a prevalent feature is poor gaze stability with ocular "drift," often accompanied by spontaneous up or downbeating directional nystagmus, which does not suppress with fixation-suppression testing added. Unilateral or bilateral gaze induced lateral nystagmus is commonly observed. There may also be a reduced ability to cancel or inhibit the vestibulo-ocular reflex (VOR) function, used for attaining simultaneous head and eye tracking maneuvers. These results may be due to the fact that the cerebellum, which is responsible for coordinating gaze-fixation functions, is thought to be involved in the vascular and neural changes associated with migraine.

Testing of other cerebellar functions (involving coordinated movements of the extremities) may give normal results, with no postural instability or ataxia/apraxia evident, but postural instability is often evident as well. Smooth pursuit tests often give abnormal results (although these must be distinguished from expected age-related changes). Thus, it may be that only those

neural processes of the cerebellum associated with coordinated eye motions are affected in migraine, and not the neural connections involving postural stability.

Computerized dynamic posturography (CDP) may give positive results for postural instability, especially when used in combination with head motions for dual tasking and otolithic system involvement. Alterations in balance strategies are commonly measured, and need to be addressed with the specific balance exercises in accord with test measures.

Saccadic eye-motion testing is usually normal, but a rebound nystagmus may be present with hyperresponsive neural findings and presence of overshoot phenomenon. Directional gaze testing is usually abnormal, as is the Halmagyi head thrust test. HIT (head impulse test) may be helpful in documenting the objective findings of VOR and gaze stability deficit. With Hallpike-Dix positional testing (unless true BPPV presents), no rotational component nystagmus is usually evident. However in acute migraine event, bilateral torsional nystagmus may present with positional testing and gaze added.

With passive VOR assessment via autorotation methods, or with mechanical rotational chair, an abnormal gain value with accompanying phase shift is usually evident. The visual-vestibular interaction can be markedly abnormal and may provoke symptoms of increased dizziness, often with accompanying nausea. Optokinetic after-nystagmus (OKAN) is commonly symmetrically prolonged. Subjective Visual Vertical assessment often is abnormal with accompanying spatial disorientation altered postural positional sense.

Active autorotation testing, which may be limited by cervicalgia and cervical muscle spasms with limited range of motion (often the patient moves "en bloc" to avoid eliciting dizziness), gives sporadic results. Gaze stability testing and dynamic visual acuity testing—after cervicalgia is resolved with appropriate treatments—are typically abnormal. Vestibular-evoked myogenic potentials (VEMP) testing has proven quite useful in determining differential diagnoses. Regularly, hyperactive VEMP responses are found in patients with MAV.

Audiometric testing in cases of migraine associated vertigo (MAV) typically reveals no changes in function other than occasional hyperacusis or noise sensitivity, which usually is temporary and resolves shortly after the migraine event ends. Tinnitus (most commonly associated with labyrinthitis rather than migraine), if present at all, is temporary. In cases of prolonged problematic tinnitus, tinnitus retraining therapy (TRT) may be helpful. Tinnitus masking devices are also commercially available.

SUMMARY

Migraine associated vertigo (MAV) afflicts a large percent of the population and continues to be a challenge to healthcare professionals. Technologies for measurement continue to expand and new medications continue to be manufactured for this affliction. Effective management of MAV

necessitates a comprehensive effort and active participation of the patient, the treating physician, and the rehabilitation professionals. Proper identification, objective diagnostic measurements, and optimized treatment approaches net the best results.

Diagnostic Tests for Vestibular Disorders

By P.J. Haybach, MS, RN, and the Vestibular Disorders Association, with revisions by Dr. Joel Goebel

The inner ear's vestibular organs and the associated nerves and brain centers form a complex system that serves many functions and can be affected by a number of outside systems. A thorough evaluation of the inner ear may therefore require several different kinds of tests. Doctors use information from a person's medical history and findings from a physical examination as a basis for ordering diagnostic tests to assess the vestibular system function and to rule out alternative causes of symptoms. Most people tolerate these tests well. However, sometimes the tests are fatiguing and can result in temporary unsteadiness.

TESTING VESTIBULAR DYSFUNCTION

The vestibular and visual systems are connected to each other and to the muscles in the eyes and neck that help maintain good balance. Head movements or other stimulation of the inner ear sends signals through the nervous system to control eye muscle movements. This forms a reflex pathway called the **vestibulo-ocular reflex, or VOR**. This system is designed to generate eye movements that maintain clear vision when the head is in motion. Many vestibular tests use equipment to monitor the eyes for normal and abnormal movements when the vestibular system is stimulated.

ELECTRO/VIDEO-NYSTAGMOGRAPHY (ENG OR VNG)

Electronystagmography (ENG) refers to a group of tests or test battery, and uses small electrodes placed over the skin around the eyes during testing. ***Videonystagmography (VNG)*** refers to the same test battery run using goggles with video cameras to monitor the eyes. Both the video cameras and the electrodes can measure eye movements to evaluate signs of vestibular

dysfunction or neurological problems. Generally these tests are performed in a room that is dark or with low lighting. The examiner asks random questions that are meant to occupy the person being tested and keep them alert. ENG/ VNG tests are the most common set of tests administered to people with dizziness, vertigo, and/ or imbalance.

VNG BINOCULAR GOGGLES

Parts of the ENG/VNG test battery evaluate the movement of the eyes as they follow different visual targets. Other parts of the ENG/ VNG observe eye movements as the head is placed in different positions. A third component of the ENG/VNG is called the ***caloric test***, which uses changes in temperature within the ear canal to stimulate part of the vestibular system. Air or water may be used to modulate the ear canal temperature, which may be warmer or cooler than body temperature. This test should provoke jerking eye movements (***nystagmus***) for a short time.

CALORIC AIR FX TESTING

ROTATION TESTS

Rotation tests are another way of evaluating how well the eyes and inner ear work together. These tests also use video goggles or electrodes to monitor eye movements. The head is rotated side to side at moderate or slow speeds, and associated eye movements are analyzed. Like the ENG/VNG, rotation tests are performed in a room that is dark with the examiner asking random questions during testing. Rotation tests provide information beyond the ENG/ VNG about how well the balance organs are functioning. Not all people in the diagnosis phase will require rotation tests.

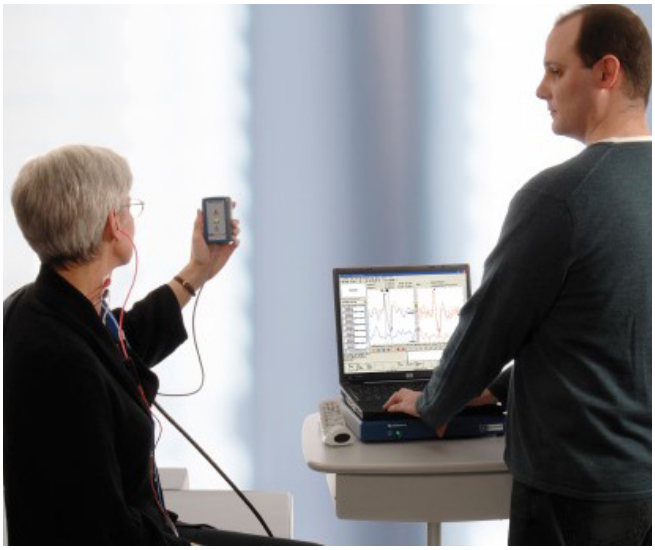
There are different kinds of rotation tests: auto head rotation, computerized rotary chair, or a screening test. In auto head rotation, the person being tested is asked to look at a fixed target and move his/her head back and forth or up and down for short periods of time. During computerized rotary chair tests, the patient sits in a motorized chair that swivels side to side at a controlled rate. Screenings can be performed with the examiner watching the eyes while turning the subject side to side in a swivel chair.

VIDEO HEAD IMPULSE TESTING (VHIT)

VHIT also evaluates how well the eyes and inner ears work together. A small set of glasses with a camera are used to monitor eye movements. The VHIT is similar to rotational testing, where the head is moved to evaluate the vestibulo-ocular reflex. However, the VHIT test uses very small and quick movements of the head to evaluate reflex function, as opposed to the slow or moderate speeds used in rotation testing. Not all people in the diagnosis phase will require VHIT tests.

VESTIBULAR EVOKED MYOGENIC POTENTIAL (VEMP)

VEMP testing is used to evaluate whether certain vestibular organs and associated nerves are intact and functioning normally. Responses in this test are measured from different muscles in the neck and around the eyes. VEMP testing uses adhesive, skin surface electrodes (like ENG or some rotational tests) and earphones (like those used during a hearing test). Sound is played for a few seconds through the earphones, the vestibular organs are stimulated and activate muscle responses, and electrodes record the results.



VEMP TESTING

COMPUTERIZED DYNAMIC POSTUROGRAPHY (CDP)

CDP tests postural stability or the ability to maintain upright posture in different environmental conditions. Maintenance of postural stability depends on sensory information from: the body's muscles/joints, eyes, and inner ears. This testing investigates relationships among these three sensory systems and records the balance and posture adjustments made when different challenges are presented. This test may also be used in a rehabilitative setting after a diagnosis has been determined, and is not performed on all people in the diagnosis phase.

CDP tests involve standing still on a platform. The platform may be still or able to shift, or a visual target may be still or able to move during testing. Pressure gauges under the platform record shifts in body weight (body sway) as the person being tested maintains balance under different conditions. A safety harness is worn as a precaution, should the patient lose their balance.

AUDIOMETRY (HEARING TESTS)

Audiometry measures hearing function. Hearing evaluations are an important part of vestibular diagnostics, because the inner ear contains both hearing and balance organs. More than one hearing test may be required when a person has a vestibular disorder, especially when there is evidence of hearing loss, a sensation of fullness in the ears, or tinnitus (ringing or noise in the ears).

The audiometric test battery is carried out in a sound-treated room. Earphones are used to present words and tones at different pitches and levels. A response is requested when these sounds are heard. Testing with words may include repeating words in a quiet room or when noise is playing.

Another part of a standard hearing test is tympanometry, which can help detect problems between the ear drum and the inner ear. Tympanometry uses a small earpiece that creates pressure and plays sound in the ear canal to gather information. The same equipment can also be used for acoustic-reflex testing, which measures the reflex of muscles in the middle ear in response to pressure and loud sound.

OTOACOUSTIC EMISSIONS (OAE)

OAE testing provides information about how the hair cells of the cochlea are working by measuring the responsiveness of hair cells to a series of clicks produced by a tiny speaker inserted into the ear canal. Most often this test is used to evaluate hearing for people who are unable to respond to a traditional hearing test (such as infants).

ELECTROCOCHLEOGRAPHY (ECOG)

ECoG measures a response to sound from the nervous system. It utilizes an earphone and electrodes while the person being tested lays still in a comfortable position. Not all people in the diagnosis phase will need ECoG tests.

An earphone plays sound in the ear and an electrode measures a response. Different electrodes can be used in this test. Some may be adhesive, skin-surface electrodes. Others may fit in the ear canal like an earphone, while a third type of electrode is designed to gently rest against or touch the eardrum. A fourth type of electrode is a needle that is placed through the eardrum to touch the inner ear. Most clinics use the first three types of electrodes to measure an electrical signal while sound is playing.

AUDITORY BRAINSTEM RESPONSE TEST (ABR; OR BER, BSER, OR BAER)

The ABR measures how the nervous system responds to sound. The test setup and procedure is similar to the ECoG. Most often ABR is used to test hearing for people who are unable to respond for audiometry (such as

infants). Occasionally this test is used when someone cannot have imaging performed (such as people with a metal plate in the body/brain).
Under certain circumstances, this test can indicate the presence of an acoustic neuroma (a rare, benign tumor of the vestibulo-cochlear nerve). It may also help identify conditions such as multiple sclerosis if they have affected the auditory pathway to the brain.

MAGNETIC RESONANCE IMAGING (MRI)

MRI uses a magnetic field and radio waves to produce cross-sectional images of body tissues being scanned. An MRI of the brain can reveal the presence of tumors, stroke damage, and other soft-tissue abnormalities that might cause dizziness or vertigo. MRIs of structures in and around the inner ear can be helpful in the diagnosis of some vestibular disorders.

COMPUTERIZED AXIAL TOMOGRAPHY (CAT, OR CT)

A CT scan is an X-ray technique that is best for studying bony structures. The inner ear is inside of the skull's temporal bone on each side. These scans are often used to look for abnormalities around the inner ear, such as fractures or areas with thinning bone.

OTHER TESTS

Depending on your circumstances, other tests may be necessary to discover the cause of a balance disorder. Blood work, allergy tests, vision tests, and other exams may help rule out causes of imbalance that are unrelated to the vestibular system.

WHO PERFORMS VESTIBULAR TESTING?

Generally your primary care physician, ENT or neurologist will refer you to: an audiologist for hearing or balance related testing, a physical therapist for gait or balance related testing, or a radiologist for imaging testing. These specialists will send your test results back to your physician with an analysis, and your physician will explain them to you.

Dietary Considerations

By Vestibular Disorders Association (VeDA) with updates by
Dr. Maggie Bloom, PhD

Many people with Ménière's disease (also called primary idiopathic endolymphatic hydrops), secondary endolymphatic hydrops, or migraine-associated dizziness find that certain modifications in diet are helpful in managing their disorder.

INNER EAR FLUID BALANCE

The fluid-filled hearing and balance structures of the inner ear normally function independently of the body's overall fluid/blood system. The fluid that bathes the sensory cells of the inner ear (known as endolymph) maintains a constant volume and contains specific and stable concentrations of sodium, potassium, chloride, and other electrolytes. With injury or disease, the volume and composition of endolymph may fluctuate with changes in the body's fluid/blood.

This fluctuation is thought to cause the symptoms of endolymphatic hydrops or Ménière's disease: pressure or fullness in the ears, tinnitus (ringing in the ears), hearing loss, dizziness, and imbalance. Thus, for people with Ménière's disease or secondary endolymphatic hydrops, maintaining fluid/blood stability is important.

DIETARY GOALS

Dietary strategies for regulating fluid balances involve modifying the amount and fluctuations of certain substances consumed and reducing or eliminating other substances that can adversely affect the inner ear. These dietary strategies may be incorporated into an individualized nutritional plan developed with the help of a physician or dietitian.

GENERAL GUIDELINES

1. Distribute food and fluid intake evenly throughout the day and from day to day. This includes consuming approximately the same amount of food at each meal, not skipping meals, and eating snacks, if needed, at regular intervals. Evenly spacing food and fluid intake helps with inner-ear fluid stability; hypoglycemia (low blood sugar) can trigger migraine attacks. Having breakfast soon after rising can help stabilize your system for the day.
2. Avoid foods and beverages that have a high salt or sugar content. In general, a diet high in fresh fruits, vegetables, and whole grains and low in canned, processed frozen food, and other processed foods helps control salt and sugar intake. Be careful of drinking fruit juices as they may have a very high sugar content.
3. Drink adequate amounts of fluid daily. Fluids can include water, milk, and low-sugar fruit juices but not coffee, caffeinated tea, alcohol, or soft drinks. If possible, extra fluids should be drunk before and during exercise and in hot weather. It is important to make sure that you drink at least 5 or more glasses of water over the course of the day. You should not have all your fluid intake a one time.
4. Avoid foods and beverages with caffeine.
5. Limit or eliminate alcohol consumption.
6. Do not use tobacco.
7. Check with your Physician or a Natural Health Practitioner before taking extra herbs, vitamins and supplements as they may cause your symptoms to increase.

SPECIFIC GUIDELINES

Salt and sodium: Sodium intake affects body-fluid levels and their regulation. Salt and sodium are not identical; table salt (sodium chloride) is made up of 40% sodium and 60% chlorine. Sodium occurs naturally in all foods and in drinking water.

The American Heart Association recommends that healthy adults limit their sodium intake to no more than 2,400 mg (milligrams) per day. People on restricted-sodium diets may be limited to 1,000–2,000 mg of sodium per day, or about one-half to one teaspoon of salt. Each individual's physician will be the best judge of appropriate levels of sodium intake.

Strategies for reducing sodium intake at home: Some people find that it is difficult to adapt to a reduced-sodium diet because salt is so often used to add flavor to foods. It's important to be selective about meal ingredients and seasonings and look for hidden sodium.

Foods that are naturally low in sodium include fresh fruits and vegetables,

unprocessed grains, and most fresh meats, poultry, and fish. Some frozen or canned food items are available without added salt. For those who have been accustomed to using salt, foods may initially taste bland, but introducing herbs and spices can help make them more flavorful and palatable.

Many commercially packaged salt substitutes contain mixtures of herbs and spices. However, such products also often include potassium, which can complicate certain medical conditions (particularly those involving the kidneys), and thus should not be used without first consulting a physician.

Strategies for reducing sodium intake when dining out: In restaurants, batter-fried foods tend to be salty, as do combination dishes such as soups or pasta with sauce. Selecting plain foods from the menu—such as grilled or roasted entrees, baked potatoes, and salad dressed with oil and vinegar—can reduce salt intake. Most restaurants comply with requests for sauces and dressings to be served on the side or for dishes to be prepared without added salt. It is helpful to substitute a side salad or fresh fruit in place of fries or other salty items. You can also request that they leave off salt when they are cooking vegetable side dishes. Beware of words like "smoked" or "blackened" as these foods are generally high in salt. (See page 3 for tips on selecting a restaurant.)

Looking for hidden sodium: Many kinds of convenience foods, such as frozen dinners, items from restaurant take-out menus, and foods with MSG (monosodium glutamate) contain large amounts of sodium. Foods that are usually very high in sodium include cured meats such as ham and bacon, processed foods such as canned meats and vegetables, and condiments such as soy sauce, ketchup, mustard, pickles, and olives. Canned and dehydrated soups, cereals, cheeses, salad dressings, sauces, chips, and salted nuts may also be high in sodium.

Reading labels for sodium content: It is essential for those on restricted-sodium diets to read labels on packaged food, particularly because some foods with added salt do not taste salty. Foods that list salt as one of the first three ingredients on the label should be avoided. Ingredient lists with the words sodium or soda (which is sodium bicarbonate, or baking soda) or Na (the chemical symbol for sodium) indicate the presence of sodium in food. Compare various brands to see which ones have the least sodium as many times there is a big discrepancy.

DIETARY CONSIDERATIONS

- Sodium
- Sugar
- Caffeine
- Suppliements
- Alcohol
- Specific Migraine Triggers
- Medications
- Nicotine

The FDA (US Food and Drug Administration) has established definitions for sodium and salt content in food labeling.

- ***Sodium-free or salt-free:*** less than 5 mg of sodium per serving
- ***Very low sodium:*** 35 mg or less per serving or 50g of food
- ***Low-sodium:*** 140 mg or less per serving or 50g of food
- ***Light in sodium:*** sodium is reduced by at least 50 percent
- ***Reduced/less sodium:*** at least 25 percent less sodium
- ***Lightly salted:*** 50 percent less sodium than normally added
- ***Unsalted, without added salt, or no salt added:*** no salt added during processing

Sugar: Meals or snacks with a high sugar content can cause fluctuations in the volume of body fluids, which may increase vestibular symptoms. For the purpose of minimizing such fluctuations, foods with complex sugars (such as those found in legumes, whole grains, potatoes, and vegetables) are better choices than foods with a high concentration of simple sugars (such as table sugar, brown sugar, honey, maple syrup and corn syrup). Tips for lowering overall sugar consumption include cutting the amount of sugar in recipes in half, substituting fresh fruit for sweetened baked goods, and possibly the use of sugar substitutes. Sprinkling a few currants or berries can help to sweeten up dish.

Reading labels for sugar content: On packaged-food labels, ingredients that end in ose are sugars (for example, dextrose, fructose, and sucrose). Corn syrup, honey, molasses, sorbitol, and mannitol are also sugars. If one of the first three ingredients listed on the label is a sugar, the sugar content of that product will be high, but it is always good to check the actual sugar amount (listed in grams) to be sure as sometimes there may only be three or four ingredients total.

Sugar substitutes: A physician can provide the best advice about whether sugar substitutes are appropriate to use in reducing sugar intake. For use in foods, the FDA has approved four sugar substitutes: saccharin (Sweet'N Low), aspartame (Equal), acesulfame-K (Sweet One), and sucralose (Splenda). The chemical composition of some sugar substitutes, however, may include sodium (for example, sodium saccharide); some substitutes, including aspartame and sucralose, are not always suitable for use in cooking or baking. Additionally, there is some indication that sugar substitutes can influence metabolic syndrome imbalances.

OTHER DIETARY SUBSTANCES

Caffeine is a stimulant that can make tinnitus louder and increase other symptoms. The diuretic properties of caffeine also cause excessive urinary loss of body fluids. Foods and beverages that often contain caffeine include chocolate, coffee, soft drinks, and tea.

Some **supplements**, like licorice root, can interfere with blood pressure and fluid control. Not all supplements are equal in quality or manufacturing

process, so it is best to check with your Physician or Natural Health Practitioner to find out which supplements are appropriate.

Alcohol can directly and adversely affect the inner ear by changing the volume and composition of its fluid.

Avoiding **migraine triggers** may help control migraine-associated dizziness. Migraine triggers

include foods that contain the amino acid tyramine. Examples include red wine, chicken liver, smoked meats, sour cream, yogurt, pickled herring, chocolate, bananas, citrus fruits, figs, ripened cheeses (such as cheddar, Stilton, Brie, and Camembert), nuts, and peanut butter. Other migraine triggers include foods containing large amounts of MSG, nitrite/nitrate-preserved foods (such as hot dogs and pepperoni), and yeast. Not all people with migraines are affected by these triggers.

NON-DIETARY SUBSTANCES

Some **medications** contain substances that can increase symptoms of vestibular disorders. For example, aspirin can increase tinnitus, and nonsteroidal anti-inflammatory drugs (NSAIDs) such as ibuprofen can interfere with the body's fluid-control mechanism, causing water retention or electrolyte imbalance. Some medications contain caffeine. **Antacids** may have significant amounts of sodium. If product ingredients are not readable on the package label, a pharmacist can provide information.

Nicotine (found in tobacco products and some cease-smoking aids) can increase symptoms, because it decreases the blood supply to the inner ear by constricting blood vessels; it also causes a short-term increase in blood pressure. In addition, nicotine is a migraine trigger.

DINING OUT

Crowded, busy social settings such as restaurants may be very difficult to navigate if you have a chronic vestibular disorder. By making some adaptations, you may still be able to meet friends and eat in relative comfort. However, even with the best planning, you may become dizzy or disoriented. It will be easier on you and your dining companions if you explain your problem and suggest ways you can be helped before you actually need assistance.

Selecting a restaurant

- Pick a restaurant with small separate rooms.
- No matter where you go, avoid rush hours.
- Avoid loud background music.
- Seek carpeted floors that reduce conversational noise and vibrations caused by waiters moving nearby.
- Avoid visually distracting shiny, checkered floors and surfaces, as well as ceiling fans and busy wallpaper.
- If the restaurant has a website, download a menu in advance and plan the meal to avoid visual strain and confusion.

Lighting

- Fluorescent lights may cause visual difficulty; sit away from and with your back to the light.
- Be aware that many restaurants control lights with a central rheostat, which can be visually disorienting.
- Ask that flickering candles be removed.

Seating hints

- Seat yourself in the corner of a restaurant, avoiding the bustling middle.
- Sit away from kitchens, cash registers, and bars.
- Sit in chairs rather than benches to reduce motion caused by others seated next to you. Booths may help block noise and activity.
- To reduce the amount of head turning required to converse, choose a round table or sit at the head.

ADDITIONAL RESOURCES

Some helpful documents available from VeDA:

- Ménière's Disease—What You Need to Know
- Secondary Endolymphatic Hydrops
- Migraine-Related Dizziness: An Updated Understanding

More information about food content may be found through the American Heart Association (www.americanheart.org) and the US Food and Drug Administration (www.fda.gov). Many of the guidelines presented in this article are commonly recommended to people with Ménière's disease, endolymphatic hydrops, or vestibular migraine. A physician or dietitian may incorporate some of these principles into an individualized treatment plan.

Pharmacologic Treatment

By Dario A. Yacovino, MD (Neuro-Otology Department. Neurology Research Institute "Dr. Raul Carrea" (FLENI), Buenos Aires, Argentina) and Leonel Luis, MD (Clinical Physiology Translational Unit, Institute of Molecular Medicine, Faculty of Medicine, University of Lisbon, Portugal, and Otolaryngology Department, Hospital de Santa Maria, Lisbon, Portugal)

INTRODUCTION

Vertigo and dizziness are among the most common complaints, having a lifetime prevalence of about 30%[1]. They are symptoms of a variety of disorders that involve the peripheral (otologic vertigo) and/or the central vestibular (brain-induced vertigo) systems. These produce asymmetric input into the central vestibular apparatus or asymmetrical central processing. If this process is acute, vertigo, nausea and vomiting may result. If it is more chronic, dizziness and/or disequilibrium may be the manifest symptoms.

Depending on their etiology, treatment options of vestibular disorders may be summarized as (Table 1, Page 35):

- Pharmacological treatments;
- Liberatory and repositioning maneuver for BPPV treatment (specific maneuvers according to the location(s) of the otoconial debris; Epley and Semont maneuvers are common examples for repositioning debris located in the posterior semicircular canal);
- Vestibular rehabilitation (e.g. exercises for eye and head stabilization, proprioceptive training or habituation exercises);
- Psychotherapeutic measures (particularly important in psychogenic vertigo);
- Surgical treatments - in less frequent lesions such as semicircular canal dehiscence, where there is a lack of bone covering one or more semicircular canals and ear tumors (e.g. vestibular schwannoma); some drugs (namely gentamicin and dexamethasone) may also be applied transtympanically as a simple procedure under topical anesthesia.

With this paper we aim to introduce the reader to the complexity of decision-making when treating vestibular disorders, as well as to analyze the most used pharmacologic strategies for the most common etiologies of vertigo and dizziness.

PREREQUISITES FOR PHARMACOLOGICAL TREATMENT

While vestibular diagnosis has tremendously evolved with the development of new instruments – vHIT (video Head Impulse Test) and VEMP (vestibular evoked myogenic potentials), just to mention a few examples – the treatment of vestibular pathology has undergone many changes not so much by the discovery of new medications, but rather by the use of medications originally used for non-vestibular pathologies. Many of these drugs are still used in off-label manners (i.e., are used in a way not specified in the FDA's approved label). This is because only a few medications have proven, in controlled trials, to be effective. As in all cases and particularly with these drugs, patients should therefore be informed before starting treatment of the balance between risks and benefits.

The prerequisites for successful pharmacological treatment of vertigo and dizziness are the "4 D's" 2: correct diagnosis, correct drug, appropriate dosage and sufficient duration (Table 2, Page 36).

CLARIFYING SYMPTOMS

The first step for successful treatment, establishing a diagnosis, is especially important because vertigo and dizziness are not diseases – they are symptoms: just as headaches, nausea or fever relate to specific pathologic conditions, so do vertigo and dizziness.

Recording a patient's clinical history should search for the clarification of these symptoms:

- Is there vertigo or dizziness? With vertigo the patient will have a sensation of false or distorted self-motion.
- Are the patient's symptoms spontaneous or triggered (e.g., by head movement or position changes)?
- How long has the patient had symptoms, and how often do they occur? When did symptoms first begin?
- Are there accompanying symptoms, namely ear symptoms or neurological symptoms?

Clinical examination is also mandatory for diagnosis and should be carried out in every patient. Eye movement evaluation is one of the major windows in this respect because particular eye movements are evoked by particular vestibular conditions. A precise and brief neurological and otological examination should also be conducted.

TABLE 1: VERTIGO AND DIZZINESS TREATMENT OPTIONS
Pharmacological
Liberatory and reposition maneuvers
Vestibular rehabilitation
Psychotherapeutic measures
Surgical treatments

Treatment is dictated by the patient's diagnosis. The use of medication for the treatment of vestibular disorders may be directed to treat the etiology, control the symptoms, accelerate central compensation or diminish the psychological comorbidity that often accompanies the syndrome (Table 3, Page 36).

There are six major groups of drugs that can be used for to treat vertigo and dizziness (Table 4, Page 37): antiemetics; anti-inflammatories, anti-Ménière's, anti-migrainous; antidepressants and anticonvulsants.

PHYSIOLOGY

Vertigo is the illusion of rotational motion. Most vertigo with definable cause is otologic, caused by dysfunction of the labyrinth in the inner ear. Normal persons continuously process three types of sensory input: visual, vestibular (inner ear) and somatosensory (sense for position and movement of body parts) to estimate the orientation and motion of the head and body. Physiologic and pathologic vertigo is caused by asymmetric input into the central vestibular apparatus or asymmetrical central processing. Many pathways and neurotransmitters are involved in causing the vertigo and autonomic complaints. This explains why so many classes of drugs are used in the management of this disorder. Occasionally in some oculomotor disturbances accompanied by nystagmus (rhythmic and involuntary eye movement) the patient can feel oscillopsia: the illusion that the world is jumping or swinging back and forth. There are some medications to diminish this disabling symptom and improve the visual support (e.g. clonazepam for certain cerebellar induced nystagmus).

In addition to the symptom of vertigo, motion sickness (the malaise and nausea which may follow real or illusory sensations of motion) should also

TABLE 2: PREREQUISITES FOR PHARMACOLOGICAL TREATMENT
Correct diagnosis
Correct drug
Appropriate Dosage
Sufficient duration

TABLE 3: MEDICATION TARGETS IN VERTIGO AND DIZZINESS
Treat the etiology
Control the symptoms
Accelerate central compensation
Diminish the psychological comorbidity

be considered. Vertigo and motion sickness are not synonymous. For example, reading in a moving car may, in susceptible persons, induce nausea and autonomic symptoms but not the false sensation of self-motion.

VERTIGO AND DIZZINESS PHARMACOLOGICAL APPROACH

Clinically, treatment options for patients with vertigo include symptomatic, specific and prophylactic approaches. Symptomatic treatment involves controlling the acute symptoms and autonomic complaints (e.g., vertigo and vomiting). Specific treatment involves targeting the underlying cause of the vertigo (e.g., ear infection). Prophylactic treatment aims to reduce the recurrence of specific vertiginous conditions, as in Ménière's disease, migrainous vertigo or vestibular paroxysmia.

SYMPTOMATIC CONTROL: VESTIBULAR SUPPRESSANTS AND ANTIEMETICS

Symptomatic control involves managing the acute symptoms and autonomic complaints (e.g., vertigo and vomiting). There is a connection between the part of the brain involved in vomiting and the vestibular system. If the vestibular system is strongly stimulated, either by real motion or by vertigo, the vomit center becomes active and nausea and vomiting occurs. Nausea and vomiting can be even more stressful than vertigo itself, therefore being one of the main targets for pharmacological treatment. Other associated symptoms named "autonomic symptoms" are pallor, swelling, salivation, diarrhea and abdominal distention.

TABLE 4. DRUG GROUPS IN VERTIGO AND DIZZINESS TREATMENT
Anti-inflammatories
Anti-migrainous
Anticonvulsants
Anti-Ménière's
Antidepressants
Antimetics

VESTIBULAR SUPPRESSANTS

Vestibular suppressants are drugs that reduce the intensity of vertigo and nystagmus evoked by a vestibular imbalance. These also reduce the associated motion sensitivity and motion sickness. Conventional vestibular suppressants consist of three major drug groups: anticholinergics, antihistamines and benzodiazepines.

BENZODIAZEPINES

Diazepam (Valium®), clonazepam, lorazepam and alprazolam are benzodiazepines commonly prescribed for their effect as anxiolytics and antidepressants. These drugs also act as vestibular suppressants and can, in small dosages, be extremely useful for the management of acute vertigo[3]. They are also useful in controlling motion sickness[4] and can also minimize anxiety and panic associated with vertigo. Habituation, impaired memory, increased risk of falling and vestibular compensation are potential side effects. Their use as vestibular suppressants should therefore be limited in time. Nevertheless, they should not be stopped suddenly because of potential withdrawal syndrome.

ANTIHISTAMINES

Antihistamines include meclizine (Antivert®), dimenhydrinate, diphenhydramine (Benadryl®) and promethazine. These drugs can prevent

motion sickness and reduce the severity of symptoms even if taken after the onset of symptoms[5]. Dry mouth and blurry vision are side effects that result from their anticholinergic action.

ANTICHOLINERGICS

Anticholinergics are vestibular suppressants that inhibit firing in vestibular nucleus neurons[6] as well as reduce the velocity of vestibular nystagmus in humans. The most effective single anticholinergic drug for the prophylaxis and treatment of motion sickness is scopolamine. All anticholinergics conventionally used in the management of vertigo or motion sickness have prominent side effects, often including dry mouth, dilated pupils and sedation.

ANTIEMETICS

Antiemetics are drugs that are commonly used to control vomiting and nausea. The choice for vertiginous patients depends upon the route of administration and the side effect profile. Injectables are mostly used in the emergency room or inpatient settings. Dexamethasone (Decadron®) and ondansetron (Zofran®) are powerful and well-established inpatient-setting antiemetics. While not FDA approved, droperidol (Droleptan®) is widely used outside the U.S. The oral agents are only used for mild nausea, with sublingual administration preferable for outpatients. When an oral agent is appropriate, meclizine or dimenhidrinate (Dramamine®), antihistamines commonly used also as vestibular suppressants, are generally the first to be used because they rarely cause adverse effects any more severe than drowsiness. Phenothiazines, such as prochlorperazine (Compazine) and promethazine (Phenameth®, Phenergan®), are also effective antiemetics but side effects include sedation and the possibility of extrapyramidal symptoms (dystonia and Parkinsonism). Drugs that speed gastric emptying, such as metoclopramide (Reglan®) and Domperidone may also be helpful in managing vomiting[7].

TREATMENT OF INDIVIDUAL SELECTED CONDITIONS

VESTIBULAR NEURITIS

Vestibular neuritis is the most common cause for acute vestibular syndrome (acute vertigo with acute nystagmus). Although it is believed to be caused by the reactivation of a virus (Herpes simplex virus: type 1) in the vestibular

nerve (vestibular neuritis), it does not benefit from antiviral treatment but rather from methylprednisolone (Medrol®), a corticosteriod. In fact, this drug alone has proven to significantly improve the recovery of peripheral vestibular function in patients with vestibular neuritis[8].

Symptomatic treatment should also be provided in the first days (symptomatic control: vestibular suppressants and antiemetics). In the emergency room Dexamethasone, also a corticoid, may be particularly useful for both its anti-emetic and anti-inflammatory properties. Treatment with vestibular suppressors should be discontinued once the acute symptoms are controlled; chronic treatment with these drugs is discouraged to prevent the inadequate compensation. Vestibular rehabilitation has shown to be most effective strategy in reaching complete clinical recovery[9].

VESTIBULAR MIGRAINE

This long-ignored condition is currently recognized as one of the most common causes for vertigo and dizziness. A number of criteria have to be addressed, but simplistically both migraine and vertigo or dizziness must be related in time in order to diagnose this condition. The treatment includes trigger avoidance, pharmacotherapy and vestibular rehabilitation. For acute attacks only symptomatic control is eventually effective (symptomatic control: vestibular suppressants and antiemetics) as migraine abortive agents such as triptans have reached inconclusive results. Prophylactic treatment protocols are based on the ones from migraine headache, and include beta-blockers such as propranolol or metoprolol; calcium-channel blockers such as verapamil, antidepressants such as amitriptyline, fluoxetine, or venlafaxine[10]; anticonvulsants such as valproate or topiramate, and carbonic anhydrase inhibitors such as acetazolamide.

MÉNIÈRE'S DISEASE

Ménière's disease is the second most common cause of vertigo of otologic origin and is classically attributed to dilation and periodic rupture of the endolymphatic compartment of the inner ear. The pathognomonic symptoms include episodic vertigo, ipsilateral fluctuating hearing loss,

aural fullness and tinnitus[11]. The treatment should therefore address these symptoms, i.e. stop vertigo attacks, abolish tinnitus and reverse or preserve the hearing loss. Clinically the pharmacological treatment is addressed at the acute episode management, prevention of new attacks and the treatment of audio-vestibular dysfunction. There is no consensus on prophylaxis of Ménière's syndrome, with major differences between the U.S. and Europe regarding whether betahistine offers therapeutic benefits.

The treatment during the attack is symptomatic and similar to other etiologies of spontaneous vertigo, with vestibular suppressors and antiemetics being the most appropriated strategies. Irrespective of the prophylactic treatment used, remission may eventually occur in 60% to 80% of cases[12-13]. At start, patients should follow dietary salt restriction (1-2 gram salt diet) and adequate hydration (35 ml/kg of liquids). Patients should also avoid caffeine and stop smoking. If the patient does not achieve a good control of symptoms by following this regimen, a mild diuretic, such as Dyazide® or Maxide® (hydrochlorothiazide-triamterene), may reduce the frequency of attacks[14]. It should be noted that diuretics may cause significant hyponatremia and low blood pressure, especially in the elderly and in those who are already on salt-restricted diets.

This treatment with betahistine regimen is widespread worldwide, with a survey in England reporting that 94% of ENT surgeons prescribe betahistine to their Ménière's patients[14]. The underlying mode of action is believed to be through increased inner-ear blood flow, with local vasodilation and increased permeability, thereby relieving pressure from the inner ear. A long-term high-dose treatment with betahistine (at least 48 mg three times daily), has shown a significant effect on the frequency of the attacks[15]. Some patients also respond well to corticoids. Studies on transtympanic steroids have shown evidence of good preservation of hearing and tinnitus control with substantial decrease in the number of vertigo spells16. Before considering nonconservative measures, using transtympanic steroids could be a good approach in patients refractory to betahistine, those with bilateral Ménière's and those with relatively good hearing in the affected ear.

Patients with Ménière's disease may become disabled by recurrent vertigo; in this situation surgical treatment to inactivate all or part of the labyrinth could correctly be indicated.

In recent years, Ménière's treatment has been revolutionized by the use of transtympanic "low-dose gentamicin." In 1997, Driscoll reported that a single dose of gentamicin through the eardrum eliminated recurrent vertigo in 84% of his patients[17]. This procedure has made it possible to control vertigo after other drug treatments have failed.

There is not much evidence that treatment of chronic audio-vestibular dysfunction prevents further progression of hearing loss. Hearing aids and vestibular rehabilitation could be indicated.

VESTIBULAR PAROXYSMIA – NEUROVASCULAR CROSS-COMPRESSION

Vestibular paroxysmia is believed to be caused by the neurovascular compression of the cochleovestibular nerve, as it occurs with other neurovascular compression syndromes (e.g. trigeminal neuralgia). The irregular and unpredictable spells are the most disabling aspect of this condition, making some daily activities, like driving, extremely dangerous. In theory, given its pathophysiology, surgical treatment could be considered. Still, due to the substantial surgical risks involved, this approach is reserved for particular cases where pharmacological treatment is not effective or tolerated. Treatment with carbamazepine (Tegretol®) or oxcarbamazepine (Trileptal®), both anticonvulsants primarily used in the treatment of epilepsy, is usually not only effective in small dosages, but is also diagnostic. Vestibular depressants are not effective.

CONCLUSIONS

Together with physical therapy and lifestyle changes, the pharmacological approach is one of the three pillars for vestibular disorder treatment. The use of medication in each case comes from a proper assessment of symptoms, severity of disease and side effects. Vestibular suppressants should only be used in acute cases to alleviate the stressful symptoms because prolonged use may generate a chronic vestibular imbalance. Preventive medications generally do not cure the underlying disease but may decrease or abolish the number of attacks of vertigo and dizziness. Most of the drugs used for vertigo treatment act specifically on certain receptors or ion channels, but there are several neurotransmitters and pathways involved in causing the vertigo and autonomic complaints. The knowledge of some of these pathways and drug mechanisms has enabled recent advances in the treatment of specific vestibular disorders, such as vestibular migraine, vestibular paroxysmia or some central nystagmus. Still, the main focus should be kept on establishing a correct diagnosis, then developing an effective treatment regime, for patients suffering from vertigo and dizziness.

Vestibular Rehabilitation Therapy (VRT)

By Lisa Farrell, PT, PhD, AT,C; Clinical Faculty, Department of Physical Therapy, Nova Southeastern University, Fort Lauderdale, Florida'

Evidence has shown that vestibular rehabilitation can be effective in improving symptoms related to many vestibular - inner ear - disorders. [1, 2] People with vestibular disorders often experience problems with vertigo, dizziness, visual disturbance, and/or imbalance. These are the problems that rehabilitation aims to address. Other problems can also arise that are secondary to the vestibular disorder like nausea and/or vomiting, reduced ability to focus or concentrate, and fatigue.

Symptoms due to vestibular disorders can diminish quality of life and can impact all aspects of life from economic to social participation as well as can contribute to emotional problems, like anxiety and depression. Additionally, one of the consequences of having a vestibular disorder is that the symptoms frequently cause people to adopt a sedentary lifestyle in order to avoid bringing on, or worsening, dizziness and imbalance that occurs with movement. As a result, decreased muscle strength and flexibility, increased joint stiffness, and reduced stamina can occur from this lifestyle. Treatment strategies used in rehabilitation can also be beneficial for these secondary problems.

WHAT IS VESTIBULAR REHABILITATION?

Vestibular rehabilitation (VR) is a specialized form of therapy intended to alleviate both the primary and secondary problems due to vestibular disorders. It is an exercise-based program primarily designed to reduce vertigo and dizziness, reduce gaze instability, and/or reduce imbalance and fall risk as well as address any secondary impairments that are a consequence of the vestibular disorder.

For most people who have a vestibular disorder, the deficit is permanent because the amount of restoration of vestibular function is very small. However, after vestibular system damage, symptoms can reduce and function can improve because of compensation. This occurs because the brain learns to use other senses (vision and somatosensory - body sense) to substitute for the deficient vestibular system. For many, compensation occurs naturally over time, but for patients whose symptoms do not reduce and who continue to have difficulty returning to daily activities, VR can assist in recovery by promoting compensation. [3]

The goal of VR is to use a problem-oriented approach to promote compensation. This is achieved by customizing exercises to address the specific problem(s) of each individual. Therefore, before an exercise program can be designed, a comprehensive clinical examination is needed to identify problems related to the vestibular disorder. Depending on the vestibular-related problem(s) identified, three principal methods of exercise can be prescribed:

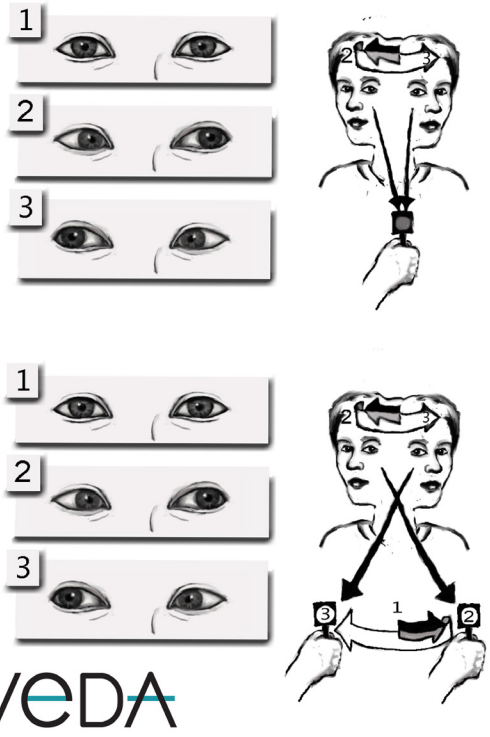
1) Habituation,
2) Gaze Stabilization, and/or
3) Balance Training. [4]

Habituation exercise is used to treat symptoms of dizziness that is produced because of self-motion[3] and/or produced because of visual stimuli. [5, 6] Habituation exercise is indicated for patients who report increased dizziness when they move around, especially when they make quick head movements, or when they change positions like when they bend over or look up to reach above their heads. Also, habituation exercise is appropriate for patients who report increased dizziness in visually stimulating environments, like shopping malls and grocery stores, when watching action movies or T.V., and/or when walking over patterned carpets and shiny floors. The goal of habituation exercise is to reduce the dizziness through repeated exposure to specific movements or visual stimuli that provokes patients' dizziness. These exercises are designed to mildly, or at the most, moderately provoke the patients' symptoms of dizziness. Over time, with good compliance and perseverance, the dizziness intensity can reduce due to the brain learning to ignore the abnormal signal.

Gaze Stabilization exercises are used to improve control of eye movements so vision can be clear during head movement. These exercises

are appropriate for patients who report problems seeing clearly because their visual world appears to bounce or jump around, such as when reading or when trying to identify objects in the environment, especially when moving about. There are two types of eye and head exercises used to promote gaze stability. The choice of which exercise(s) to use depends on the type of vestibular disorder and extent of the disorder.

One example (see image on the right):



Balance Training exercises are used to improve steadiness so that daily activities for self-care, work, and leisure can be performed successfully. Exercises used to improve balance should be designed to address each patient's specific underlying balance problem(s). [7] Also, to promote changes in balance, the exercises need to be moderately challenging, but safe enough so patients do not fall while doing them.

Additionally, balance exercises should be designed to reduce environmental barriers and fall risk. For example, the exercises should help improve patients' ability to walk outside on uneven ground or walk in the dark.

For patients with **Benign Paroxysmal Positional Vertigo (BPPV)**, the exercise methods described above are not appropriate to resolve this type of vestibular disorder. Through assessment, the type of BPPV is identified, and depending on the type, different repositioning maneuvers can be performed to help resolve the spinning that occurs due to position changes. [8, 9]

WHAT SHOULD PATIENTS EXPECT FROM VESTIBULAR REHABILITATION?

VR is usually performed on an outpatient basis, although in some cases, the treatment can be initiated in the hospital. Patients are seen by a licensed physical or occupational therapist with advanced post-graduate training.

VR begins with a comprehensive clinical assessment that should include collecting a detailed history of the symptoms and how these symptoms affect daily activities. The therapist will document the type and intensity of symptoms as well as discuss the precipitating circumstances. Additionally, information about medications, hearing or

vision problems, other medical issues, history of falls, previous and current activity level, and the living situation will be gathered. The assessment also includes administering different tests to more objectively evaluate the problems. The therapist will screen the visual and vestibular systems with various tests that observe how well eye movements, body movements and balance are being controlled by these systems. The examination may also include tests of: sensation (which includes gathering information about pain), muscle strength, extremity and spine range of motion, coordination and posture.

A customized exercise plan is developed from the findings of the clinical assessment, results from laboratory testing and imaging studies that may have been done, and input from patients about their goals for rehabilitation. An important part of the VR is to establish an exercise program that can be performed regularly at home. Compliance with the home exercise program is essential to help achieve rehab and patients' goals.

Along with prescribing and progressing exercise, patient and caregiver education is an integral part of VR. Education is important for patients because it takes away much of the mystery of what they are experiencing, which can help reduce any anxiousness that may occur because of the vestibular disorder.

ARE VESTIBULAR REHABILITATION EXERCISES DIFFICULT TO DO?

The exercises are not difficult to learn, but that doesn't mean they are easy to do!

The exercises can sometimes be tedious; however committing to doing them is key to helping you achieve success. Setting up a regular schedule so that you incorporate them into your day is very important.

Exercises may, at first, make your symptoms seem worse. But with time and consistent work, your symptoms should steadily improve, and then, you will find that you are able to participate more in the activities of your daily life.

FACTORS THAT CAN IMPACT RECOVERY

When patients participate in VR, different factors can impact the potential for recovery. For example, the type of vestibular disorder affects recovery. Patients that have a stable vestibular disorder, such as vestibular neuritis or labyrinthitis, have the best opportunity to have a satisfactory resolution of symptoms. When patients have a progressive vestibular disorder, like with multiple sclerosis, or a fluctuating condition, like with Migraine and Meniere's, which causes spontaneous attacks of dizziness or vertigo,

compensation can be difficult to achieve, and therefore, success with VR is more difficult. There are also differences in response to VR depending on whether you have one or both inner ears involved, or whether the problem lies within the vestibular parts of the brain as opposed to the ear(s).

Symptomatic relapses can occasionally occur because the brain de-compensates. This can be due to different emotional and/or physical stressors, like personal or job-related pressures, periods of inactivity, a bad cold or flu, extreme fatigue or chronic lack of sleep, changes in medication, or sometimes surgery. [3] Although it is important for patients to consult with their physician to make sure nothing new has occurred, returning to the exercises that promoted the initial compensation can help promote recovery again. Additionally, recovery after de-compensation usually occurs more quickly as compared to the initial compensation.

OTHER FACTORS THAT CAN POTENTIALLY LIMIT RECOVERY
Sedentary lifestyle
Pain
Presence of Other Medical Conditions
Certain Medications or Multiple Medications
Emotional Concerns
Decompensation

WHERE CAN I FIND A VESTIBULAR REHABILITATION SPECIALIST?

The Vestibular Disorders Association (VeDA) provides a directory of health professionals who are specially trained to assess and treat vestibular disorders. This online directory offers users the ability to search for providers according to specialty and geographical location. To locate this online directory, visit **vestibular.org/healthcare-directory**.

Light Sensitivity

By: Greg Bullock

The effects of chronic light sensitivity, also known as photophobia, are profound for those who experience it on a daily basis. It can cause pain in the eyes and brain, trigger physical symptoms of an underlying condition and even produce feelings of anxiety and isolation—and sometimes it only takes just a few seconds or minutes of exposure.

The connection between light sensitivity and certain vestibular disorders is no different. A significant number of patients with these conditions must also endure the challenges of being light sensitive. And in their case, light acts as a catalyst for dizziness, vertigo, lightheadedness, headache and other symptoms that are often associated with these disorders. In this article, we take an in-depth look at the experiences of those with vestibular-related photophobia and offer tips for keeping it in check.

VESTIBULAR MIGRAINE

It should come as no surprise that vestibular migraine carries perhaps the highest risk of light sensitivity symptoms. Migraine and headache disorders have long been associated with external sensitivities to light, noise and/or smell; and in fact, sensitivity to light is often cited as the second most common symptom during attacks after only head pain. In addition, photophobia is included as a key diagnostic indicator for migraine with and without aura, according to the International Classification of Headache Disorders (3rd Edition).

Patients with vestibular migraine (also known as migraine-associated vertigo) are similarly affected by photophobia. More than 90% have reported negative or painful reactions to light during their vestibular episodes, ahead of other issues like nausea and headache. Even between attacks, people with vestibular migraine can have a heightened sensitivity to their environment and feel discomfort under bright lighting. [1]

LIGHT SENSITIVITY & VESTIBULAR DISORDERS

More than 90% of people with **Vestibular Migraine** are sensitive to light during attacks

Exposure to bright light is a **known trigger** for vestibular symptoms

Light sensitivity affects patients with Vestibular Migraine, traumatic brain injuries, Meniere's Disease, and other vestibular disorders

TREAT LIGHT SENSITIVITY:

01 Receive a proper diagnosis

Avoid extended time in a dark room 02

03 Try FL-41 glasses for light sensitivity

Use vestibular rehabilitation therapy 04

Infographic designed by Gregory Bullock, Marketing Director for TheraSpecs

Similarly, we cannot discount the role of light exposure in the onset of vestibular migraine attacks. Bright and/or flashing lights are frequent triggers for more than one-quarter of patients with the condition. [1]

Light sources that may cause the most problems include:

- Fluorescent lighting
- Digital screens (computer, smartphone)
- Bright sunlight and outdoor glare

As a result, exposure to this and other forms of bright light can lead to hallmark vestibular symptoms (e.g. dizziness and lightheadedness) as well as typical migrainous features (e.g. head pain, visual aura and nausea).

TRAUMATIC BRAIN INJURIES AND POST-CONCUSSION SYNDROME

Traumatic brain injuries (TBI), regardless of severity, are one of the leading causes of vestibular complications, and they also can negatively impact a patient's tolerance for light as well. Not only are dizziness and light sensitivity regular symptoms within the first few days after a concussion (>40%), but their presence during this time may predict the likelihood of long-term symptoms a year or more later. [2] Sadly, they can also become a chronic problem for many, persisting for years after the initial trauma.

Only recently have researchers begun to explore how light sensitivity and other visual inputs can lead to the vestibular effects associated with brain injuries. One study found that more than half of teenage athletes who endured a sports concussion cited computer use and television watching as triggers of their dizziness. [3] While motion sensitivity may also be a compounding factor, it is likely that the artificial light exposure also contributed to these experiences. And this is reinforced by repeated findings that people with concussion have a lower threshold for bright light. [4,5] Much like migraine, this makes patients susceptible to having their post-concussion and post-TBI symptoms induced by light—even at levels that others may not consider bright at all!

MENIERE'S DISEASE

Another leading cause of dizziness and vertigo is Meniere's Disease, which is caused by a chronic buildup of fluid in the inner ear. You might be wondering then: How does an inner ear disorder translate to symptoms of light

sensitivity? The answer likely lies in the connection between Meniere's Disease and migraine.

Research has shown that people with Meniere's Disease are twice as likely to also have migraine. Even in the absence of a formal diagnosis of headache disorder, nearly half of Meniere's patients have at least one migrainous symptom as part of their vestibular attacks—including sensitivity to light. Even though people with migraine and vestibular migraine are far more likely to experience regular photophobia, evidence suggests that at least 40% of those with Meniere's are still subject to these sensitivities.

Similar to other vestibular conditions, Meniere's patients can also be triggered by visual environments that feature intense lighting and/or disorienting movement. Some examples include:

- Bright, flashing or flickering lights
- Supermarket or shop environments (with fluorescent lighting)
- Watching television or using the computer

Severe vertigo and unsteadiness are just two of the symptoms that they can experience as a result of being exposed to these types of environments. [7] While visual triggers may not be as prominent as other factors (such as stress or posture), they still cannot be discounted as significant concerns for those with Meniere's Disease.

OTHER VESTIBULAR DISORDERS

Other forms of vestibular dysfunction may also feel the negative impact of light and visual stimuli. Visual vertigo is a known side effect for many vestibular disorders, and it can be brought on by complex, distorted and/or moving imagery. Although not directly linked to light sensitivity, experts believe that harsh or high-contrast lighting (such as flashing emergency lights) can activate the central nervous system and ultimately compound the disorientation that patients may feel in these environments. [8]

In addition, dizziness and related vestibular symptoms have been shown to increase with age, regardless of cause. And light sensitivity has been anecdotally connected to the onset of dizziness and vertigo in older populations. In fact, here is how one patient described the feeling:

"Also the light, irritating the eyes. I don't look into the shop-window, instead I walk through dark or quiet streets..." [9]

No matter your age or vestibular condition, the unpleasantness of photophobia is often a debilitating complication.

HOW TO TREAT VESTIBULAR-RELATED LIGHT SENSITIVITY

Proper diagnosis. The first step of any treatment plan begins with appropriate diagnosis. Patients and their physicians need to know exactly what is causing the dizziness symptoms. This approach can thus lead to individualized therapies for specific symptoms while also addressing the primary disorder with time-tested treatment options.

Avoid dark adaptation. The instinct for many patients who suffer from light sensitivity may be to avoid light exposure by hiding in a dark space or wearing sunglasses inside. However, this can worsen a person's photophobia over time as their eyes adapt to the darkness. Recent CDC guidelines further reinforced patients to steer clear of "cocoon therapy" (staying in a dark room with no stimulation) and gradually re-introduce regular activities, particularly after a concussion.

FL-41 glasses. Dating back to early-1990's clinical research, a specialty tint known as FL-41 has provided therapeutic benefit to those with photophobia. The tint has been shown to decrease light-induced migraine attacks by 74% and help alleviate fluorescent- and artificial-light sensitivity. It has also been effective with thousands of patients with vestibular migraine, post-concussion syndrome and other vestibular disorders. The reason it is so effective has to do with its filtering of specific wavelengths of blue-green light—wavelengths which are known to activate sensitive brains and bring about symptoms of chronic vestibular and neurological conditions. 10,11

Vestibular rehabilitation. Another evidence-based treatment method is vestibular rehabilitation. This process utilizes simple exercises to improve vestibular symptoms like balance issues, visual complications and motion-induced dizziness. In addition, neurologic physical therapists (Neuro PTs for short) have specialization in neurological disorders like migraine and concussion, giving them additional insight into the unique complaints of patients. They can help retrain the brain through a combination of rehabilitation exercises, assistive technologies and other innovations in treatment.

Hormones and Vestibular Disorders

By P. J. Haybach, RN, MS

Health professionals and women with vestibular disorders have often suggested that hormones might play a role in Ménière's disease and other vestibular disorders.[1, 3, 10, 14] Little research has been done in this area, and not much information is available in print. The exact connection, if any, between hormones and vestibular disorders and the percentage of women affected is unknown. This article explores some of the possible links.

Hormonal changes do not produce one tidy set of symptoms occurring at a precise point in time. Some of these changes have been reported to increase symptoms, and some have been reported to reduce symptoms. The idea that hormones might increase symptoms in women or cause a physical problem is not limited to vestibular disorders. Other conditions thought to be linked in some way to hormonal changes in women include:

- Migraines. These occur more often in women, and attacks have been attributed to the use of birth control pills, pregnancy and delivery, estrogen-replacement drugs, the start of the first menstrual period, menopause, and menstruation in general.[11]
- Autoimmune diseases. These are seen in women nine times more often than in men.[3]
- Acoustic neuroma. Research suggests that these tumors grow faster in female test mice given estrogen than in mice not given estrogen.[17]

Rheumatoid arthritis, heart disease, osteoporosis, glaucoma, and Parkinson's disease may also be affected by hormones.[3, 6]

FEMALE HORMONES

Hormones are chemicals produced in one body organ and carried in the blood stream to another organ, where they cause functional activity or possibly a change in a structure.

Women manufacture a number of hormones specific to them, particularly during their childbearing years. The two best-known female hormones are estrogen and progesterone, sometimes also referred to as sex hormones. These are produced predominantly in the ovaries, although the adrenal glands and the placenta of pregnant women also make them. Some research has indicated that estrogen can be manufactured in the brain as well.[3]

Estrogen and progesterone are referred to as female hormones because they are found most abundantly in women, but men's bodies also produce small amounts of estrogen, and women also manufacture small amounts of the male hormone testosterone. Other female hormones include prolactin, oxytocin, luteinizing hormone, follicle-stimulating hormone, and human chorionic gonadotropin.

FEMALE HORMONE FUNCTION

Female hormones are responsible for the physical changes of puberty, the menstrual cycle, the changes of pregnancy, and milk production for nursing. The end of their production results in menopause.

Estrogen levels are highest on days 10-15 and lowest on days 1-5. The time between ovulation and menstruation is sometimes referred to as the luteal phase.

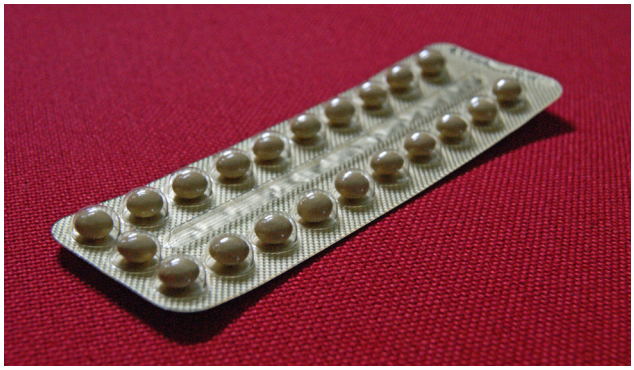
PREGNANCY

Human chorionic gonadotropins are produced throughout pregnancy. Estrogen and progesterone are at their lowest concentrations during the first eight weeks of pregnancy. They have a large increase between

THE MENSTRUAL CYCLE	
Days 1-5	Menstruation
Days 6-14	Increasing estrogen levels (estrogen stimulates egg production)
Day 14	Ovulation
Days 15-25	Increasing progesterone levels
Days 26-28	Falling progesterone and estrogen
Days 1-5	Cycle repeats if a fertilized egg is not imanted in the uterus

weeks 12 and 24. These higher levels of estrogen and progesterone are maintained from week 24 through the birth.

Oxytocin, produced at the end of the pregnancy by the brain and the placenta, induces contractions and assists in milk production. Prolactin, which is also responsible for milk production, is produced in larger amounts near the end of the pregnancy. It may be involved in the breast growth seen in pregnancy.



BIRTH CONTROL PILLS

Birth control pills contain estrogen and progestin (a hormone similar to progesterone) and are taken from day 1 of the menstrual cycle through day 24. Menstruation begins on the 29th day, which restarts the cycle. Birth control pills work by making the brain think from day 1 to day 24 that pregnancy has occurred, thus preventing ovulation. Birth control pills cause the level of prolactin to rise.[13]

PHYSICAL EFFECTS OF HORMONES

Although the primary effects of female hormones are related to reproduction, many of these hormones can have other effects through-out the body.

Among the general effects in the days before menstruation begins are that blood may become more viscous (thick),[1] appetite may be stimulated,[4] and the volume of cerebro-spinal fluid may increase.[9] Prolactin may be implicated in the breast swelling and tenderness some woman experience before menstruation. Pregnancy can cause nasal stuffiness, nasal allergy, and other allergy problems. It predisposes some woman to motion sickness.[10] Swelling around the Eustachian tube and a feeling of pressure or fullness in the ears may be caused by pregnancy[5] and the use of birth control pills.

Progesterone is capable of causing diuresis (ridding the body of some of its water).[13] It may increase body temperature[11, 13] and can cause hyperventilation (over-breathing, often in response to anxiety).[2, 11] Progesterone can produce dizziness, faintness, and sleepiness and might also have a depressant effect.[11] In some women, estrogen causes fluid retention before menstruation, during pregnancy, or while taking birth control pills. It may slightly increase serum triglycerides and decreases fasting levels of glucose and insulin in some women.[11] Estrogen might also have a role in brain function, such as solving spatial-orientation problems and how pain is sensed.[3] It might also cause a change in the auditory brainstem test.

INNER EAR-RELATED SYMPTOMS

A number of inner ear symptoms have been attributed to the female hormones. Women with vestibular disorders have reported discrete episodes of vertigo, general imbalance and/or disequilibrium, increases in tinnitus, hearing loss or change of some type, aural fullness, and ear pressure in apparent response to hormonal changes. Not only do these symptoms vary from one woman to the next, but the circumstances in which they are experienced also vary. Andrews et al. described women experiencing dizziness, aural pressure, and low-frequency hearing loss during the premenstrual time. They also found that those experiencing the largest fluid weight gain tended to have the most severe attacks at this time.[1] Abdel-Nabi et al. reported on women experiencing nausea, vomiting, vertigo, and dizziness in the premenstrual time.[13]

Naftalin and Mallett wrote about a woman who had a remission from symptoms during the beginning of pregnancy and an exacerbation during mid-pregnancy. They also described her as having worse symptoms during breastfeeding.[14]

Increased symptoms or the return of symptoms have been reported during the premenstrual time, menstruation, pregnancy, the use of birth control pills, and estrogen replacement therapy. Women have also occasionally reported improvement or remission of symptoms during pregnancy, while using birth control pills, during hormone replacement therapy, or during the premenstrual time. On the other hand, some women have reported that their vestibular impairments began during pregnancy or under one or more of these other conditions.

TESTING FOR HORMONE-PROVOKED INNER EAR SYMPTOMS

No single, specific test exists to determine whether or when a woman's inner ear symptoms are provoked by hormonal changes. In some research studies, blood levels of estrogen and progesterone have been compared with either hearing and vestibular tests or a symptom diary kept by the woman to determine whether a relationship exists. Two studies comparing electronystagmo-graphy (ENG) test results for groups of women found that their premenstrual and postmenstrual results differed. Unfortunately, this type of testing over time is seldom done. Instead, if a health care practitioner is interested, a woman might be asked to keep a diary noting her menstrual cycle and daily vestibular symptoms to determine whether the two are related.

TREATMENT FOR HORMONE-PROVOKED SYMPTOMS

Only one treatment has been described in the medical literature specifically for hormone-provoked vestibular and hearing symptoms. Price et al. described successful use of leuprolide acetate in one woman. Leuprolide acetate works by blocking gonadotropin-releasing hormone, a hormone partially

responsible for regulating estrogen and progesterone. Unfortunately, this drug could not be used on a long-term basis because of the possibility of side effects such as heart disease and osteoporosis.[15]

Andrews et al. described the use of dietary sodium restriction and diuretics in a woman with Ménière's disease who experienced increased symptoms premenstrually.[1]

Uchide et al. described decreasing sodium intake and using a diuretic, diazepam, and isosorbide (a fluid-manipulating drug) to treat increased symptoms during one woman's pregnancy.[19]

Hansen et al. described giving small doses of dimenhydrinate and meclizine hydrochloride during pregnancy but avoiding diuretics in the first trimester.

Some of the treatments reported anecdotally
by members of the Vestibular Disorders Association and others with vestibular disorders include the following:

- A low-sodium diet
- Six small meals per day (to spread intake
- of food and fluid over the entire day)
- Diuretics taken daily or taken during the premenstrual time
- Estrogen replacement therapy
- Birth control pills
- Antidepressant drugs
- Vestibular-suppressant drugs taken during times of increased symptoms

THEORIES ABOUT POSSIBLE LINKS

It must be kept in mind that Ménière's disease and some other vestibular disorders can wax and wane seemingly randomly; thus the possibility exists that inner ear symptoms apparently associated with hormonal changes in any particular woman can be explained by coincidence.

On the other hand, non-coincidental explanations have appeared in the medical literature. These include that of Abdel-Nabi et al., who attribute increased symptoms to inner ear swelling.[13] Also, Uchide et al. suggest that Ménière's disease might be expected to worsen during early pregnancy because suddenly decreasing serum osmolality would induce an osmotic gradient between the outer and inner endolymphatic sac. This would allow free water to enter the endolymphatic space and produce or exacerbate endolymphatic hydrops.[19] In other words, chemical changes that occur in the body during pregnancy cause water to move into sensitive parts of the inner ear. This additional water causes changes that produce symptoms such as

disequilibrium.

Andrews et al. offer multiple explanations:

- Thyroid changes caused by female hormones cause vestibular symptoms.
- "Endolymphatic hydrops represents a fluid imbalance within the inner ear and, when combined with an additional fluid shift, may produce symptomatic dysfunction."
- Aldosterone increases during the pre-menstrual time as estrogen also increases and progesterone decreases. Since aldosterone (as well as estrogen) causes water retention, the combined effect of these two substances causes a shift of water into sensitive parts of the inner ear. This produces vestibular symptoms.

SPECULATIONS BY THE AUTHOR

Here are other ideas that might explain an exacerbation of vestibular symptoms by hormonal fluctuations:

- The same factors causing increased motion sickness during the pregnancy of normal women may also cause vestibular symptoms in women with a vestibular disorder.
- Progesterone's depressant property may cause decompensation, which leads to vestibular symptoms. (Decompensation refers to a lessening of the brain's ability to compensate for incorrect balance information from a damaged inner ear. A variety of things can cause decompensation, which is usually temporary.)
- Increased triglycerides caused by increased estrogen levels during the menstrual cycle may lead to balance and hearing symptoms.
- Premenstrual blood viscosity (thickening) may compromise blood flow to the inner ear and cause changes in fluid balance. This could produce hearing and/or balance symptoms.
- Estrogen and/or progesterone might cause symptoms because of their chemical properties rather than their fluid-manipulating capabilities.
- Progesterone might cause hyperventila-tion during pregnancy and/or the premenstrual period and lead to feelings of dizziness and disequilibrium because of the effect on the brain.
- The increased amount of cerebrospinal fluid in and around the brain during the pre-menstrual period might cause an increase in pressure within the head. This pressure is passed into the inner ear through the cochlear aqueduct, a natural tunnel connecting the inner ear to the space around the brain that contains spinal fluid. The pressure might cause balance and/or hearing symptoms.
- The presence of estrogen increases fluids in the body. Therefore, a

woman experiencing symptoms because of a lessening of body fluids might improve temporarily whenever estrogen levels increased sufficiently.

- If prolactin can cause premenstrual breast swelling and general swelling during pregnancy, perhaps it causes the increased inner-ear symptoms women can experience at these times.
- Increases in estrogen levels perhaps cause symptoms because of an autoimmune reaction.
- Because human chorionic gonadotropins are present only during pregnancy, perhaps women who improve only during pregnancy do so because of their presence, for some unknown reason.
- Increased estrogen levels lead to a decrease in the blood sugar level, which perhaps causes dizziness and/or disequi-librium because of its effect on the brain.
- The oxytocin produced near the end of pregnancy releases prostaglandins, which might lead to hearing and/or balance symptoms.
- If a woman has balance or hearing symptoms only during lactation, perhaps oxytocin is the cause.
- The increased estrogen causes an increased appetite and increased eating. Women may eat or drink substances they are allergic to or that change the fluid balance in the inner ear and result in hearing and/or balance symptoms.
- Elevated levels of estrogen can cause swelling around the Eustachian tube, which interferes with middle ear function. This may, in some still undefined way, impact inner ear function and cause balance and/or hearing symptoms.
- A woman whose symptoms begin for the first time during labor or delivery may have suffered a perilymph fistula, an abnormal opening between the inner ear and middle ear caused by increased pressure from bearing down during delivery.

SUMMARY

Although many people have casually observed changes in hearing or balance that seem related to hormonal changes in women, it is unknown how many woman are affected, precisely what changes occur, what causes them, and what, if anything, the treatments should be.

Environmental Impacts on Vestibular Disorders

By Dr. Matthew G. Crowson, MD

Have you ever wondered if vestibular disorders and their symptoms are influenced by the environment? Recent work has demonstrated that the symptoms of common vestibular disorders may be linked with certain environmental factors.

ATMOSPHERIC PRESSURE

Changes in atmospheric pressure may affect patients with Mènière's Disease.[1]

In a recent study, investigators from Germany asked their patients with Mènière's Disease to keep a daily vertigo diary to document symptom flares. To test a hypothesis that changes in the weather solicit symptom flares, the study's investigators logged local hourly air pressure, as well as absolute and dew point temperatures over the time period the patients recorded symptoms in their vertigo diaries.

Interestingly, they found that the mean change in air pressure differed one day prior to onset of their patients' reported Mènière's Disease symptom flare.

Specifically, the authors noted an increase in symptoms after increase in air pressure, but not after a decrease in air pressure. This result was independent of the temperature and dew point measurements.

As there is little evidence published to date to suggest how an increase in air pressure might trigger Mènière's Disease symptoms, the proposed mechanism of atmospheric pressure change causing increased symptom flares in Mènière's Disease patients' warrants further exploration. The authors correctly point out that atmospheric pressure increases may affect other physiologic

processes that result in a symptom flare, so it is possible there is an alternate, but related explanation for the phenomenon observed in this study.

The German study is not the first time the idea of air pressure has been implicated in Mènière's Disease. Externally applied positive pressure therapy has been developed and commercialized for the treatment of Mènière's Disease. Positive pressure therapy works through a device not dissimilar to an aquarium pump, which emits small pulses of pressure through the ear canal and a ventilation tube placed in the tympanic membrane. The belief is that these small pulses of pressure may alter fluid dynamics within the inner ear, resulting in decreased symptoms. The efficacy of this technology has been questioned, however. A recent Cochrane Review of positive pressure therapy in Mènière's Disease found no evidence that it does not produce significant symptom improvement.[2]

ENVIRONMENTAL IMPACTS ON MIGRAINE

Sensitivity of health conditions and symptoms to weather or climate variation has been well described in qualitative patient symptom surveys.[5] Within the many health conditions surveyed, there is evidence to link migraine disorders and perturbations in weather patterns. Qualitative analyses of patient reported migraine triggers have noted that changes in weather precede migraine attacks second to psychosocial stress.[6] Migraine sufferers in northern climates have noted that migraine symptoms seem to occur more frequently in seasons with more daylight.[7] In a group of patients studied from the United States, migraine sufferers reported high humidity, low barometric pressure, and rainy days as having the ability to trigger migraine headaches.[8] There have also been objective reports of weather and climate change on migraine headache symptoms. Periods of meteorological phenomena of warm dry winds, known as the "Chinook Winds" in Canada, have correlated with a greater probability of migraine headache symptoms.[9]

What remains to be characterized is if weather patterns affect vestibular migraine as the pathophysiologic mechanism at play in classic migraine headaches may be shared similar. A common pathophysiologic link would suggest the triggers of migraine headache may also trigger vestibular migraine. However, further work is needed elucidate a role between weather and environmental factors and vestibular migraine.

ALLERGIES

Another major environmental research theme in Mènière's Disease has explored potential connections with allergic conditions. A report from the renowned House Ear Institute in Los Angeles compared the prevalence of allergic conditions in their patient population with Mènière's Disease to

those without Mènière's Disease.[3] In patients with Mènière's Disease, nearly 60% reported possible airborne allergies, 40% suspected food allergies, and 37% had had positive allergy tests. When the prevalence of these allergic conditions was compared to patients without Mènière's Disease, allergic conditions were significantly more prevalent in patients with Mènière's Disease.

A recent review of the evidence connecting allergic conditions and Mènière's Disease suggested that there is credible data to suggest patients with Mènière's Disease may have an enhanced allergic response.[4] While the authors could not conclude that there is a causal association between allergies and Mènière's Disease, they argue that practicing the principles of allergy control is a safe, relatively inexpensive adjunct to typical medical management. It remains to be seen whether the efficacy of this approach produces real benefits for patients with Mènière's Disease.

CONCLUSION

While further investigation is needed to pinpoint precise mechanisms tying environmental phenomena to Mènière's Disease, curious investigators have produced thought-provoking data to suggest possible associations. The discovery of such associations may open new frontiers for therapy in the comprehensive management of this often perplexing condition.

Natural Supplements for Vestibular Disorders

By Alicia Wolf

SUPPLEMENTS FOR VESTIBULAR MIGRAINE AND PERSISTENT POSTURAL PERCEPTUAL DIZZINESS (PPPD)

The following supplements are the most commonly recommended for migraine based on studies. Since vestibular migraine and PPPD can be comorbid, these supplements are usually prescribed for both, however if PPPD is caused by a different vestibular disorder, it may vary in combination.

Magnesium

It is challenging and expensive to measure magnesium accurately through blood tests, making low magnesium in the brain difficult to prove. Studies have found that many with migraine have low levels of magnesium in the brain and spinal fluid. This is why supplementation is important for those with migraine even if a deficiency may not be detected.

A daily dose between 400mg-800mg is recommended for migraine prevention by most clinics, including Johns Hopkins. Magnesium oxide is the most widely recommended due to its use in studies and its easy and

inexpensive availability, however it can cause diarrhea as a side effect. Because high doses are recommended for migraine prevention, a more easily absorbed form like magnesium glycinate may be better tolerated. Different types of magnesium may be recommended for different symptoms.

Riboflavin

A European study in 2004 found that migraine days were cut in half after 3 months and 6 months of daily use of 400mg of B2 Riboflavin. In addition, the number of abortive medications were also significantly reduced. This supplement was found to be well-tolerated by the participants in the study as well. There are also many natural sources of riboflavin that can be supplemented with diet, including eggs, lean protein, green vegetables, and fortified cereals and breads.

CoQ10

Two small, but effective studies have shown that CoQ10 may help with migraine prevention. In one study, patients took 150mg daily for 3 months and over half of them experienced a 50% reduction in migraine days without side effects. The other study used 100mg three times a day and compared it with a placebo. The CoQ10 was 3 times more effective at reducing migraine attacks than the placebo, yet some noted stomach upset as a side effect. Some of the side effects may be avoided by splitting the dosage throughout the day. It does interact with some medications.

Ginger

In a 2014 study, ginger was found to be as effective as sumatriptan in migraine patients without aura. Within 2 hours, pain severity was lessened. It is unclear whether this supplement helps with vestibular migraine dizziness without pain. 1/4 teaspoon of ginger powder is the recommended dosage, taken at the first sign of an attack.

Feverfew

An extremely small study of 8 patients showed that their headaches were unchanged, yet the frequency of headaches increased significantly when stopping feverfew to switch to the placebo. Some studies showed feverfew to be slightly more effective than the placebo. Symptoms of nausea and vomiting were also reduced. Other studies have shown it to not be effective for migraine prevention. Other than helping with nausea, there's no clear research on if this will help vestibular symptoms.

VESTIBULAR NEURITIS, BPPV, AND MENIERE'S

Vitamin D

Low levels of vitamin D have been found in patients with vestibular

disorders such as benign paroxysmal positional vertigo (BPPV), Meniere's Disease, and Vestibular Neuritis. A few reports have shown supplementation can be beneficial for preventing BPPV attacks and Meniere's symptoms. Dosage should be discussed with your doctor as it could depend on how deficient you are.

VESTIBULAR NEURITIS & MENIERE'S DISEASE

L-lysine

A naturally occurring amino acid, lysine has anecdotal evidence to be effective for tinnitus and treatment of vertigo.

Gingko Biloba

There is some research that points to gingko being helpful for enhancing cognitive function and improving memory loss. Some patients with tinnitus find it helps, while other research points to it being ineffective. It also thins the blood and is usually not recommended to be taken with other blood thinners. Preparations in the US vary, so it is important to look for labeling EGb 761, which is extracted from the leaves. Most studies of Ginkgo use between 120-240 mg a day. This could be effective for patients with multiple vestibular disorders.

Lemon Bioflavonoid

Although the research for this supplement isn't strong, it does appear to be helpful for some patients with Meniere's disease, particularly those with tinnitus as a symptom. This supplement is fairly well tolerated.

SUPPLEMENTS BY DIAGNOSIS	
Vestibular Migraine	• Magnesium • Riboflavin • CoQ10 • Ginger • Feverfew
PPPD	• Magnesium • Riboflavin • CoQ10 • Ginger • Feverfew
BPPV	• Vitamin D
Vestibular Neuritis	• Vitamin D • L-lysine • Gingko Biloba • Lemon Bioflavonoid
Meniere's Disease	• Vitamin D • L-lysine • Gingko Biloba • Lemon Bioflavonoid
Mal de Debarquement (MDDS)	• None
Acoustic Neuroma	• None

Currently there are no vitamin or supplement recommendations for Mal de Debarquement (MDDS) and Acoustic Neuroma.

ADDITIONAL RESOURCES	
Magnesium	thedizzycook.com/magnesium-supplements-explained-which-one-is-best-for-vestibular-migraine americanmigrainefoundation.org/resource-library/magnesium
Riboflavin	ncbi.nlm.nih.gov/pubmed/15257686
CoQ10	ncbi.nlm.nih.gov/pubmed/11972582
Feverfew	ncbi.nlm.nih.gov/pmc/articles/PMC3210009
Vitamin D	frontiersin.org/articles/10.3389/fneur.2019.00863/full
Gingko Biloba	dizziness-and-balance.com/disorders/menieres/treatment/men_alt.html
Lemon Bioflavonoid	jamanetwork.com/journals/jamaotolaryngology/article-abstract/599465https://www.dizziness-and-balance.com/treatment/drug/lipoflavonoids.html

Essential Oils

By Emily Englert, Certified Holistic Nutritionist

The information here is not intended to cure, treat, or prevent any disease. These comments have not been approved by the Food and Drug Administration and are not to be used as a substitute for medical care or medical advice. Consult your licensed medical professional before trying any product.

INTRODUCTION

Have you ever walked into a spa or hotel lobby and felt an almost immediate sense of calm? Or entered a store in the mall and instantly felt the need to just walk around and browse? Chances are, they are diffusing essential oils.

Essential oils are increasingly popular as people look for alternatives or natural remedies for their symptoms. Surprisingly, the pure oils extracted from plants have more than relaxing or invigorating aromatic properties. In pure form, they have been used for centuries as natural therapy for mental, emotional, and physical health. Oils can be uplifting, invigorating, and activating; waking up your mind and energizing your body to think, move, and create. Other oils have the opposite effect: they provide calm, relaxation, restfulness, stress relief, or sleep. The effects of natural, pure, and carefully harvested essential oils are being studied and promoted for a number of health benefits, including anti-bacterial, anti-viral, and anti-inflammatory effects depending on the origin of the extract.

Essential oils contain volatile organic compounds that exert a pharmacological effect by entering the body through the skin (aromatherapy massage or topical application), oral and mucous membranes (eating or inhaling), and olfactory administration (inhalation aromatherapy).

The classification of essential oils is based on the botanical classification of the plant from which the essential oils are extracted. The use of chemotypes is another classification of essential oils which describes the main compound within the essential oil in chemical terms.

DILATION AND DOSAGE

Frequently, essential oils are used at different concentrations depending on the route of administration:

1. For aromatherapy massage, 1–5% essential oil is used.
2. For oral administration, 8–50% essential oil is used.
3. Concentrated essential oil is used in inhalation aromatherapy.

However, the dosage and dilution of essential oils chosen have not been standardized in practice. More research needs to be done to determine the best dosage, use, and dilution of which oils, and for what benefit. The lack of standardization has not slowed the growth of this industry. People are experimenting with essential oils in a variety of ways, looking for a broad spectrum of benefits.

HOW AROMATHERAPY WORKS ON THE BRAIN

Aromatherapy is the most common use of essential oils. It is a simple and affordable way to use essential oils for general well-being.

In inhalation aromatherapy, the inhaled air containing essential oils reaches the circulation system via the blood capillary network in the nose and the bronchi in the lungs and also stimulates brain areas directly via the olfactory epithelium. Essential oils trigger mechanisms in the brain via the olfactory system. The mechanism of action of oils administered by inhalation involves stimulation of the olfactory receptor cells in the nasal epithelium, about 25 million cells, connected to the olfactory bulb. After stimulation, the signal is transmitted to the limbic system and hypothalamus in the brain through the olfactory bulb and olfactory tract. Once the signals reach the olfactory cortex, release of neurotransmitters, for example, serotonin, takes place, which results in the expected effect on emotions related to essential oil use. Increasing popularity of aromatherapy has been reported worldwide and is one of the most commonly used CAM therapies.

HOW ARE THEY USED?

Essential oils may be used in several different ways. People are most familiar with the fragrance of oils, and their pleasant aroma can be diffused or inhaled, applied topically to the skin, or ingested.

Electric diffusers can be purchased at every pharmacy now, along with essential oils that can be added to water and introduced to the air to fill a room with fragrance beyond what lighting a fragranced candle can do. Not only does it smell wonderful, but even if you cannot smell it, you may feel the relaxing or rejuvenating effect of inhaling the natural essential oil.

For a more direct application, the essential oil can be inhaled, usually by putting a few drops in your hands, rubbing them together, and inhaling while cupping your hands over your nose and mouth. A few slow, deep breaths can have an impact on your breath, your mood, and your energy level.

The most potent and effective way to experience essential oils is through oral administration in which the components of the essential oil reach the bloodstream. Since essential oils are lipophilic, they can easily be carried to all organs in the body. Ingesting essential oils is a way to receive the beneficial properties internally. Some oils can be given by dropfuls directly in the mouth, and others must be taken indirectly. By placing several drops of oils in an empty gelatin capsule, the oil can be used to treat the gut, or be absorbed through the gut for a more systemic effect. More and more people are ingesting essential oils to help with symptoms from digestive issues, intestinal irritation, inflammation, or pain.

COMMON ESSENTIAL OILS

Lavender and peppermint are two of the most common and most studied essential oils, each having a surprising number of health benefits.

Peppermint has proven to be a beneficial stimulant when inhaled. By activating the autonomic nervous system when inhaled, peppermint oil causes constriction of blood vessels, pain relief, a feeling of warmth, reduction of respiratory mucus and relief from symptoms of cough and cold.

Lavender is well known for its relaxing floral fragrance, and it has proven benefits of relaxation. This is why more and more hotels are putting lavender oil or lotion at the bedside as part of their turn-down service to provide a pleasant and relaxing sleep experience for their guests.
Ginger, rosemary, peppermint, turmeric, basil.....sounds like we are getting ready to follow a recipe! Yes, essential oils can be used in food preparation, and the internet has websites dedicated to teaching people how to use essential oils in their cooking. Recipes have been adapted to help people know how to measure precisely, because these undiluted extracts are much stronger

than the typical cooking extracts available at the grocery store.

Orange scented soaps, or lavender scented detergents have been around a long time, but using high-grade essential oils mixed with other natural cleaners may be a safe and earth-friendly way to clean your house.

Companies that produce the purest essential oils have their own lines of household cleaning products as well. Simple recipes can be found online to show you how to use drops of essential oils to make your own cleaning products. Many people who like the aromatic properties of the oils like to use the cleaning products, and many others are trying to move toward more natural products in the home.

Some essential oils, like rosemary, eucalyptus, and ginger have been shown to have analgesic (pain-relieving) and anti-inflammatory benefits when rubbed on the skin. Useful in conditions like arthritis or joint inflammation, oils can be delivered through the skin either alone or when mixed with another oil that acts as a carrier. Many popular arthritis creams or pain-relieving gels contain essential oils. Read the label of your favorite topical cream to see what essential oils it contains.

ARE THERE DIFFERENT QUALITIES OF OILS? WHICH ONE SHOULD I BUY?

With increasing demand for essential oils, and the rising popularity of Complementary and Alternative Medicine (CAM), new products are hitting the store shelves daily. The internet also provides countless ways to order products with essential oils.

COMMON ESSENTIAL OILS
Lavender
Peppermint
Basil
Rosemary
Wild Orange
Copaiba
Frankincense
Cilantro
Celery Seed
Clary Sage
Eucaplyptus
Ginger
Helichrysum
Melaleuca “Tea Tree”
Melissa
Oregano
Turmeric

Regardless of claims made, many oils are synthetic or nature-identical oils. While these may smell good and have the fragrance of the oil you are looking for, they have no therapeutic effect and may cause harm.

Only natural, organic oils that are certified and meet standard testing guidelines should be used. These oils are distilled, therapeutic-quality essential oils that retain all of the important therapeutic compounds necessary to provide health benefits. They are often labeled as medicinal quality or genuine essential oils.

ARE THERE ESSENTIAL OILS SPECIFICALLY FOR VESTIBULAR PROBLEMS?

Patients with vestibular issues may benefit from essential oils just like anyone else, to enhance mood, relieve anxiety, or improve sleep. There are a few manufacturers who have marketed products specifically for people with vertigo, nausea, and other vestibular symptoms, but these have not been independently tested for efficacy or usefulness in patients with vestibular disorders. In fact, some of these blends have been shown to be unsafe for pregnant women, infants, and children.

WARNINGS AND CONTRAINDICATIONS

Some of the essential oils mentioned here may be unsafe if used incorrectly.

This information is meant to be an introduction to the topic of essential oils and is not meant to be specific enough to suggest treatment or use in any way for any symptom or health benefit. While essential oils are generally safe and are increasingly being found to have real, scientific evidence to support their use for overall health, they are not without risks or side effects.

Some oils are toxic if ingested, unsafe for pregnant women, infants, children, or pets, or may interact with other medications. Due to the pure, concentrated nature of the extracted oils, some may cause irritation or burning of the skin, or result in sun-sensitivity where the skin can burn easily with sun exposure.

As research advances in this exploding area of complementary medicine, more information will become available to direct safe and effective use of these products. But remember, anything that has a real beneficial effect on the way one person feels can have an adverse effect on another person. The reality is, there just is not enough strong research to suggest that the claims you may read about online are correct. So, as with anything, be careful where you get your information, do your research, and discuss your plan with a licensed medical professional to make sure that you do not introduce anything that may be harmful for you into your treatment plan. In many cases,

when there is no published research to direct your practitioner, proceed with caution, follow manufacturer's directions, watch for adverse side effects, and discontinue immediately if any negative side effects occur.

Not all essential oils can be ingested. In fact, some are toxic.

TEST FOR SKIN IRRITATION

Eighty essential oils, including peppermint, lemon grass, sandalwood, ylang-ylang, lavender and jasmine, can cause skin irritation. If not diluted, some oils can burn the skin of children or others with thin or sensitive skin. All manufacturers recommend testing your skin tolerance to any essential oil with a skin patch test. In a skin patch test, you rub a drop of the oil on a small area of your inner forearm, cover it with a bandage, and examine it after 24 hours.

Also, essential oils are to be used on intact skin, in an area that does not have any breaks in the skin, or where you are using another topical medication. And be sure to keep it out of your eyes!

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