http://vestibular.org/migraine-associated-vertigo-mav

Migraine Associated Vertigo (MAV)

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.

Following is a video filmed by Dr. P. Ashley Wackym of Portland, Oregon's Ear & Skull Base Center, showing a patient who has suffered from vestibular migraine. When Dr. Wackym first started seeing this girl she had to be confined to a wheelchair due to her severe symptoms. View this and more videos by Dr. Wackym on his ***YouTube channel****.*

Migraine and vestibular dysfunction

Approximately 35% of migraine patients have some vestibular syndrome at one time or another. This may be prior to, during, after, or totally independent of their migraine headache event. Some interesting parallels exist between migraine and non-migrainous vestibular dysfunction. Many of the food and environmental triggers for migraineurs are the same as those for patients with non-migrainous vestibular dysfunction. Hormonal fluctuations 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] The clinical presentation of vestibular symptoms that often correlate with migraine[3] 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.

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. This presentation may result in a visit to the emergency room and extensive laboratory, imaging, and other diagnostic evaluations—often with normal results, which leads 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 fall risks.

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 (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 exertional migraine and benign orgasmic headaches, are becoming more frequently recognized (we have seen several patients with these variants who 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, is showing 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 vestibulo-pathy 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. Otolaryng-ologists 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 vestibular component 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.

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); audio-gram and ABR (auditory brainstem response test); functional balance and gait assessments with CDP (computerized dynamic posturography); and the newest test, VEMP.

In one 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 already started. Drugs used to prevent frequent migraine attacks include beta-blockers, tricyclics, antidepressants, calcium channel blockers, and certain anticonvulsant medications. 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]

Newer preventive medications, such as topiramate (an anticonvulsant), have been added to the physician's arsenal. In addition, the use of triptans and other abortive medications has nearly tripled in the past five years.

Vestibular rehabilitation

The benefits of vestibular rehabilitation are well documented to reduce symptoms and restore function for vestibular-related disorders.[7,13]

For patients who have alterations in oculomotor functions giving rise to visual perceptual deficits, a concentrated rehabilitation program consisting of VOR and gaze-stability exercises that emphasize visual acuity is effective. In cases where BPPV exists, performing canalith repositioning maneuvers is effective.

Postural instability and gait alterations respond to balance and gait-training tasks and 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.

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.

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 the effective reduction of the symptoms and limitations of function caused by migraine-related dizziness.[7]

Vestibular test results commonly observed in migraine-related dizziness patients

During **video-oculography**, a prevalent feature is poor gaze stability with ocular "drift," often accompanied by spontaneous up or down directional nystagmus, which does not suppress with fixation-suppression testing added. 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.

Saccadic eye-motion testing is usually normal, but a rebound nystagmus may be present. **Directional gaze testing** is usually abnormal, as is the **Halmagyi head thrust test**. With **Hallpike-Dix** positional testing (unless true BPPV presents), no rotational component nystagmus is usually evident.

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 symmetrically prolonged. **Caloric testing** does not match when all trials are compared.

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. **Subjective visual vertical assessment** with rotational chair testing appears to show promise as an adjunct assessment tool, although further assessment of this type of testing is necessary. The same is true for testing of **vestibular-evoked myogenic potentials** (VEMP).

Audiometric testing in cases of migraine-related dizziness typically reveals no changes in function. Tinnitus (most commonly associated with labyrinthitis rather than migraine), if present at all, is temporary.

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