Migraine-Associated Vertigo

Introduction

Background

Migraine is a disease characterized by periodic headaches, but patients often experience other symptoms, including dizziness. In some patients, dizziness can be the only symptom.

Since the 19th century, repeated references have been made to the clinical association of migraine and dizziness. Over the years, several syndromes have been reported of episodic vertigo associated with migraine. Some of these syndromes include benign paroxysmal vertigo of childhood and benign recurrent vertigo in adults. Some authors have even suggested an association between migraine and Ménière disease.

In 1984, Kayan and Hood reported a significant increase in the frequency of vertigo in people with migraines versus people with tension headaches. Verigo is also a known symptom of basilar artery migraine, which is a special form of migraine (see the International Headache Society classification of migraine, below). Although the definition of migraine-related vertigo and the continuum of the symptom complex remains poorly defined, the relationship is clearly more than a chance association.

One well-controlled study evaluated 200 patients from a migraine clinic, a dizziness clinic, and a control group from an orthopedic clinic. The group presenting with vertigo showed a higher lifetime prevalence of migraine (38%) than a similar group of patients in the control group (24%, \( P < 0.01 \)). Similar findings have been seen in studies evaluating migraine patients. Vertigo, as well as chronic nonspecific symptoms of vestibular system dysfunction, can be related to all forms of migraine.

The manifestations of migraine-associated vertigo are quite varied and may include episodic true vertigo, positional vertigo, constant imbalance, movement-associated dysequilibrium, and/or lightheadedness. Symptoms can occur prior to the onset of headache, during a headache, or, as is most common, during a headache-free interval. As such, many patients who experience migraines have vertigo or dizziness as the main symptom rather than headache. For this reason, this article is devoted to the description of migraine-associated vertigo.

Migraine headaches are recurrent headaches often accompanied by nausea and light sensitivity separated by symptom-free intervals. The headaches typically have a throbbing quality, are relieved after sleep, and may be accompanied by visual symptoms, dizziness, or vertigo. Patients often have a family history of migraine. Migraine can be divided into 2 categories, migraine without aura (common migraine, 90% of migraine headache cases) and migraine with aura (classic migraine, 10% of cases).

Basilar migraine, also known as Bickerstaff syndrome (1961), is an important variant of migraine with aura. Bickerstaff syndrome consists of 2 or more symptoms (ie, vertigo, tinnitus, decreased hearing, ataxia, dysarthria, visual symptoms in both hemifields of both eyes, diplopia, bilateral paresthesias or paresis, decreased level of consciousness) followed by a throbbing headache.

International Headache Society classification of migraine

- Migraine without aura (formally called common migraine)
Headache attacks last 4-72 hours untreated. In children younger than 15 years, headache may last 2-48 hours.

- Headache has at least 2 of the following characteristics:
  - Unilateral location
  - Pulsating quality
  - Moderate or severe intensity that inhibits or prohibits daily activities
  - Aggravation by walking up stairs or similar routine physical activity

- During headache, at least 1 of the following occurs:
  - Nausea and/or vomiting
  - Photophobia and phonophobia

- At least 1 of the following occurs:
  - History and physical examination findings do not suggest another disorder.
  - History and physical examination findings do suggest another disorder, but the other disorder is ruled out by appropriate investigations (eg, MRI or CT scanning of the head).

- Migraine with aura (formally called classic migraine)
  - Aura with at least 2 attacks of the following:
    - One reversible aura symptom indicating focal CNS dysfunction (ie, vertigo, tinnitus, decreased hearing, ataxia, visual symptoms in one hemifield of both eyes, dysarthria, double vision, paresthesias, paresis, decreased level of consciousness)
    - Aura symptom that develops gradually over more than 4 minutes or 2 or more symptoms that occur in succession
    - No aura symptom that lasts more than 60 minutes unless more than one aura symptom is present
    - Headache occurring before, during, or up to 60 minutes after aura is completed
  - Headache - Same as that for migraine without aura

- Migraine with prolonged aura - Fulfills criteria for migraine with aura but the aura lasts more than 60 minutes and less than 7 days

- Basilar migraine (replaces basilar artery migraine) - Fulfills criteria for migraine with aura but 2 or more aura symptoms of the following types occur: vertigo, tinnitus, decreased hearing, ataxia, visual symptoms in both hemifields of both eyes, dysarthria, double vision, bilateral paresthesias, bilateral paresis, and decreased level of consciousness

- Migraine aura without headache (replaces migraine equivalent or acephalic migraine) - Fulfills criteria for migraine with aura but no headache occurs

- Childhood periodic syndromes that may be precursors to or be associated with migraine

- Benign paroxysmal vertigo of childhood
  - Brief sporadic episodes of dysequilibrium, anxiety, and often nystagmus or vomiting
  - Normal neurologic examination findings
  - Normal findings on electroencephalography

- Migrainous infarction (replaces complicated migraine)
  - Patient has previously fulfilled criteria for migraine with aura.
  - The present attack is typical of previous attacks, but neurologic deficits are not completely reversible within 7 days and/or neuroimaging demonstrates ischemic infarction in relevant area.
  - Other causes of infarction are ruled out by appropriate investigations.

**Pathophysiology**

The pathophysiology of migraine-associated vertigo is not completely understood, however both central and peripheral defects
have been observed. In 1992, Cutrer and Baloh developed the most commonly accepted theory regarding the pathophysiology of migraine-associated vertigo. These authors propose that episodes of dizziness of a duration similar to that of a migraine aura (<60 min) that are time-locked with the headache most likely have the same pathophysiologic mechanism (eg, spreading wave of depression) as other aura phenomena.

According to the spreading depression theory, some type of stimulus (eg, chemical, mechanical) results in a transient wave front that suppresses central neuronal activity. This depression spreads in all directions from its site of origin. Neuronal depression is accompanied by large ion fluxes, including increases in extracellular K\(^+\) and decreases in extracellular Ca\(^{++}\). These changes result in a reduction in cerebral blood flow in the areas of spreading depression. However, most patients with migraine-associated vertigo have dizziness independently of the headache.

Cutrer and Baloh suggest that when dizziness is unrelated to headache, the dizziness occurs from the release of neuropeptides (ie, neuropeptide substance P, neurokinin A, calcitonin gene–related peptide [CGRP]). Neuropeptide release has an excitatory effect on the baseline firing rate of the sensory epithelium of the inner ear, as well as on the vestibular nuclei in the pons.

Asymmetric neuropeptide release results in the sensation of vertigo. When neuropeptide release is symmetric, the patient feels an increased sensitivity to motion due to an increased vestibular firing rate during head movements. Cutrer and Baloh also propose that CGRP and other neuropeptides may produce a prolonged hormonelike effect as these peptides diffuse into the extracellular fluid. This may explain the prolonged symptoms in some patients with migraine-associated vertigo, as well as the typical progression of persistent spontaneous vertigo followed by benign positional vertigo then motion sensitivity.

Some authors have suggested that peripheral cochleovestibular dysfunction in migraine patients may be attributed to vasospasm of the internal auditory artery causing ischemia to the labyrinth. Furthermore, Lee et al have reported a positive association of progesterone receptor (PGR) with migraine-associated vertigo.

Serotonin (5-HT) has also been found to be an important substrate in the development of migraine. Interestingly, 5-HT has direct effects on the firing rate of vestibular nucleus neurons. Both the serotonergic and the peptidergic pathways possibly play a role in the development of the short and prolonged periods of dizziness in migraine-associated vertigo. No single hypothesis explains the headache or dizziness process in migraine at this time. Thus, the causes of the symptoms of migraine remain controversial.

**Frequency**

**United States**

Migraine is an extremely common disorder worldwide. Migraine occurs in 18% of women and in 6% of men, totaling 25-28 million people in the United States alone. The disease is most prevalent in women of childbearing age, with an approximate prevalence of 25% in 35-year-old women. Overall, episodic vertigo occurs in about 25-35% of all migraine patients. Using these figures, roughly 3.0-3.5% of people in the United States have episodic vertigo and migraine. Comparatively, the prevalence of Ménière disease (a peripheral vestibular disorder with symptoms overlapping that of migraine-associated vertigo) is estimated to be 0.2% of the US population.

**Sex**

The epidemiology of migraine-associated vertigo corresponds to that of migraine in general. Migraine is present in 18% of females and in 6% of males aged 12-80 years. Peak ages are 30-45 years.

**Clinical**

**History**

As with any type of dizziness evaluation, the history is the most important means to diagnose migraine-associated vertigo. Patients with migraine-related vestibulopathy typically experience a varied range of dizzy symptoms throughout their life and even within individual attacks. These symptoms may be solitary or may be a combination of vertigo, lightheadedness, or imbalance. At the time of presentation, dizziness symptoms may have been present for a few weeks or for several years. Vertigo may occur spontaneously, provoked by head motion or provoked by visual stimuli. Symptoms may last for a few
minutes or may be continuous for several weeks or months. In women, dizziness may often occur during the menstrual cycle.

- Patients with migraine-associated vertigo often provide a long history of motion intolerance during car, boat, or air travel or all 3. Some patients are very sensitive to motion of the environment and to busy environments. Vertigo, which is an illusion of movement of the environment or of the patient in relation to the environment, is the most common type of dizziness reported. Vertigo is present at some time in approximately 70% of patients. The attacks of vertigo may awaken patients and are usually spontaneous, but they may be provoked by motion.

- The duration of the vertigo can also be quite variable. When vertigo is present, it may be indistinguishable from the spontaneous vertigo of Ménière disease. One clue that the vertigo is not of the Ménière type is that the vertigo of migraine-associated vertigo may last longer than 24 hours. In fact, a rocking sensation may be a continuous feeling for many weeks to months. In contrast, the vertigo of Ménière disease typically does not last longer than 24 hours. (For further information regarding migraine-associated vertigo and Ménière disease, see Differentials and Table 1). The frequencies of different durations of vertigo spells in migraine-associated vertigo are as follows:
  - A duration of seconds (7%)
  - A duration of minutes to up to 2 hours (31%)
  - A duration of 2-6 hours (5%)
  - A duration of 6-24 hours (8%)
  - A duration longer than 24 hours (49%)

- Unexplained sensorineural hearing loss has been variously reported in 0-31% of unselected patients with migraine. Changes in sensorineural hearing are rarely a significant feature of migraine-related vertigo and help to differentiate it from other causes of vertigo, especially Ménière disease. Up to 80% of patients with basilar migraine have been reported to have sensorineural hearing loss. The hearing loss of basilar migraine often affects the lower frequencies and may be bilateral. Fluctuation is also possible, similar to the sensorineural hearing loss of Ménière disease. Unlike in Ménière disease, the sensorineural hearing loss rarely progresses.

Table 1. A Comparison of the Symptoms of Migraine-Associated Vertigo and Ménière Disease

<table>
<thead>
<tr>
<th>Symptom</th>
<th>Migraine-Associated Vertigo</th>
<th>Ménière Disease</th>
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<tbody>
<tr>
<td>Vertigo</td>
<td>May last &gt;24 h</td>
<td>Lasts ≤24 h</td>
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<tr>
<td>Sensorineural hearing loss</td>
<td>Very uncommon; when present, often low frequency; very rarely progressive; may fluctuate in cases of basilar migraine</td>
<td>Nearly always progressive; most often unilateral; may be bilateral; fluctuation is common</td>
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<tr>
<td>Tinnitus</td>
<td>May be unilateral or bilateral; rarely obtrusive</td>
<td>May be unilateral or bilateral; often of significant intensity</td>
</tr>
<tr>
<td>Photophobia</td>
<td>Often present; may or may not be associated with dizziness</td>
<td>Never present unless a concurrent history of migraine exists</td>
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</table>

- A thorough headache history is also important when evaluating patients for possible migraine-associated vertigo. Many patients with recurrent headaches are unaware that their headaches may be from migraine. Therefore, the examining physician should have a thorough knowledge of the strict diagnostic criteria for migraine diagnosis (see the International Headache Society classification of migraine, in Background).

- Patients may or may not have a history of concurrent migraine headaches. In fact, most patients have dizziness symptoms during headache-free intervals or even numerous years following their last migraine headache. Some patients with migraine-associated vertigo have never experienced a migraine headache but have a family history of migraine.

- No diagnostic tests exist for migraine-associated vertigo. The diagnosis is made by clinical history or, when the history is
unclear, by a therapeutic response to treatment. A definite diagnosis of migraine-associated vertigo can be made when patients have migraine with aura that is accompanied by concurrent episodes of vertigo or when they have migraine without aura that is repeatedly associated with vertigo immediately before or during the headache.

- A probable diagnosis of migraine-associated vestibulopathy is suggested when patients experience recurrent or continuous vertigo or dizziness sensations without neurologic symptoms, when the dizziness is not time-locked to headache, when a past or family history of migraine headaches exists, and when the dizziness cannot be fully explained by other vestibular disorders. In these patients, a trial of migraine therapy can be started for both diagnostic and therapeutic purposes.

- Proposed criteria by Neuhauser and Lempert for diagnosis of definite migrainous vertigo are as follows:
  - Episodic vestibular symptoms of at least moderate severity (rotational vertigo, other illusory self or object motion, positional vertigo, head motion intolerance, ie, sensation of imbalance or illusory self or object motion that is provoked by head motion)
  - Migraine according to the IHS criteria
  - At least one of the following migrainous symptoms during at least 2 vertiginous attacks: migrainous headache, photophobia, phonophobia, visual or other auras
  - Other causes ruled out by appropriate investigations

- Proposed criteria for diagnosis of probable migrainous vertigo are as follows:
  - Episodic vestibular symptoms of at least moderate severity (rotational vertigo, other illusory self or object motion, positional vertigo, head motion intolerance)
  - At least one of the following: migraine according to the criteria of the IHS; migrainous symptoms during vertigo; migraine-specific precipitants of vertigo, eg, specific foods, sleep irregularities, hormonal changes; response to antimigraine drugs
  - Other causes ruled out by appropriate investigations

**Physical**

Findings on a complete neurotologic examination are often normal. Horizontal rotary spontaneous nystagmus may be present during an acute attack of vertigo. Dix-Hallpike examination may elicit symptoms of vertigo or nonvertigo dizziness, each without nystagmus.

**Causes**

Migraine headache and migraine-associated vertigo are often triggered by certain factors. These factors include stress, anxiety, hypoglycemia, fluctuating estrogen, certain foods, and smoking.

**Genetics**

The genetic cause of a rare type of migraine has been discovered. Familial hemiplegic migraine, a form of migraine with aura, is associated with mutations in the CACNA1A gene located on chromosome arm 19p13. This gene codes for a neuronal calcium channel. Defects involving this gene are also involved with other autosomal dominant disorders that have neurologic symptoms (see Table 2, below). One example is that of episodic ataxia type 2 (EA2), which is also known as periodic vestibulocerebellar ataxia and acetazolamide-responsive hereditary paroxysmal cerebellar ataxia). In cases of EA2, a pH abnormality has been discovered, and it often resolves with medication (eg, acetazolamide, valproic acid, calcium channel blocker).

<table>
<thead>
<tr>
<th>Gene Defect</th>
<th>Syndrome</th>
<th>Symptoms and Signs</th>
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<tbody>
<tr>
<td>Point mutation</td>
<td>Familial hemiplegic migraine</td>
<td>Episodic hemiparesis for ≤ 60 min followed by headache; gaze-evoked and downbeat nystagmus may persist after spells</td>
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http://emedicine.medscape.com/article/884136-print
Differential Diagnoses

Acute Laryngitis
Benign Paroxysmal Positional Vertigo
CNS Causes of Vertigo
Inner Ear, Labyrinthitis
Inner Ear, Meniere Disease, Medical Treatment
Inner Ear, Perilymphatic Fistula

Other Problems to Be Considered

The differential diagnosis of migraine-associated vertigo includes peripheral and central vestibular disorders. Peripheral disorders include Ménière disease, perilymphatic fistula, benign paroxysmal positional vertigo, recurrent vestibular neuritis, and recurrent vestibulopathy. Central disorders include multiple sclerosis, central paroxysmal positional vertigo, vertebrobasilar artery insufficiency, and cervico-medullary compression from abnormalities of the craniovertebral junction.

The principal differential is with Ménière disease. The overlapping symptoms of Ménière disease and migraine-associated vertigo include episodic vertigo, sensorineural hearing loss, and tinnitus. Differentiating migraine-associated vertigo from Ménière disease may be difficult because of the overlapping nature of the symptoms of these diseases. However, often the patient’s history offers clues that may help make the diagnosis. These differences are outlined in Table 1 in the History section.

The following is a list of symptoms that would support the diagnosis of migraine-associated vertigo as opposed to Ménière disease: photophobia, nonprogressive sensorineural hearing loss, vertigo of longer than 24 hours in duration, a long-standing history of motion intolerance, and dizziness occurring only during the menstrual cycle. Childhood benign positional vertigo is strongly related to migraine-related vertigo.

Migraine and vestibular disease can coexist. Patients who meet the clinical criteria for Ménière disease should be treated appropriately for Ménière disease, even if a history of migraine headache exists.

Workup

Imaging Studies

- MRI
  - An MRI of the brain with gadolinium is necessary when patients present with unilateral symptoms or signs or if the patient’s symptoms do not respond to appropriate treatment.
  - If symptoms are those of unilateral sensorineural hearing loss or tinnitus, the MRI should be directed to the internal auditory canals.

Other Tests

* Adapted from Tusa, 1999
† ENG
‡ SCA6
No pathognomonic abnormalities on either imaging or vestibular testing confirm migraine-associated vertigo. When the clinical history is wholly consistent, no other evaluation should be necessary to confirm the diagnosis.

Full audiometric evaluation, including pure-tone audiometry, word recognition scores, and reflex testing, is appropriate for any patient being evaluated for dizziness.

Other tests may include electronystagmography (ENG) and electrocochleography (ECoG).
  - ENG is typically not helpful in differentiating migraine-associated vertigo from Ménière disease. However, for patients with a several-year history of dizziness, normal findings on ENG are suggestive of migraine-associated vertigo.
  - Patients with a several-year history of Ménière disease often have a reduced vestibular response on at least one side. ECoG may help to differentiate Ménière disease and perilymphatic fistula from migraine-associated vertigo.
  - Celebisoy et al detected peripheral and central findings on balance function tests in 35 patients with migrainous vertigo. Of note, 20% exhibited caloric unilateral weakness while all the migraine patients in the control group without vertigo had normal caloric testing.

**Treatment**

**Medical Care**

Because most patients equate migraine with headache exclusively, convincing them that symptoms other than headache are due to migraine may be difficult. Dizziness secondary to migraine usually responds to the same treatment used for migraine headaches. The 3 broad classes of migraine headache treatment include a reduction of risk factors, abortive medications, and prophylactic medical therapy.

In general, drugs used to abort migraine headaches have not been found effective in treating dizziness secondary to migraine. Reduction of risk factors includes an attempt to avoid certain conditions (eg, stress, anxiety, hypoglycemia, fluctuating estrogen, certain foods, smoking) that can trigger migraine. Elimination of birth control pills or estrogen replacement products may be helpful. Specific foods to avoid are discussed in the Diet section. Prophylactic medications are described in the Medication section.

**Consultations**

Consultation with a neurologist is warranted if the patient has or develops focal neurologic deficits, if the patient develops migrainous infarction (see International Headache Society classification of migraine in the Background section), or if the examining physician is uncomfortable using prophylactic medications that may be appropriate in the treatment of migraine-associated vertigo.

**Diet**

Avoiding certain foods helps fewer than 25-30% of all people who experience migraines. In general, the following foods should be avoided: monosodium glutamate (MSG), certain alcoholic beverages (eg, red wine, port, sherry, scotch, bourbon), aged cheese (eg, Colby, Roquefort, Brie, Gruyere, cheddar, bleu, mozzarella, Parmesan, Boursault, Romano), chocolate (including carob), and aspartame. MSG is often found in certain soups, Chinese food and fast food, soy sauce, yeast, yeast extract, meat tenderizers, seasoned salt, and several salad dressings.

Because dietary restrictions are helpful in fewer than 25-30% of migraine cases, an elimination diet for 1 month may be prescribed. If, after 1 month, symptoms are not better, diet modification is not helpful. If foods are a trigger for symptoms, the offending food(s) can be identified by adding back one food at a time until symptoms return. A food diary is an alternative option to an elimination diet. A food diary may be helpful because certain foods cause migraine symptoms almost immediately (eg, red wine, MSG), whereas other foods (eg, chocolate, cheese) may cause symptoms the next day. The diary should include all foods consumed for 24 hours prior to the onset of a dizzy spell.

**Medication**
Prophylactic medical therapy should be used when migraine-associated vertigo occurs several times a month, is continuous over several weeks or months, or has severely affected the patient's lifestyle. First-line prophylactic medications include calcium channel blockers (verapamil), tricyclic antidepressants (nortriptyline), and beta-blockers (propranolol). Second-line treatment includes topiramate, valproic acid, venlafaxine, and methysergide. Acetazolamide has also been reported as an effective treatment by several authors.

The actual mechanism of action for migraine control with these medications is unknown. However, the calcium channel blockers, tricyclic antidepressants, beta-blockers, and methysergide are believed to block the release of neuropeptides into dural blood vessel walls because of their antagonist effect on 5-HT2 receptors.

One class of prophylactic medication does not seem to be more effective than the others. Therefore, unless contraindicated, verapamil is often used initially because this medication has the lowest side effect profile among the prophylactic medications. If dizziness is not controlled with one class of medication, another class should be used. If dizziness is controlled with one of these medications, the drug should be administered continuously for at least 1 year (except for methysergide, which requires a 3- to 4-wk drug-free interval at 6 mo). The medication can be restarted for another year if the dizziness returns after discontinuing therapy.

**Calcium channel blockers**

This agent inhibits calcium ions from entering slow channels, select voltage-sensitive areas, or vascular smooth muscle.

### Verapamil (Calan, Calan SR, Covera-HS, Verelan)

Relaxes smooth muscles and increases oxygen delivery during vasospasms.

**Dosing**

**Adult**

Starting dosage: 120-240 mg/d; not to exceed 480 mg/d; start at closest dosage in equivalent weight in pounds; titrate upward until symptoms relieved or severe constipation develops; clinical response usually observed in 2-8 wk after maximum tolerated dose started

**Pediatric**

Not established

**Interactions**

Verapamil may increase carbamazepine, digoxin, and cyclosporine levels; coadministration with amiodarone can cause bradycardia and a decrease in cardiac output; when administered concurrently with beta-blockers, may increase cardiac depression; cimetidine may increase verapamil levels; verapamil may increase theophylline levels

**Contraindications**

Documented hypersensitivity; systolic pressure of <90 mm Hg; cardiac conduction abnormalities; gastrointestinal obstruction; severe CHF; sick sinus syndrome; second- or third-degree AV block; hypotension

**Precautions**

**Pregnancy**

C - Fetal risk revealed in studies in animals but not established or not studied in humans; may use if benefits outweigh risk to fetus

**Precautions**

Hepatocellular injury may occur; transient elevations of transaminases with and without concomitant elevations in alkaline phosphatase and bilirubin have occurred (elevations have been transient and may disappear with continued verapamil
treatment); monitor liver function periodically

**Tricyclic antidepressants**

Mechanism of action is unknown. These agents inhibit the activity of such diverse agents as histamine, 5-HT, and acetylcholine.

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**Nortriptyline (Aventyl, Pamelor)**

By inhibiting the reuptake of serotonin and/or norepinephrine by the presynaptic neuronal membrane, this drug increases the synaptic concentration of these neurotransmitters in the central nervous system. Pharmacodynamic effects such as the desensitization of adenyl cyclase and down-regulation of beta-adrenergic receptors and serotonin receptors also appear to play a role in its mechanisms of action.

**Dosing**

**Adult**

10 mg/d PO for 1 wk initially; if tolerated, increase in 10- or 25-mg increments; not to exceed 100 mg/d; may have some benefit in 7-10 d but most require 4-8 wk at therapeutic dosages to observe beneficial effects. Alternatively, it can be started 25mg/d qhs for 3 weeks with increase in 25mg increments every 3 weeks up to 75mg/d.

**Pediatric**

Not established

**Interactions**

Cimetidine may increase nortriptyline levels when used concurrently; nortriptyline may increase prothrombin time in patients stabilized with warfarin

**Contraindications**

Documented hypersensitivity; history of cardiac disease/arrhythmia; closed-angle glaucoma; thyroid disease; ileus; urinary retention

**Precautions**

**Pregnancy**

D - Fetal risk shown in humans; use only if benefits outweigh risk to fetus

**Precautions**

Drowsiness, dry mouth, constipation, and photosensitivity are common side effects; urinary retention also possible; when discontinued, dosage should be tapered over 2 wk to prevent rebound cholinergic effects

**Beta-adrenergic blockers**

These agents are effective in prophylactic therapy, possibly by blocking vasodilators, decreasing platelet adhesiveness and aggregation, stabilizing the membrane, and increasing the release of oxygen to tissues.

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**Propranolol (Inderal, Betachron ER)**

Controls cardiac and psychomotor manifestations within minutes.

**Dosing**

**Adult**

20 mg PO q12h; increase in 20-mg increments q3-7d, depending on patient tolerance and heart rate; not to exceed 240-320
mg/d; use long-acting propranolol (80-120 mg cap) once appropriate dosage found; effect usually observed at 4 wk, but a 3-mo trial at max tolerated dose should be administered before considering patient a nonresponder

**Pediatric**

1 mg/kg/d PO divided bid initially; increase to maintenance dose of 2-4 mg/kg/d PO divided bid; not to exceed 16 mg/kg/d

**Interactions**

Coadministration with aluminum salts, barbiturates, NSAIDs, penicillins, calcium salts, cholestyramine, and rifampin may decrease propranolol effects; calcium channel blockers, cimetidine, loop diuretics, and MAOIs may increase toxicity of propranolol; toxicity of hydralazine, haloperidol, benzodiazepines, and phenothiazines may increase with propranolol

**Contraindications**

Documented hypersensitivity; uncompensated congestive heart failure; bradycardia; cardiogenic shock; AV conduction abnormalities; orthostatic hypotension; occlusive peripheral vascular disease

**Precautions**

**Pregnancy**

C - Fetal risk revealed in studies in animals but not established or not studied in humans; may use if benefits outweigh risk to fetus

**Precautions**

Adverse effects include fatigue (tends to diminish over the course of a few wk), 10% incidence of impotence, and depression; withdrawal should be tapered over a 2-wk period to prevent rebound headache or angina in patients with preexisting coronary artery disease

**Ergot alkaloids and derivatives**

These agents are direct vasoconstrictors of smooth muscle in cranial blood vessels. Their activity depends on the CNS vascular tone at the time of administration.

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**Methysergide (Sansert)**

Causes constriction of peripheral and cranial blood vessels.

**Dosing**

**Adult**

2 mg/d PO initially; increase in 2-mg increments q3-4d to maximum of 4-8 mg/d in divided doses for up to 6 mo; a drug-free interval of 3-4 wk should follow each 6-mo course of treatment; dosage should be gradually reduced in 2- to 3-wk period prior to beginning of drug-free interval to prevent rebound headaches; if improvement does not occur within 3 wk of initiation of treatment, methysergide should be weaned and discontinued

**Pediatric**

Not established

**Interactions**

None reported

**Contraindications**

Documented hypersensitivity; angina; coronary artery disease; hypertension; peptic ulcer disease; peripheral vascular disease; pregnancy; pulmonary disease; renal disease; rheumatoid arthritis; thrombophlebitis; valvular heart disease
**Precautions**

**Pregnancy**

X - Contraindicated; benefit does not outweigh risk

**Precautions**

Nausea/vomiting, diarrhea, heartburn, and abdominal pain are common adverse effects (may be minimized with food ingestion); retroperitoneal fibrosis, pulmonary fibrosis, or fibrosis of cardiac tissue rare (drug withdrawal usually reverses these conditions)

**Anticonvulsants**

Anticonvulsants, particularly those that interact with the GABA-ergic system, seem to have a positive effect in reducing migraine attacks.

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**Valproic acid (Depakote, Depakene, Depacon)**

Chemically unrelated to other drugs that treat seizure disorders. Although mechanism of action not established, activity may be related to increased brain levels of GABA or enhanced GABA action. Valproate may also potentiate postsynaptic GABA responses, affect potassium channel, or have a direct membrane-stabilizing effect.

**Dosing**

**Adult**

250 mg PO bid initially; titrate weekly to maximum 500 mg bid; use ER form (500-mg tab) once appropriate dosage found; titrate to maintain blood level at 75-100 µg/mL; effect may usually be observed at 4 wk

**Pediatric**

10-15 mg/kg/d PO for children > 2 y; increase dose by 5-10 mg/kg/d at weekly intervals to maximum 60 mg/kg/d depending on patient's symptoms; divide doses >250 mg/kg/d into bid/qid

**Interactions**

Coadministration with cimetidine, salicylates, felbamate, and erythromycin may increase toxicity; rifampin may significantly reduce valproate levels; in pediatric patients, protein binding and metabolism of valproate decrease when taken concomitantly with salicylates; coadministration with carbamazepine may result in variable changes of carbamazepine concentrations with possible loss of seizure control; valproate may increase diazepam and ethosuximide toxicity (monitor closely); valproate may increase phenobarbital and phenytoin levels, while either one may decrease valproate levels; valproate may displace warfarin from protein binding sites (monitor coagulation test results); may increase zidovudine levels in HIV seropositive patients

**Contraindications**

Documented hypersensitivity; hepatic disease; pancreatitis; thrombocytopenia; bone marrow suppression

**Precautions**

**Pregnancy**

D - Fetal risk shown in humans; use only if benefits outweigh risk to fetus

**Precautions**

Drowsiness, ataxia, anorexia, nausea, and vomiting may occur; 10% incidence of dose-related hand tremor reported; thrombocytopenia and abnormal coagulation parameters have occurred; risk of thrombocytopenia increases significantly at total trough valproate plasma concentrations >110 mcg/mL in females and 135 mcg/mL in males; at periodic intervals and prior to surgery, determine platelet counts and bleeding time before initiating therapy; reduce dose or discontinue therapy if hemorrhage, bruising, or a hemostasis/coagulation disorder occurs; hyperammonemia may occur, resulting in hepatotoxicity;
monitor patients closely for appearance of malaise, weakness, facial edema, anorexia, jaundice, and vomiting; may cause drowsiness

**Topiramate (Topamax)**

Indicated for migraine headache prophylaxis. The precise mechanism is unknown, but the following properties may contribute to its efficacy: (1) electrophysiological and biochemical evidence showing blockage of voltage-dependent sodium channels, (2) the augmentation of the activity of the neurotransmitter GABA at some GABA-A receptor subtypes, (3) the antagonization of the AMPA/kainate subtype of the glutamate receptor, and 4) the inhibition of the carbonic anhydrase enzyme, particularly isozymes II and IV.

**Dosing**

**Adult**

Slowly titrate upward at a minimum of 1 wk intervals as follows:
- Week 1: 25 mg PO qhs
- Week 2: 25 mg PO bid
- Week 3: 25 mg PO qAM and 50 mg PO qhs
- Week 4: 50 mg PO bid
- Week 5: 50 mg PO qAM and 75 mg PO qhs
- Week 6: 75 mg PO bid

**Pediatric**

Not established

**Interactions**

Phenytoin, carbamazepine, and valproic acid can significantly decrease topiramate levels; topiramate reduces digoxin and norethindrone levels when administered concomitantly; concomitant use with carbonic anhydrase inhibitors may increase the risk of renal stone formation and should be avoided; use topiramate with extreme caution when administering concurrently with CNS depressants since they may have an additive effect in CNS depression, as well as other cognitive or neuropsychiatric adverse events.

**Contraindications**

Documented hypersensitivity

**Precautions**

**Pregnancy**

C - Fetal risk revealed in studies in animals but not established or not studied in humans; may use if benefits outweigh risk to fetus

**Precautions**

Risk of developing a kidney stone formation is increased 2-4 times over that of an untreated population (the risk may be reduced with increased fluid intake); caution in renal or hepatic impairment; patients taking topiramate should seek immediate medical attention if they experience blurred vision or periorbital pain; continued usage after symptoms develop can lead to glaucoma; the primary treatment is discontinuation of topiramate; if left untreated, serious sequelae, including permanent vision loss, may occur; oligohidrosis and hyperthermia have been reported, predominantly in children, during vigorous exercise or exposure to warm environmental temperatures (ensure proper hydration prior and during activity and warm temperatures); may cause hyperchloremic, non–anion gap metabolic acidosis, acute or chronic metabolic acidosis resulting in hyperventilation, and nonspecific symptoms, such as fatigue and anorexia, or more severe adverse effects, including cardiac arrhythmias or stupor; chronic, untreated metabolic acidosis may increase nephrolithiasis or nephrocalcinosis risk, osteomalacia (ie, rickets in pediatric patients), or
Follow-up

Further Outpatient Care

Vestibular rehabilitation therapy is recommended when movement-associated dysequilibrium is present. Movement-associated dysequilibrium may be the predominant symptom, or it may be a continuing symptom despite adequate vertigo control with prophylactic medication. In either case, vestibular rehabilitation often is quite beneficial. Vestibular rehabilitation is not indicated for the treatment of spontaneously occurring vertigo.

Patient Education

For excellent patient education resources, visit eMedicine's Brain and Nervous System Center and Headache Center. Also, see eMedicine's patient education articles Migraine Headache, Vertigo, Dizziness, and Understanding Migraine and Cluster Headache Medications.

References


15. Headache Classification Committee of the International Headache Society. Classification and diagnostic criteria for


**Keywords**

migraine-associated vertigo, migraine associated vertigo, migraine, vertigo, migraine vertigo, headaches, dizziness, benign paroxysmal vertigo of childhood, benign recurrent vertigo in adults, basilar artery migraine, basilar migraine, Bickerstaff syndrome, episodic true vertigo, positional vertigo, constant imbalance, movement-associated dysequilibrium, common migraine, migraine without aura, classic migraine, migraine with aura, migraine with prolonged aura, migraine aura without headache, migraine equivalent, acephalic migraine, migrainous infarction, complicated migraine, familial hemiplegic migraine, migraine headaches, migraine headache, migraines

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