

CNS Causes of Vertigo

Marcelo B Antunes, MD, Resident Physician, Department of Otorhinolaryngology-Head and Neck Surgery, University of Pennsylvania Health System

Michael J Ruckenstein, MD, MSc, FACS, FRCS, Professor, Residency Program Director, Department of Otorhinolaryngology-Head and Neck Surgery, University of Pennsylvania Health System

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Introduction

Background

Dizziness is a vague and nonspecific symptom. It refers to an abnormal sensation in relation to space and position. Vertigo is a specific type of dizziness that is defined as a spinning or rotatory sensation. Patients with vertigo report that things are rotating around them or that they are rotating around things.

Vertigo could be either from a peripheral (labyrinth and vestibular nerve) or a central disorder (central nervous system). Central vertigo is usually a result of an abnormal processing of the vestibular sensory input by the central nervous system.

Pathophysiology

The sensation of balance is the result of appropriate information detected by the vestibular, ocular, and proprioceptive sensory receptors that is then properly integrated within the cerebellum and brain stem. Proper gait, posture, and visual focus during head movement all depend on an intact sense of balance. Loss of sensory information, central integration, and output control mechanisms all result in a sense of imbalance.

Central causes of vertigo result from either a disruption of central integrators (ie, brain stem, cerebellum) or a sensory information mismatch (ie, from the cortex). Lesions that affect the vestibular nerve or root entry zone (ie, cerebellopontine angle [CPA] lesions) result in imbalance by affecting primary vestibular sensory information.

Race

No racial predilection exists for CNS causes of vertigo.

Sex

Men and women are affected differently by different causes of CNS vertigo. Vestibular migraine, for example, shows a predilection for women.

Age

CNS causes of vertigo typically affect older population groups because of the associated risk factors of vascular causes of vertigo, such as hypertension, atherosclerosis, and diabetes mellitus.

Younger population groups are more commonly affected by migraine headaches and multiple sclerosis (MS). Cerebellar tumors affect a bimodal population of children and adults. CPA tumors typically affect people in the fifth to eighth decades of life.

Clinical

History

The history is of critical importance to determine if the vertigo is from a peripheral or central origin because usually physical findings and vestibular testing can only provide supportive information. Peripheral vertigo could be positional, lasting for seconds to a few minutes (such as in benign paroxysmal positional vertigo); recurrent, lasting for hours and associated with hearing loss, aural fullness, and tinnitus (such as in Meniere syndrome), or a single episode lasting days without associated auditory or neurological symptoms (such as in vestibular neuronitis).

Central vertigo should be considered when the patient's history is not consistent with any of the well-defined peripheral syndromes. The history could be acute or chronic vertigo, usually associated with neurologic symptoms. The presence of neurologic symptoms such as headaches, aura, visual, sensory or motor symptoms is more suggestive of a central disorder.^[1,2,3]

Physical

The physical examination should also focus on findings that are consistent with either peripheral or central vestibular disorders. Findings suggestive of peripheral vestibular dysfunction include a positive head thrust test indicative of an abnormal vestibuloocular reflex (VOR), turning greater than 45° on the Fukuda stepping test, ability to perform tandem gait testing with eyes open but not closed, or a positive Dix-Halpike maneuver suggestive of benign positional vertigo (BPV).

Findings suggestive of central pathology are abnormalities on the neurologic examination such as diplopia, dysarthria, aphasia, weakness, and sensation abnormalities.

Signs of a cerebellar dysfunction such as dysmetria on finger-to-nose testing and dysdiadochokinesia are also characteristic of a central problem.^[3,4]

One of the most helpful tools during physical examination is an evaluation of any nystagmus. Nystagmus is defined as a repetitive involuntary movement of the eyes. This is initiated by the slow phase, which is generated by the underlying disorder. The slow phase takes the eye away from the preferred position, and the fast phase (or saccade) brings the eye back to the visual target. The direction of the fast phase describes the direction of the nystagmus; the velocity of the nystagmus is determined by the slow phase.^[5,6]

Causes

Migraine-related vertigo or vestibular migraine

Migraine is a neurovascular disorder of the central nervous system that is caused by constriction and dilatation of intracranial blood vessels. The problem appears to be a dysfunction of ion channels in the aminergic nuclei of the brainstem and

diencephalon. It is characterized by recurrent episodes of headaches, usually unilateral, and throbbing, with associated phonophobia, photophobia, and nausea. Headaches may occur with or without prodromal symptoms (aura). The aura consists of neurologic symptoms, mostly visual (such as scotomas-flashing or colored lights), photophobia, and/or paresthesias, likely a result of decreased local cerebral perfusion that passes across the cortex. Vestibular symptoms such as dizziness, vertigo, and tinnitus are less common but are increasingly being recognized.

Migraine affects about 6-20% of men and 18-29% of women.^[7,8]Vertigo is 3 times more common in patients who suffer from migraine than controls. Therefore, establishing which patients have migrainous vertigo (MV) and which ones just have these conditions as separate entities is important.

The relationship between vertigo and headache is variable.^[9]Some patients experience the vertigo and headache as a completely separate event. In others, the headache can occur simultaneously with vertigo (25% of patients) or prior to the onset of vertigo (25% of patients). Vestibular symptoms precede headache in 50% of cases. Head motion intolerance is present in 30% of patients and typically persists after the acute attack of vertigo subsides. The duration of the vertigo can last from seconds (10%), minutes (30%), hours (30%) to days (30%).^[9]Therefore, 10-30% of patients present with symptoms that are of typical duration of a migraine aura (5-60 minutes).

Most patients present with other migrainous symptoms that include photophobia, phonophobia, osmophobia, visual, or other auras. Photophobia is the most prevalent, present in 70%. These symptoms are extremely important to recognize because sometimes they are the only connection between the vertigo and migraine. Also, the patients should be asked about triggering factors such as food, hormonal changes (menstrual cycle), excessive stress, and sensory stimuli.

Hearing loss and tinnitus are not prominent symptoms. Hearing loss, when present, is usually mild and transient, without progression in the course of disease. Some patients can present with fluctuating hearing loss during the episodes, confusing the diagnosis with Meniere disease. Around 20% of patients report aural pressure for seconds to minutes at the beginning of the acute attack.^[9]

Benign paroxysmal vertigo of childhood is considered to be an early manifestation of MV and a predictor for the development of migraine headaches. It is characterized by vertigo with nystagmus, nausea/vomit, and anxiety in an otherwise healthy child. Many of these children present with migraine later in life.^[10]

A consensus about the diagnosis and treatment of migrainous vertigo is evolving. Like migraine itself, MV is diagnosed based on history and physical examination. Several authors proposed different criteria in retrospective studies. One proposed criteria defines definite MV and probable MV:^[11]

- Definite migrainous vertigo
 - Episodic vestibular symptoms of at least moderate severity ("moderate" is considered to interfere with daily activities)
 - Current or previous history of migraine per International Headache Society criteria

- One of the following symptoms during 2 or more vertigo attacks: migrainous headache, photophobia, phonophobia, visual or other auras
- Other causes of vertigo ruled out after appropriate investigation
- Probable migrainous vertigo
 - Episodic vestibular symptoms of at least moderate severity ("moderate" is considered to interfere with daily activities)
 - One of the following: current or previous history of migraine per International Headache Society criteria; migrainous symptoms during a vestibular symptoms; migraine precipitants of vertigo in more than 50% of the attacks (food, sleep disturbances, hormonal changes); response to migraine medications in more than 50% of the attacks
 - Other causes of vertigo ruled out after appropriate investigation

Notice that the response to treatment is not considered a diagnostic criteria.

Basilar migraines are relative rare causes of migraines that involve pathology within the posterior circulation of the brain. Multiple neurologic symptoms including vertigo, dysarthria, dysphagia, and paresthesias precede the onset of the headache.

Examination of patients with a history of migrainous vertigo is typically normal. Spontaneous or positional nystagmus may be visualized during an acute attack. ENG testing may be normal or reflect a peripheral vestibular loss or central pathology.^[5]

Treatment of headaches includes modification of lifestyle, elimination of trigger factors, treatment of acute attacks, and prophylactic medications, when indicated. Evidence-based guidelines for the management of migraine headaches have been published and are reviewed elsewhere. Prophylactic medications aim to reduce frequency of attacks and severity and may be indicated, depending on the number and severity of attacks, degree of disability, and response to acute attack treatments. The options include propranolol, valproate, amitriptyline, fluoxetine, gabapentin, and, more recently, topiramate. About 60% of patients have a reduction in symptoms.

Similarly to headaches, the vestibular symptoms of migrainous vertigo are managed with treatment of acute attacks and prophylaxis.^[12] Acute attacks are managed initially with a vestibular suppressant.^[13] The use of triptans for migraine-related vestibular symptoms is not well studied and may or may not benefit vertiginous attacks. Prophylactic treatment is indicated with frequent, severe, and disabling attacks. Beta-blockers, tricyclic antidepressants, and calcium-channel blockers are useful in preventing vestibular symptoms. Antiepileptic drugs such as valproate, gabapentin, and topiramate are effective and well tolerated. Changes in lifestyle and avoidance of trigger factors are important in preventing vestibular symptoms.

Vertebrobasilar disease

The vertebrobasilar system provides blood supply to the occipital, parietal and temporal lobes, thalamus, inner ear, cerebellum, and brainstem. The most common causes of vertebrobasilar disease (VBD) are embolism, large artery atherosclerotic disease, small (penetrating) artery disease, and arterial dissection.^[14] Pathology within the vertebrobasilar system results in either recurrent episodes of transient neurologic deficits lasting minutes (vertebrobasilar insufficiency), TIA, or CVA.

Up to 25% of patients with VBD present with isolated vertigo. More frequently, VBD manifests with a combination of dizziness/vertigo, hearing loss, tinnitus, headache, nausea, visual disturbances, gait disturbances, paresthesias, motor weakness, and drop attacks. VBD symptoms are summarized into the “four Ds”: dizziness, diplopia, dysphasia, and drop attacks. Neurologic examination could be normal between attacks. Usually patients have risk factors for CVA and atherosclerosis, such as diabetes, hypertension, hyperlipidemia, hypercholesterolemia, hyperviscosity, and hypercoagulability.

Several specific syndromes are associated with ischemia to the territory supplied by anterior inferior cerebellar artery (AICA) or posterior inferior cerebellar artery (PICA).^[15,16] The territories vary among individuals; therefore, the clinical findings vary depending on the areas supplied by the ischemic vessel. Ischemia to the AICA distribution causes pontine syndrome.^[16] The AICA supplies the lateral pons and gives off the internal auditory artery (IAA). Occlusion of this artery produces ischemia of the entire labyrinth and a degree of pontine ischemia, producing a syndrome that manifests as vertigo, tinnitus, and hearing loss. If the occlusion is distal, only the labyrinthine blood supply is compromised, producing symptoms and ENG findings that are identical to a peripheral disorder.

Ischemia to the PICA distribution causes Wallenberg syndrome (lateral medullary syndrome).^[15] It is characterized with acute vertigo, nausea, and vomiting; ipsilateral facial pain and numbness; ipsilateral ataxia and decreased contralateral pain; and temperature sensation over the rest of the body; and ipsilateral Horner syndrome. Patients can also present with laryngeal and pharyngeal paralysis, leading to hoarseness and dysphagia.

In all patients in whom vertebrobasilar disease is suspected, imaging should be obtained. Imaging should include a MRI scan of the brain, as well as imaging of the vasculature with either a MR angiogram or a CT angiogram of the head and neck. A transcranial ultrasound that reveals flow in the vertebral arteries may also be helpful.

Treatment of VBD consists of treating the underlying pathology. Treatment is focused on controlling the risk factors for CVA and antiplatelet agents such as aspirin, dipyridamole, and pentoxifylline.^[17] Chronic dizziness and disequilibrium usually improves with vestibular physical therapy. Treatment of TIA and stroke are beyond the scope of this review.

Prognosis of ischemia to the posterior circulation differs significantly from anterior circulation. Acute basilar artery occlusion carries a very poor prognosis with a mortality rate higher than 80%. On the other hand, unlike the cerebral cortex, the brainstem is relatively resistant to ischemia, and recanalization therapy up to a period of 24 hours can still reverse the symptoms and sequelae.

Arnold-Chiari malformation

Arnold-Chiari Malformation consists of an abnormal displacement of the cerebellum and brainstem through the foramen magnum. The degree of displacement classifies the malformation in types I through IV. The most common is the type II, in which the cerebellar vermis, lower pons, and medulla is displaced.^[18]

The associated symptoms are a result of the compression of the cerebellum and brainstem structures and stretching of cranial nerves. Patients can present with vertigo, ataxia, dysequilibrium, sensorineural hearing loss, headache, cranial nerve dysfunction (such as unilateral hypoglossal palsy), cervical pain, or weakness.

The diagnosis can be made by MRI, with the sagittal view being the most helpful in determining the degree of herniation.^[19] The treatment is surgical. The results in terms of alleviating the vestibular symptoms are not well established, but case reports show some improvement.

Multiple sclerosis

Multiple sclerosis is an idiopathic demyelinating disorder of the central nervous system. It is thought to be an autoimmune disease, likely due to an autoantigen to one of the myelin proteins. The demyelination occurs in different areas of central nervous system, most commonly in the white matter, with formation of plaques that disrupt signal conduction and lead to symptoms.

Typical onset is with optic neuritis, but in 5% of the cases vertigo is the presenting symptom. Over 50% of patients present with vertigo at one point in the course of the disease. The vertigo can last from hours to days. Nystagmus, when present, can be consistent with peripheral or central lesion. MS can mimic an VIIIth nerve lesion, when a plaque develops in the entry of the nerve root in the brainstem, producing vertigo, unilateral caloric weakness, and horizontal nystagmus.^[20]

The diagnosis is made with imaging and CSF exam. On MRI, plaques can be seen in the white matter. On CSF, elevated levels of IgG can be identified. The treatment options are limited. Exacerbation episodes are treated with high-dose steroids and other immunosuppressants.^[3]

Workup

Laboratory Studies

Few laboratory studies facilitate the diagnosis of the CNS causes of vertigo.

If vertigo is accompanied by prolonged nausea and vomiting in elderly patients, monitoring and replacing fluids and electrolytes is prudent.

In the rare case of suspected Lyme neuroborreliosis, serology for Lyme disease with enzyme-linked immunosorbent assay (ELISA), Western blot analysis, and lymphocyte antigen stimulation assay are indicated. Obtain cerebrospinal fluid for Lyme antibody tests and polymerase chain reaction analysis to evaluate for *Borrelia burgdorferi* DNA.

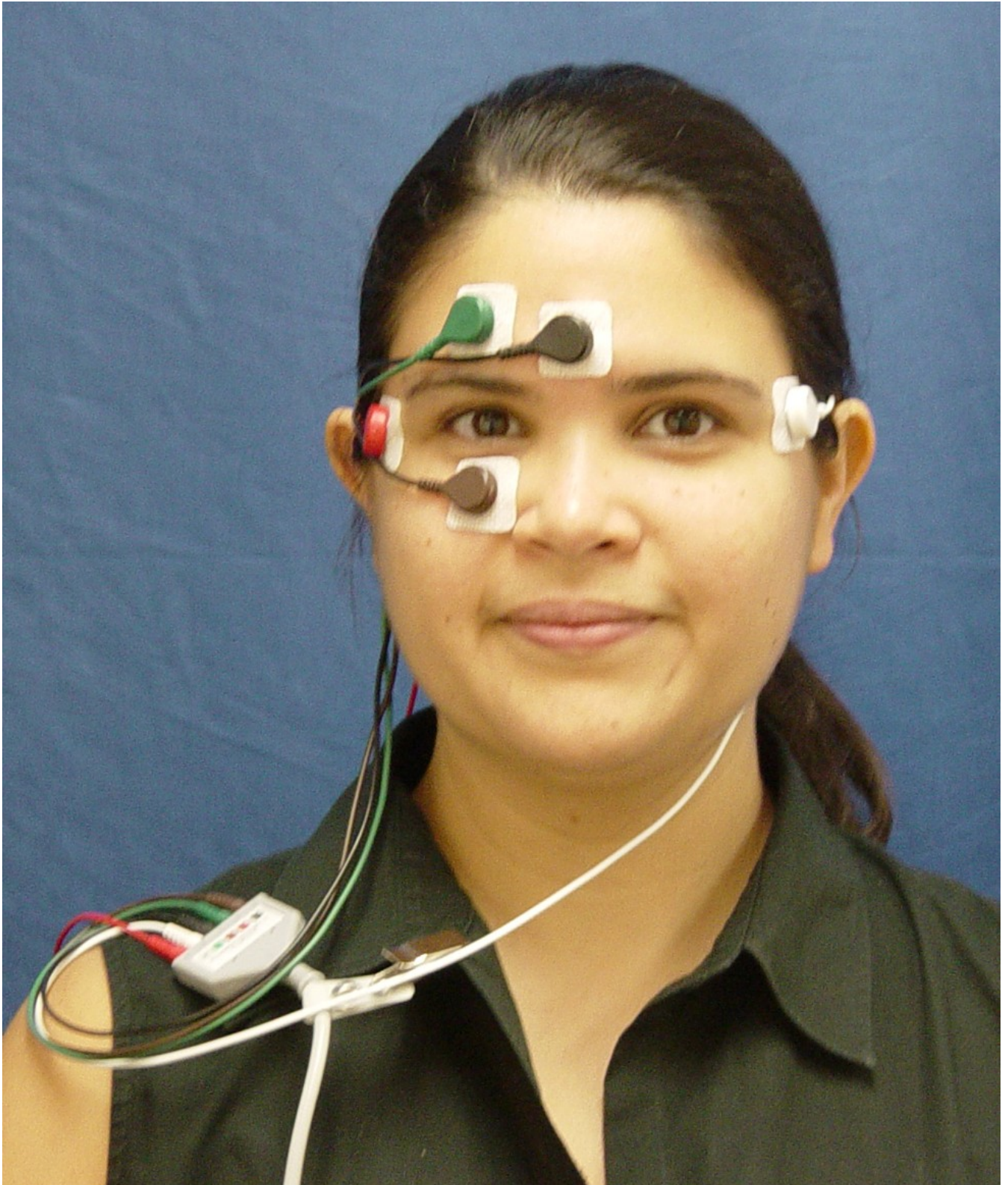
Imaging Studies

Imaging studies are indicated when the symptoms are suspected to result from ischemia. MRI and MR angiography are the most helpful studies in assessing posterior circulation disorders and acute infarction. Diffusion-weighted MRI is sensitive and specific for early detection and differentiation between vasogenic and cytotoxic edema in patients with acute neurologic deficits.

Other Tests

Electronystagmography (ENG) is the most used vestibular test. When combined with the patient's history and examination, the results of the ENG can be used to support a diagnosis of a peripheral or central etiology.^[1,2] Nystagmus patterns may be

spontaneous or may be elicited by a change in gaze, head position, or head shake. Patterns of central and peripheral nystagmus were discussed in the Clinical section. Abnormalities in smooth pursuit or saccades are typically central in origin.



Electrode montage for electronystagmography (ENG) testing.

Formal evaluation with vestibular testing is indicated if the diagnosis is not apparent after obtaining a history and performing a physical examination. Vestibular testing can facilitate distinction between central, peripheral, and mixed causes of imbalance and vertigo. The test battery assesses labyrinthine function with caloric testing, rotational chair testing, and vestibular evoked myogenic potential. Oculomotor integrity is evaluated with eye tracking during smooth pursuit, saccades, and optokinetic stimulation. The evaluation of spontaneous and gaze-evoked nystagmus can provide critical clues to central pathology.

Abnormalities found by oculomotor testing that suggest a central balance problem include saccade inaccuracy and smooth pursuit dysmetria. Failure to suppress nystagmus with visual fixation is often a sign of disease that affects the cerebellar flocculus or neural connections between the flocculus and the vestibular nuclei.

Positional testing with infrared oculography can be used to reveal nystagmus and to clearly define nystagmus patterns. Multidirectional nystagmus, spontaneous nystagmus, or positional nystagmus that is downbeat, torsional, or dissociated suggests a central lesion.

If symptoms suggest hypoperfusion, embolic events, or arrhythmia as the cause, perform a complete cardiac and peripheral vascular examination, including ECG, Holter monitoring, echocardiography, and carotid and vertebral Doppler ultrasonography.

Treatment

Medical Care

Medical treatment includes supportive care with fluid replacement and vestibular suppressants for intractable vertigo with nausea and vomiting.

Treatment of migraine-associated vertigo includes analgesics and vestibular suppressants. Drugs useful in the treatment of migraines include sumatriptan, propranolol, imipramine, amitriptyline, and nortriptyline, and the vestibular suppressants diazepam and alprazolam. For further information on the diagnosis and treatment of migraine headaches, please refer to the eMedicine topic Migraine Headache .

Control of hypertension, diabetes mellitus, and atherosclerosis is indicated for long-term prevention of further complications. Cardiac arrhythmia should also be controlled.

Drugs useful in the treatment of vertebrobasilar insufficiency include aspirin, ticlopidine, pentoxifylline, heparin, and warfarin.

Acetazolamide is indicated for the treatment of familial ataxia syndrome.

Surgical Care

Surgical treatment of central vertigo is limited to urgent posterior fossa decompression of cerebellar and brainstem edema that complicates the infarction.

Cerebellopontine angle (CPA) tumors are surgically removed on an elective basis. If a medical contraindication exists, radiotherapy for tumor control is an option.

Consultations

Otolaryngologists, neurosurgeons, neurologists, and cardiologists are consulted for further diagnosis and treatment of vertigo of CNS origin.

Diet

Address dietary management of migraine-associated vertigo with the patient. Avoidance of triggers, such as red wine, chocolate, and cheese, may reduce the frequency of attacks.

Medication

The goal of pharmacotherapy is to reduce morbidity and prevent complications.

Antihistamines

These agents prevent the histamine response in sensory nerve endings and blood vessels. They are also effective in treating vertigo.

Meclizine (Antivert, Antrizine, Meni-D)

Decreases excitability of middle ear labyrinth and blocks conduction in middle ear vestibular-cerebellar pathways. This decrease is associated with therapeutic effects in the relief of nausea and vomiting.

Dosing

Adult

25 mg PO q4-6h

Pediatric

>12 years: Administer as in adults

Interactions

May increase toxicity of CNS depressants, neuroleptics, and anticholinergics

Contraindications

Documented hypersensitivity

Precautions

Pregnancy

B - Fetal risk not confirmed in studies in humans but has been shown in some studies in animals

Precautions

Caution in angle-closure glaucoma, prostatic hypertrophy, pyloric or duodenal obstruction, and bladder neck obstruction

Dimenhydrinate (Dimetabs, Dramamine)

1:1 salt of 8-chlorotheophylline and diphenhydramine thought to be useful in the treatment of vertigo.

Diminishes vestibular stimulation and depresses labyrinthine function through central anticholinergic effects. However, prolonged treatment may decrease rate of recovery of vestibular injuries.

Dosing

Adult

50 mg PO or IM q4-6h or 100-mg supp q8h

Pediatric

Neonates: Contraindicated

2-5 years: Up to 12.5-25 mg PO q6-8h; not to exceed 75 mg/d

6-12 years: 25-50 mg PO q6-8h; not to exceed 150 mg/d

>12 years: Administer as in adults

Alternatively, 1.25 mg/kg or 37.5 mg/m² IM qid; not to exceed 300 mg/d

Interactions

Alcohol or other CNS depressants may have additive effect on dimenhydrinate; may mask ototoxic symptoms caused by certain antibiotics, and irreversible damage may result

Contraindications

Documented hypersensitivity; do not administer to neonates (IV products may contain benzyl alcohol, which has been associated with fatal gasping syndrome in premature and LBW infants)

Precautions

Pregnancy

B - Fetal risk not confirmed in studies in humans but has been shown in some studies in animals

Precautions

Do not treat severe emesis with antiemetic drugs alone; may contain sulfites or tartrazine, which may cause allergic-type reactions in susceptible persons; may impede diagnosis of conditions such as brain tumors, intestinal obstruction, and appendicitis; may obscure signs of toxicity from overdosage of other drugs

Anticholinergics

These agents work centrally by suppressing conduction in the vestibular cerebellar pathways.

Scopolamine (Isopto)

Blocks action of acetylcholine at parasympathetic sites in smooth muscle, secretory glands, and the CNS. Antagonizes histamine and serotonin action.

Transdermal scopolamine may be most effective agent for motion sickness. Use in vestibular neuronitis is limited by slow onset of action.

Dosing

Adult

0.6 mg PO q4-6h or 0.5 mg transdermally q3d

Pediatric

6 mcg/kg/dose IM/SC, not to exceed 0.3 mg/dose; alternatively, 0.2 mg/m² IM/SC and repeat q6-8h

Interactions

Antipsychotic effectiveness of phenothiazines may be decreased by coadministration with scopolamine; anticholinergic adverse effects may be increased by concurrent therapy, and phenothiazine dosages should be adjusted as necessary; coadministration with tricyclic antidepressants may increase anticholinergic adverse effects (eg, dry mouth, constipation, urinary retention) because of additive effect (tricyclic antidepressants with less anticholinergic activity may be beneficial)

Contraindications

Documented hypersensitivity; primary glaucoma (including initial stages); pyloric obstruction; toxic megacolon; hepatic disease; paralytic ileus; severe ulcerative colitis; renal disease; obstructive uropathy; myasthenia gravis

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

Caution in the elderly patients because of increased prevalence of glaucoma; large doses may suppress intestinal motility and precipitate or aggravate toxic megacolon; anticholinergics may aggravate hiatal hernia associated with reflux esophagitis; patients with prostatism can have dysuria and may require catheterization; use cautiously in asthma or allergies; reduction in bronchial secretions can lead to inspissation and formation of bronchial plugs

Benzodiazepines

By binding to specific receptor sites, these agents appear to potentiate the effects of γ -aminobutyric acid (GABA) and facilitate inhibitory GABA neurotransmission and other inhibitory transmitters. These effects may prevent vertigo and emesis.

Diazepam (Valium)

Depresses all levels of CNS (eg, limbic and reticular formation), possibly by increasing activity of GABA. Individualize dosage and increase cautiously to avoid adverse effects.

Dosing

Adult

5-10 mg PO/IV/IM q4-6h

Pediatric

>6 months: 0.05-0.3 mg/kg/dose IM/IV over 2-3 min; repeat in 2-4 h prn
Alternatively, 0.12-0.8 mg/kg/d PO divided q6-8h; not to exceed 10 mg/dose

Interactions

Increases toxicity of benzodiazepines in CNS with coadministration of phenothiazines, barbiturates, alcohols, and MAOIs

Contraindications

Documented hypersensitivity; narrow-angle glaucoma

Precautions

Pregnancy

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

Precautions

Caution with other CNS depressants, low albumin levels, or hepatic disease (may increase toxicity)

Phenothiazines

These agents are effective in treating emesis, possibly because of effects in dopaminergic mesolimbic system.

Promethazine (Phenergan)

For symptomatic treatment of nausea in vestibular dysfunction.

Antidopaminergic agent effective in treating emesis. Blocks postsynaptic mesolimbic dopaminergic receptors in brain and reduces stimuli to brainstem reticular system.

Dosing

Adult

25-50 mg PO/IM/PR q4-6h

Pediatric

>2 years: 0.25-1.0 mg/kg PO/IV/IM/PR 4-6 times/d prn

Interactions

May have additive effects when used concurrently with other CNS depressants or anticonvulsants; coadministration with epinephrine may cause hypotension

Contraindications

Documented hypersensitivity; children younger than 2 y (incidences of death due to respiratory depression)

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

Some adverse effects include CNS depression, dry mouth, extrapyramidal symptoms, hypertension, and rash; caution in cardiovascular or hepatic disease, seizures, sleep apnea, and asthma; may enhance effects of other medications that cause CNS depression, including alcohol, narcotics, sedatives, and hypnotics

Prochlorperazine (Compazine)

May relieve nausea and vomiting by depressing reticular activating system and blocking postsynaptic mesolimbic dopamine receptors through anticholinergic.

Dosing

Adult

5-10 mg PO/IM q6h or 25-mg supp PR q12h

Pediatric

2.5 mg PO/PR q8h or 5 mg q12h prn; not to exceed 15 mg/d

0.1-0.15 mg/kg/dose IM; change to PO as soon as possible

IV dosing not recommended for children

Interactions

Coadministration with other CNS depressants or anticonvulsants may cause additive effects; coadministration with epinephrine may cause hypotension

Contraindications

Documented hypersensitivity; bone marrow suppression; narrow-angle glaucoma; severe liver or cardiac disease; coma or large amounts of CNS depressants (eg, alcohol or barbiturates)

Precautions

Pregnancy

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

Precautions

Adverse effects include CNS depression, blurred vision, and hypotension; neuroleptic malignant syndrome and extrapyramidal dystonic reactions may rarely occur

Drug-induced Parkinson syndrome or pseudoparkinsonism develops quite frequently; akathisia is most common extrapyramidal reaction in elderly patients; lowers seizure threshold and should be used cautiously with history of seizures

Monoaminergics

These agents may treat vertigo, possibly through modulating the sympathetic system.

Ephedrine (Pretz-D)

Stimulates release of epinephrine stores, producing alpha- and beta-adrenergic effects.

Dosing

Adult

25 mg PO q4-6h

Pediatric

2-5 years: 3 mg PO q6-8h

>5 years: 6.25 mg PO/SC q6-8h

Interactions

Theophylline, atropine, or MAOIs may increase toxicity; alpha- and beta-blockers decrease vasopressor effects; cardiac glycosides and general anesthetics increase cardiac stimulation of ephedrine

Contraindications

Documented hypersensitivity; angle-closure glaucoma; cardiac arrhythmias

Precautions

Pregnancy

B - Fetal risk not confirmed in studies in humans but has been shown in some studies in animals

Precautions

Adverse effects include excitation, tremulousness, insomnia, nervousness, palpitation, tachycardia, and symptoms associated with sympathetic activation; bladder sphincter spasm can occur and cause transient acute urinary retention; caution in elderly patients and patients with diabetes mellitus, hyperthyroidism, hypertension, cardiovascular disease, prostatic hypertrophy, or cerebrovascular insufficiency

Follow-up

Further Outpatient Care

After the underlying cause of vertigo has been identified and treated, supportive care with vestibular suppressants and antiemetics are indicated to relieve the vertigo. Early initiation of vestibular rehabilitation is an important and effective intervention.

Patient Education

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

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Keywords

CNS causes of vertigo, vertigo, dizziness, imbalance, disequilibrium, migraine-associated vertigo, presyncopal, vertebrobasilar artery insufficiency, vestibular function, VBA insufficiency, posterior fossa cerebrovascular disease, cerebellar tumors, temporal lobe tumors, brainstem lesions, cerebellopontine angle tumors, CPA tumors, multiple sclerosis, posttraumatic vertigo, familial periodic ataxia syndromes, Lyme neuroborreliosis, psychogenic vertigo, migraine-associated vertigo, migraine, migraine headache, nystagmus