Vertigo

Vertigo Algorithm

Vertigo

R/O
1. Orthostasis
2. Cardiac arrhythmia
3. CVA
4. Multiple sclerosis
5. CNS drugs

Positional or
+ Dix-Hallpike

Ent Exam (1)

Observe (1)
or Refer for
Semont/Epley Maneuver

Recent viral illness

Audio if hearing decreased

Observe (2)

Persistence
> 6 weeks

Ent Referral

Vertigo Diagnosis Algorithm

Vertigo

History & Physical Examination
1. Vertigo Summary: Vertigo is so non-specific that it is a diagnostic dilemma, not a treatment dilemma. The 3 common labyrinthine causes are Meniere's disease, viral labyrinthitis and benign positional vertigo. The following is reproduced from the Clinical Manual of Otolaryngology-Head and Neck Surgery.

Vertigo is a feeling that the world is spinning around. People with extreme vertigo feel nauseous, often vomit, and talk about lying down and holding onto the carpet to keep from falling off the earth. Many patients complain of dizziness rather than of a true whirling sensation. A whirling sensation is usually associated with an identifiable labyrinthine etiology. The patient who complains of being dizzy may have a clear-cut and identifiable significant disorder, but often the diagnosis may remain
obscure. Dizziness takes a long time to evaluate, and may require a complete history, physical and laboratory examination. Time spent on the history will help direct the physician in decisions regarding testing and treatment. Failure to be thorough will result in a missed diagnosis. Physicians in different specialties have different experiences with vertigo. A triage officer at a Veterans Administration hospital, for example, may cite the leading causes of vertigo as cardiac arrhythmia and orthostatic hypotension. A neurologist might consider multiple sclerosis the most common cause, while a head and neck surgeon might believe that Meniere's disease or vestibular neuronitis is most common. A general practitioner may believe most causes are idiopathic or functional. Each of these physicians reflects the nature of his or her own practice. Table 2.4 is the evaluation for each of my patients who complains of dizziness; it can be used as a guide for developing personal approaches. For differential diagnosis see algorithm on Vertigo-Diagnosis.

Table 2.4

Work-Up for Vertigo

A. History
   1. Vertigo (what does the patient mean by dizziness?)
      a. Onset
      b. Intensity
      c. Duration
      d. Association with nausea and vomiting
      e. Feeling of faintness or loss of consciousness
   2. Hearing loss
   3. Tinnitus
   4. Feeling of fullness in ear
   5. History of ear pain, infection, surgery
   6. Recent illness
   7. Current medications
   8. Previous neurologic disorders (transient ischemic attack, stroke, multiple sclerosis, migraine headache)

B. Examination
   1. Hearing (tuning forks)
   2. Otoscopic
   3. Ophthalmic (to include extraocular movements, examination for nystagmus, and retinoscopy)
   4. Cranial nerves, with particular attention to nerves 3,4,5 (especially corneal branch), 6,7,9, and 10
   5. Neck examination (to recognize carotid artery disease) and range of motion.
   6. Blood pressure (to consider hypertension and orthostatic changes
   7. Pulse (to diagnose arrhythmia)
   8. Neurologic (to exclude neurologic disease, especially multiple sclerosis and a cerebrovascular accident)

C. Laboratory Tests
   1. Complete blood cell count (to rule out anemia)
   2. Electrolytes (to detect any imbalance)
3. Calcium (to detect hypercalcemia)
4. Tetraiodothyronine, T4 and TSH (to detect hypothyroidism)
5. FTA-ABS or TPA (to rule out tertiary syphilis)
6. Cholesterol and triglycerides (to detect hyperlipoproteinemia)
7. Tests for diabetes and reactive hypoglycemia
8. Electrocardiogram with rhythm strip (to diagnose any cardiac disease in elderly patients or with history suggestive of cardiac dysfunction)
9. Audiogram and tympanogram (to evaluate hearing as well as evaluate type of loss) and BERA (to evaluate retrocochlear sensorineural hearing loss (Fig. 1-9)
10. Electronystagmogram (to evaluate labyrinthine function). This test measures gaze nystagmus, spontaneous nystagmus, positional nystagmus, and response to caloric irrigation. It is extremely useful to identify labyrinthine disease and also helps localize lesions in either the labyrinth, the acoustic nerve, or the central nervous system.
11. MRI scan with gadolinium of internal auditory canal indicated when acoustic neuroma, cerebellar-pontine angle tumor, multiple sclerosis or other central problem suspected.
12. X-rays of the cervical spine. The cervical spine is closely connected to the labyrinth via a vestibulospinal reflex arc. Cervical spine disease can cause vertigo and hence this must be evaluated.

D. **Differential Diagnosis** (see algorithm Vertigo-Diagnosis)

This is not intended as an exhaustive differential plan, but rather to provide some insight into the different diseases that can cause vertigo. If the investigator is persistent a diagnosis can be made in over 90 percent of vertiginous patients.

1. Ear
   a. Acute otitis media
   b. Serous otitis media
   c. Chronic otitis media
   d. Perilymph fistula
      i. Trauma
      ii. Post-stapedectomy
      iii. Barotrauma (round window rupture)
   e. Labyrinthitis
      i. Serous
      ii. Bacterial
      iii. Viral
      iv. Toxic
   f. Meniere's disease
   g. Vestibular neuronitis
   h. Benign positional vertigo
      i. Acoustic neuroma or other cerebellar-pontine angle tumor

2. Central nervous system
   a. Stroke (cerebrovascular accident)
   b. Transient ischemic attacks
3. Neck
   a. Cervical arthritis
   b. Carotid artery stenosis
   c. Vertebral-basilar artery insufficiency
   d. Subclavian steal syndrome
   e. TMJ

4. Metabolic disorders
   a. Hyper- or hypoglycemia
   b. Hyper- or hypothyroidism
   c. Electrolyte imbalance
   d. Hypercalcemia
   e. Anemia
   f. Polycythemia
   g. Leukemia
   h. Allergy

5. Infections
   a. Influenza
   b. Herpes zoster
   c. Measles
   d. Mumps
   e. Other viral illnesses

6. Drugs
   a. Streptomycin
   b. Kanamycin
   c. Gentamicin
   d. Diazepam
   e. Sedatives
   f. Opiates
   g. Alcohol
   h. Neuroleptics
   i. Aspirin
   j. Nicotine
   k. Caffeine

7. Cardiac Problems
   1. Arrhythmia
   2. Hypertension
   3. Hypotension
   4. Poor cardiac output

**Treatment**
The treatment of vertigo often falls closer to the art than to the science of medicine. It sometimes seems that all the physician's energy has been used in obtaining the history, conducting the laboratory examination, and reaching a reasonable diagnosis, and there is none left for creative therapy.

Specific causes of vertigo are treated. Bacterial labyrinthitis is a severe
disease and must be treated with antibiotics, usually in the hospital. It is often considered a surgical emergency and cause for labyrinthectomy to prevent spread of infection to the central nervous system. Although some physicians have very elaborate therapeutic regimens, a simple approach is equally effective: Phenothiazines are the mainstay of treatment, and promethazine hydrochloride is as effective as any. For mild cases, 25 mg of promethazine can be taken orally every 6 hours. For some patients diazepam is useful alone or in combination with promethazine. For moderate intensity attacks, IV promethazine is indicated to stabilize the vertigo, after which oral or rectal suppositories can be used. Patients with severe cases frequently are dehydrated and need IV fluids. Promethazine is given IV frequently with diazepam. Alternatively, 0.5 mg to 1.5 mg IV droperidol is effective in those patients unresponsive to diazepam. Promethazine should not be given in conjunction with the droperidol therapy. Hospitalization is often necessary. Intractable labyrinthine vertigo can be treated surgically, with cure rates approaching 90% to 95%.

Many patients will request medication to combat motion sickness and a number of medications are useful. The first choice of drugs for airsickness or seasickness is usually a non-prescription medicine such as Dramamine® or Meclizine®. An effective prescription is Antivert®. These are effective and, although they cause some sleepiness, this tends to be mild. If the patient complains of motion sickness symptoms with very mild stimulation, such as flying in a modern jet or a long trip in a car, the cause may be psychologic. For these conditions, diazepam is effective, because it allays the patient's anxiety and it is also an effective vestibular sedative.

The most difficult cases are those people with sensitive vestibular systems who wish occasionally to go boating in ocean waters where they are exposed to intense vestibular stimulation. Oral promethazine is effective in these situations; 25 mg can be taken the evening before boating, and should be repeated approximately 1 to 1.5 hours before embarking. All of the phenothiazines have a long onset time; that is, they are not effective for at least 1 to 1.5 hours, and they also have a very long half-life. Therefore, the promethazine taken 12 hours earlier will still have some vestibular sedating effect when the patient embarks. Many patients do not like to take the evening dose of promethazine and simply begin with the first dose 1.5 hours before going aboard. Unfortunately, such a dose will put most people to sleep. If it is possible to board the boat and sleep for the first several hours and allow their vestibular systems to adjust to the rocking of the boat while they are asleep, many patients will require little or no additional medicine. If any is needed, the original dose can be repeated every 6 hours. If it is important that the person be alert and functional at the beginning of the trip, it will be necessary to give some stimulant to counteract the sedative effects of the promethazine, such as 25 mg promethazine with 25 mg of ephedrine, both to be taken orally at least 1.5 hours before boarding and not to be repeated more than once every 6 hours.
Another drug combination that has been popular with many sailors is 0.5 mg scopolamine with 2.5 to 10 mg of dextroamphetamine. This combination tends to be less sedating than promethazine and ephedrine. Another popular medication with many sailors is scopolamine supplied as a sticky patch to be placed on the skin behind the ear (Transderm-Scop). The scopolamine is absorbed slowly and is reputed to be effective for periods of 2 to 3 days. Its side effects - which some find irritating - include a dry mouth and pupillary dilatation. For some, the side effects are not tolerable. It is, at the time of this writing, the most popular prescription treatment for motion sickness. It is contraindicated in the geriatric population.

Many times "on board physicians" are asked to treat motion sickness once it has occurred. In such circumstances, the previous recommendations are not effective. Promethazine given intramuscularly or as a rectal suppository is effective. If this fails, IV fluids combined with promethazine, diazepam or droperidol can be required.

Individual head and neck surgeons organize their thoughts and their therapies regarding vertigo differently. Table 2.5 outlines an alternative differential diagnosis, evaluation, and treatment of vertigo.

Table 2.5
Differential Diagnosis, Evaluation, and Treatment of Vertigo

I. **Vestibular Neuronitis**
   A. Presenting signs and symptoms: acute onset of severe vertigo may be episodic and may be associated with preexistent upper respiratory infection, spontaneous nystagmus, and normal hearing.
   B. Etiology: probably viral neuronitis, with degeneration of Scarpa's ganglion and peripheral neurons.
   C. Laboratory tests: reduced vestibular response (RVR) in affected ear found on caloric testing; normal CNS examination.
   D. Treatment:
      2. Drugs:
         a. Meclizine, 12.5-25 mg/d po, divided into equal doses given q4h
         b. Dimenhydrinate, 25-50 mg pp or IM q4-6h
         c. Diazepam, 5-10 mg IM or IV q4-6h
         d. Promethazine, 25-50 mg IM or po q6-8h
         e. Prednisone 60-80 mg/d taper x3 wks
   E. Prognosis: patient usually improves over a 1-month period; however, there may be exacerbations for as long as 1 year. Canal paresis persists.
   F. Vestibular rehabilitation:
   II. **Acute Labyrinthitis (sudden hearing loss)**
      A. Presenting signs and symptoms: acute onset of severe vertigo associated hearing loss (mild to profound) and
spontaneous nystagmus.

B. Etiology: probably virally induced cochleolabyrinthitis (widespread, with damage to inner ear structures).

C. Laboratory tests:
   2. Electronystagmography: reduced vestibular response must be evaluated for CNS disease.
   3. Other tests:
      a. Mastoid tomograms.
      b. VDRL, FTE-ABS.
      c. T3, (triiodothyronine), T4 (thyroxine)
      d. Complete blood count, glucose tolerance test.
      e. Sedimentation rate, cholesterol, triglycerides, ANA qantinuclear antibody), and RF (rheumatoid factor).
      f. Western-blot for anti-68KD antibodies.

D. Treatment:
   2. Drugs: prednisone, 60-80 mg/d, tapering dose over 3 weeks (if not contraindicated).

E. Prognosis: usually dizziness subsides with time. If patient has U-shaped or upward-sloping audiogram, there is a good chance for recovery.

III. Meniere's Disease
   A. Presenting signs and symptoms:
      1. Attacks of episodic vertigo, pressure in ear, hearing fluctuation, roaring tinnitus.
      2. Nystagmus (only during acute attack).
      3. Low-frequency sensorineural hearing loss.
      4. Normal findings between episodes (early in the disease).

   B. Etiology: secondary to endolymphatic hydrops.
      1. Idiopathic
      2. Following temporal bone fracture.
      3. Following meningitis.
      4. Following sudden hearing loss (from mumps, etc.).
      5. Immune-mediated.

   C. Laboratory tests: document low-frequency hearing loss by audiometry. Same evaluation as for acute labyrinthitis (II-C).

   D. Treatment: same as for vestibular neuronitis (I-D). Reduce salt to $1500 \text{ mg/day}$ and caffeine intake, food additives. Give diuretics if symptoms do not respond to dietary changes alone. Surgery may be indicated if vertigo becomes incapacitating. (E. sac shunt, labyrinthectomy or vestibular nerve section)

   E. Prognosis: Variable. Symptoms may stop altogether or be episodic and eventually cause total sensorineural hearing loss with severe disabling vertigo. Disease is bilateral in 20-40%.

IV. Benign Positional Vertigo (BPV), Cupulolithiasis
   A. Presenting signs and symptoms:
1. Attacks of true vertigo occurring with the patient in supine position and typically with involved ear down.
2. Latency of 5-6 seconds before vertigo begins.
3. Nystagmus is generally rotatory toward the down ear.
4. Fatigues with repeated testing.
5. Normal hearing; may be without trauma.
6. Attacks last seconds to minutes.

B. Etiology: degenerative otoliths from utricular macula drift by gravity and become embedded in cupula of posterior canal crista or debris trapped within membranes labyrinth.

C. Laboratory tests: Positional testing in office. (Dix-Hallpike) Electronystagmography demonstrates positional rotatory nystagmus, delay in onset, fatiguing, fixation, or suppression. No CNS signs are present.

D. Treatment:
   1. Advise patient to repeatedly assume the positions causing vertigo; provide information and reassurance. Vestibular conditioning exercises will speed recovery in most patients.
   2. Semont maneuver, or Epley maneuver - Otolith repositioning techniques.
   3. Cawthorne exercises (vestibular conditioning exercises).
   4. Surgery: singular nerve section, vestibular nerve section, or posterior canal plugging procedure.

E. Prognosis: usually subsides with time, especially in young patients. If present longer than 6 months, consider surgery.

V. Acoustic Neurinoma (Schwannoma)

A. Presenting signs and symptoms:
   1. Unilateral, progressive, sensorineural hearing loss (typically high frequency).
   2. Tinnitus in affected ear.
   3. Mild disequilibrium, which may mimic Meniere's disease.
   4. Occasional pain or pressure in affected ear (not always present).

B. Etiology: Schwann cell or eighth nerve tumor (superior vestibular nerve most common origin); may be intracanalicular or extend into the cerebellar-pontine angle and compress the brain stem.

C. Laboratory tests:
   1. Brain stem evoked response audiometry (BERA) (see Fig. 1-9): delay in wave V must be compared with other ear; latency wave V greater than 0.02 msec is significant.
   2. Electronystagmography: reduced vestibular response.
   3. Audiometry: poor speech discrimination in 50-60%, tone decay, high-frequency sensorineural hearing loss, reflex decay.
   4. MRI scan with gadolinium of internal auditory canals and cerebella pontine angles is study of choice.
5. If MRI unavailable then high resolution CT scan with contrast.

D. Treatment
   1. Surgical removal.
   2. If patient is older than 70 years, or disease bilateral
      Gamma-knife radio surgery.

E. Prognosis: excellent if operated on early. However, there is
   a good chance of dead ear resulting from removal of tumor.
   Facial nerve paralysis may occur from removal of larger
   tumors. Untreated, they cause death by brain stem
   compression.

VI. **Neuro-otosyphilis (Congenital or Late)**
   A. Presenting signs and symptoms: fluctuating sensorineural
      hearing loss, episodic vertigo, tinnitus; may be bilateral.
      Other stigmata of syphilis may be present. Positive
      Hennebert's sign (pressure in the ear canal causes
      nystagmus).
   B. Etiology: endolymphatic hydrops, periostitis, obliterative
      endarteritis.
   C. Laboratory tests:
      1. VDRL is negative in 70% of patients.
      2. FTA-ABS: false positive in 6%, false-negative in 5%.
         May be positive in collagen-vascular disorders,
         autoimmune hemolytic anemias, cirrhosis, and
         occasionally pregnancy. Test must be repeated if +1.
   D. Treatment (Antibiotics plus steroids):
      1. Penicillin G (crystalline), 2-4 million U IV q4h for 10
         days OR
      2. Penicillin G (procaine), 60,000 U/day IM for 25 days,
         OR
      3. Penicillin G (benzathine), 2.4 million U/wk IM for 3
         weeks, OR
      4. Tetracycline hydrochloride, 500 mg p.o. q6h for 30
         days, OR
      5. Erythromycin, 500 mg po q6h, with probenecid, 0.5 g
         q6 for 30 days,
      6. Prednisone, 40-60 mg/day p.o. for 3 weeks and then
         5-10 mg/day for maintenance.
   E. Prognosis: often exacerbates, requiring boost in steroid
      therapy or retreatment.

VII. **Inner Ear Fistula (Round Window or Oval Window)**
   A. Presenting signs and symptoms:
      1. Sudden onset of mild, moderate, or severe hearing
         loss (may fluctuate) associated with vertigo or ataxia.
      2. Most often related to barotrauma, exertion, trauma, or
         surgery.
      3. Spontaneous nystagmus.
      4. Positional vertigo.
   B. Etiology: small leakage of perilymph out of inner ear via
      round window membrane or oval window.
   C. Laboratory tests: (all tests maybe negative)
1. Fistula test positive.
2. Electronystagmography: may be reduced vestibular response, positional nystagmus, positive fistula test.

**D.** Treatment: strict bed rest for 5 days. Surgical exploration and repair of fistula.

**E.** Prognosis: good for recovery from vertigo, poor for hearing improvement.

**VIII. Suppurative Labyrinthitis**

**A.** Presenting signs and symptoms:
1. Foul-smelling otorrhea.
2. History of chronic otitis media or cholesteatoma.
3. Severe vertigo or dizziness.
4. Fever.

**B.** Etiology: bacterial invasion of inner ear (commonly Pseudomonas).

**C.** Laboratory tests:
1. Gram strain.
2. Culture and sensitivity tests.
3. CT scan of temporal bones.
4. Lumbar puncture.
5. Audiometry.

**D.** Treatment:
1. Hospitalization.
2. IV antibiotics.
3. Mastoidectomy and possible labyrinthectomy.

**E.** Prognosis: if diagnosed early enough, the condition may be cured with medical or surgical therapy. Otherwise can lead to deaf ear, meningitis, or brain abscess.

*Table courtesy of Jeffrey P. Harris, M.D., Ph.D.*

2. Medical Treatment: - Vertigo

Phenothiazines are the most commonly used vestibular depressants. Most will sedate as they suppress the vertigo. Meclizine (AntivertR) 12.5-25 mg P.O. q. 6 hours prn Procloperazine (CompazineR) 5-10 mg P.O. q 6 hours prn 25 mg per rectum bid prn Diazepam is an excellent vestibular tranquilizer. Whether its effect is vestibular or CNS is debatable. It often provides superior results with less side effects than the phenothiazines. Diazepam 2.5-25 mg P.O. q 6 hrs prn.

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**Overview of Vertigo**

Vertigo is an incredibly complex and difficult illness. It cannot be evaluated in the usual 7-15 minute time slot, and in all fairness to the patient and the health provider, the patient should be scheduled or rescheduled for a long enough period to obtain a full history and perform a thorough examination.
To a cardiologist the most common cause of vertigo is arrhythmia; to an gerontologist the most common cause of vertigo is orthostatic hypotension; to a neurosurgeon all vertigo is brain tumor until proven otherwise; to an infectious disease expert vertigo is neurophilis until ruled out; an endocrinologist will report hypothyroidism as the most common cause of vertigo and to an otolaryngologist vertigo is a diagnostic challenge.

A good history, physical examination and laboratory assessment will point to the correct diagnosis.

Drugs, orthostatsis, arrhythmias, dementias, neurologic illness, TIA's, carotid artery stenosis and chemical imbalances of sodium, potassium, calcium or metabolic hormones, specifically thyroid, should be diagnosed.

The above aside, vertigo can be anything from a rather diffuse complaint of imbalance or unsteadiness to a very specific whirling vertigo in which the patient describes holding onto the rug to keep from falling off the earth. In severe cases, nausea and even vomiting can occur.

The three most common labyrinthine vestibulopathies are benign positional vertigo, viral vestibuloneuronitis and Meniere's disease.

Benign positional vertigo (BPV) is often, but not always, induced by head trauma. This can be minor or major. It classically presents as an acute onset dizziness brought on by tilting one's head back and turning to the side. One can do this on the exam table, a maneuver which is called the "hallpike maneuver". This will induce the dizziness. The dizziness lasts for several minutes and diminishes with time. If the maneuver is immediately repeated, a similar response is elicited, although diminished in intensity. BPV or BPPV, as it is also called, continues for weeks to months. Some dissipate over several weeks, many will take months and even longer to disappear.

Current recommendations are to diagnose the benign positional vertigo by electronystagmography (ENG) and then to perform a treatment described by Semont and separately by Eply. This maneuver works on the premise that there is floating debris in the posterior semi-circular canal. The maneuver consists of positioning the head in such a fashion that all the debris settles into the bottom of the canal. The head is then rotated in a series of maneuvers that trap the debris. The patient holds their head in this position for 24 hours. In 90 percent of patients, the vertigo is cured.

Vestibuloneuronitis is felt to be a post viral disease. It can occur concomitant with the viral infection or up to several weeks later as an autoimmune like illness. Otologists are increasingly recommending steroids in the treatment of vestibuloneuronitis. In the acute phase, current recommendations are 100 mgs of solumedrol IV push followed by 60mgs of oral prednisone daily for 1-week followed by a decreasing dose for the second week.

If a concomitant hearing loss is present, an audiogram should be obtained, steroids should be prescribed and if the hearing loss has not returned or if the
dizziness persists for longer than 6-weeks, an ENT referral is recommended.

The third diagnosis is Meniere's disease. We begin with the caveat that "Not all that spins is Meniere's. This is a diagnosis which has been used as a waste basket diagnosis and too frequently is over utilized. While Meniere's is probably a multiplicity of illnesses with a similar end result, the classic Meniere's is an acute onset illness with vertigo, decreased hearing and a feeling of aural fullness. Typically, the vertigo dissipates over several days, the hearing returns to normal, the illness resolves. Meniere's disease unfortunately is recurrent and episodes can occur frequently, regularly or irregularly.

There is an association between Meniere's disease and allergic rhinitis. There is an association between Meniere's disease and psychiatric illness with the usual discussions as to whether or not the psychiatric illness is causing the Meniere's disease or whether the Meniere's disease is causing the psychiatric illness. There are those that believe Meniere's is a fluid or salt imbalance.

Another name for Meniere's is labyrinthine hydrops. It is postulated that the efflux of endolymph is impaired. The endolymphatic fluid pressure increases with resultant symptoms.

Certainly, those with allergic rhinitis should be evaluated and treated. Those with psychiatric illness always benefit from psychiatric consultation. Endocrine and electrolyte imbalances must be diagnosed. Many otologists will recommend a trial of diuretics with potassium replacement. Vestibular sedatives diminish the symptoms.

Acoustic neuroma and other cerebellopontine angle tumors can present with symptoms mimicking the above illnesses. Diagnosis is made by MRI.