

Best Practices for the Evaluation and Management of Dizziness

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Biography

Stephen P. Cass, M.D., M.P.H., is Associate Professor in the Department of Otolaryngology at the University of Colorado Health Science Center. He is fellowship trained in Neurotology, specializing in disorders of the ear, hearing and balance. His research interest involves basic and clinical studies of the vestibular system. Dr. Cass is co-author with Dr. Joseph Furman of *Vestibular Disorders: A Case-Study Approach*.

We held a two-day course, "Best Practices for the Evaluation and Management of Dizziness: A Workshop with Leading Clinicians," in Chicago on June 25-26, 2004. The course was presented by the Department of Otolaryngology, University of Colorado School of Medicine and sponsored by the University of Colorado School of Medicine, Office of Continuing Medical Education. Educational support was provided by GN Otometrics, North America. I served as course director. This is the second course in this series. The first course was held in Chicago on October 11-12, 2002. A copy of these proceedings was published as an Insights in Practice article that can be accessed on www.bsurre4balance.com.

In the first course we heard from clinicians in the specialties of primary care, neurotology, neurology, audiology, physical therapy, and psychiatry. We learned that there is little controversy about management, but a great deal more controversy about evaluation. Dizzy patients usually see a primary care physician first. Most of them have benign disorders that can be successfully managed by the primary care physician, but a few have serious disorders that require referral to a specialist. Most of us agreed that the specialist who accepts dizzy patient referrals should be prepared to take a comprehensive history and perform a thorough physical examination, but we disagreed about which clinical observations are required. Sometimes the history and physical examination fail to yield a definite diagnosis and we need additional information provided by laboratory tests. We disagreed about which tests should be ordered for which patients.

In the second course, I wanted to get closer to a clear definition of best practices for the evaluation and management of the dizzy patient. I assembled a faculty of experts in the specialties of neurotology, neurology, and vestibular testing. I asked them to describe their methods, to present the evidence underlying their decision-making, and to indicate where the evidence is weak. I allowed plenty of time for discussion among faculty members and the audience. In this manner, I hoped to identify areas of consensus and to hear various viewpoints in areas of disagreement.

I began the course with a quick review of vestibular anatomy and physiology. I described the anatomy of the vestibular labyrinth and orientation of the labyrinth in the head, and explained the structure and function of the sensory receptors in the ampullae of the semicircular canals and the maculae of the utricle and saccule. I traced the neural pathways of the vestibular nerve, vestibular nuclei, and the vestibulo-ocular and vestibulo-spinal systems.

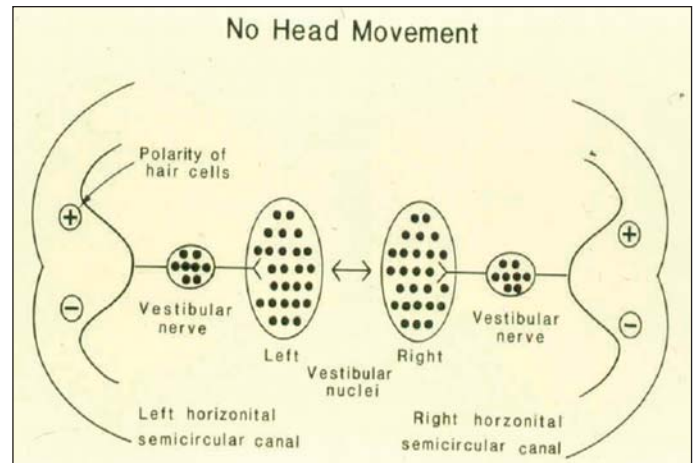


Fig. 1. Normal horizontal canal system at rest.

It is essential to understand the response of the vestibular system to injury.

Fig. 1 shows a normal horizontal semicircular canal system, viewed from above. When the head is at rest, tonic neural input comes from the two horizontal canals and the level of input from the two canals is exactly the same.

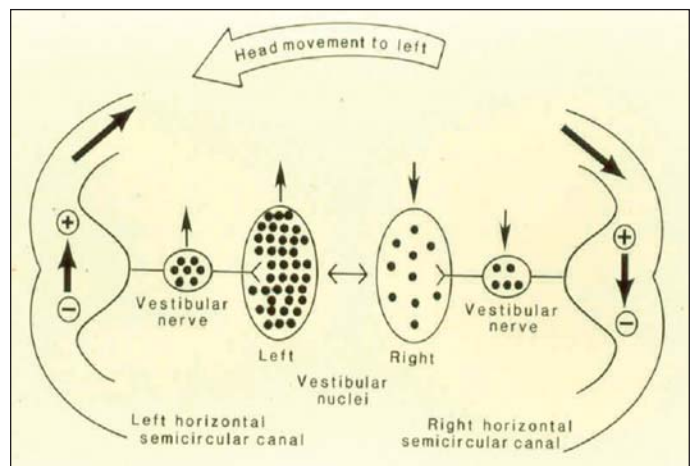


Fig. 2. Normal horizontal canal system during angular acceleration to the left.

Fig. 2 shows what happens when the head undergoes angular acceleration to the person's left (counterclockwise in our view). Endolymph movement lags behind head movement. In the left horizontal canal, this lag deflects the cupula upward (in our view), causing an increase in the level of neural input. In the right horizontal canal, this lag deflects the cupula downward (in our view),

continues

causing a decrease in the neural input. Thus the level of neural input from the left ear is greater than the level of neural input from the right ear. As a result, the person has a sensation of leftward rotation, left-beating nystagmus, and a tendency to fall to the right.

Fig. 3 shows what happens when a person suffers an acute lesion of the right horizontal canal. Neural input from the right canal is abolished, whereas tonic

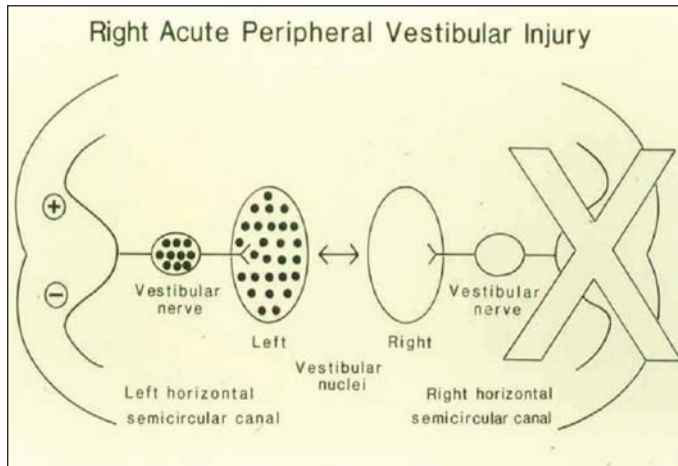


Fig. 3. Acute lesion of right horizontal canal.

neural input from the left horizontal canal remains. Thus the level of neural input from the left ear is greater than the level of neural input from the right ear. This asymmetry is the same as the one that occurs when a person rotates leftward and the reaction is also the same—a sensation of leftward rotation (which we now call “vertigo”), left-beating nystagmus (which we now call “spontaneous nystagmus”), and a tendency to fall to the right (which we can detect with postural tests). Acute lesions in other parts of the vestibular system cause similar (but not identical) signs and symptoms, and as we will see later, we can often localize the exact site of lesion by carefully noting the features of these signs and symptoms.

The lesion may be permanent, but nevertheless the patient’s signs and symptoms abate over a period of days and weeks due to vestibular compensation. A major part of the compensation process is a reduction of the asymmetry due to the gradual reappearance of tonic neural activity in the vestibular nuclei on the side of the damaged horizontal canal, as shown in **Fig. 4**. Compensation is never complete, however. Our physical exam and tests still can detect subtle

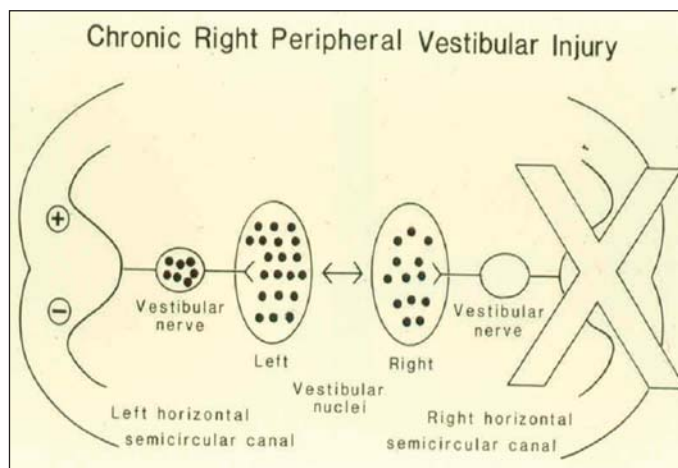


Fig. 4. Chronic lesion of right horizontal canal.

vestibular abnormalities for many years after the event. Some patients compensate better than others, and those who compensate poorly usually receive benefit from vestibular rehabilitation therapy.

Then I described how I take a history from the dizzy patient. It is often said that history taking is the most important part of the evaluation of the dizzy patient. I agree. Dizziness can be caused by an unusually large number of diseases and it is important to develop a working knowledge of all the medical conditions and disorders associated with dizziness. A good place to start with this task is the Compendium of Vestibular Disorders that can be accessed on www.bsurre4balance.com. The patient’s history offers an important opportunity to gain information needed to distinguish among them and to create a working list of potential diagnoses.

I take a comprehensive (Level V) history from every new dizzy patient—a daunting task. To make it easier, I use an electronic patient records system. In the waiting room, the patient fills out an eight-page questionnaire that asks questions in a forced-choice format. An optical character recognition system reads the patient’s responses and inserts them into a custom-designed template that yields a finished chart note. I take my laptop into the exam room and review my chart note with the patient, correcting and amplifying as needed.

A complete list of the items on my questionnaire can be accessed on www.bsurre4balance.com.

David Solomon, M.D., Ph.D., Assistant Professor of Neurology at the Johns Hopkins University School of Medicine, described how he conducts a physical examination of the dizzy patient.

Dr. Solomon performs a thorough examination of eye movements, as follows:

1. **Eye movement exam.** He examines the patient’s eye movements with eyes open in the light and with vision denied using Frenzel lenses, or video-oculography. He looks for spontaneous (horizontal) nystagmus, vertical nystagmus, torsional nystagmus, gaze-evoked nystagmus, and dissociated nystagmus.
2. **Head thrust test.** He asks the patient to look straight ahead and then jerks the patient’s head quickly rightward and leftward, looking for “catch up” saccades that denote a weak vestibulo-ocular response when the head is jerked toward the side of a labyrinthine loss.
3. **Head-shaking test.** He shakes the patient’s head rapidly back and forth 15 times and then looks for nystagmus while the patient’s head is held motionless. Head-shaking nystagmus usually (but not always) beats away from the side of a labyrinthine loss.
4. **Hyperventilation test.** Hyperventilation sometimes induces nystagmus in patients with a fistula or a compressive lesion (such as acoustic neuroma, cholesteatoma, or blood vessel) or an inflammatory lesion (such as multiple sclerosis) that causes demyelination in peripheral or central vestibular pathways.
5. **Valsalva maneuver.** The Valsalva maneuver sometimes induces nystagmus in patients with Arnold-Chiari malformation, perilymphatic fistula, or superior semicircular canal dehiscence.
6. **Ocular tilt reaction (OTR).** A unilateral otolithic lesion sometimes causes tonic ocular torsion and skew deviation when the head is tilted toward the side of the lesion.
7. **Dix-Hallpike maneuver.** He starts with the patient seated on the examination table with legs extended and the head turned 45 deg rightward. Then he brings the patient back rapidly to the supine position with the head still turned rightward and hanging backward over the end of the

examining table, as shown in **Fig. 5**. After performing the maneuver, he looks for a nystagmus response, noting its latency, direction, duration, and

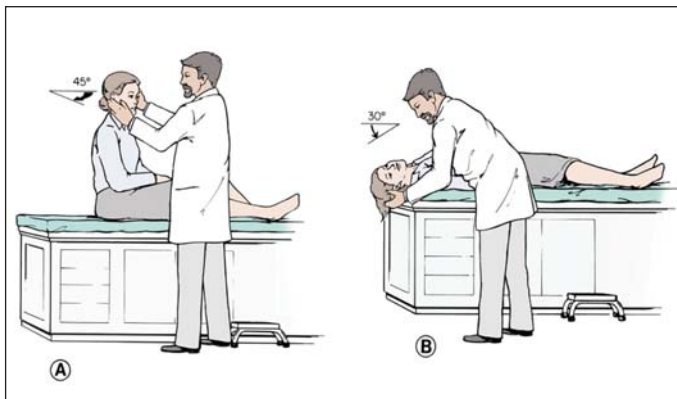


Fig. 5. Right Dix-Hallpike maneuver. Position of the patient at the start of the maneuver (A) and at the end of the maneuver (B). (Courtesy of Dr. Parnes).

presence or absence of accompanying vertigo. Then he returns the patient to the sitting position and repeats the maneuver with the patient's head turned leftward. This test detects posterior and anterior canal BPPV, which is denoted by a vertical-torsional nystagmus response that is delayed in onset, transient, and accompanied by vertigo. The direction of the nystagmus specifies the involved canal.

8. *Nylen-Barany positional test* (also called the "roll test"). With the patient in the supine position, he rapidly turns the head 90 deg rightward and then 90 deg leftward. This test detects nonlocalizing positional nystagmus (either geotropic or ageotropic) and horizontal canal BPPV.

Dr. Solomon also conducts *vestibulospinal tests*. While the patient is sitting, he looks for head tilt, past pointing, and axial postural asymmetry. With the patient standing, he first performs one or more static tests (Romberg, sharpened Romberg, stance on a foam cushion, and Valsalva maneuver) and then performs one or more dynamic tests (tandem gait, the Fukuda stepping test, and the circular walking test).

He also conducts a *general neurological examination*, which includes evaluation of cranial nerves, deep tendon reflexes, distal vibration sensation, and cerebellar function (finger to nose, rapid alternating hand movements, heel to shin). He looks for dysarthria, dysphagia, dysmetria, diplopia, Horner's syndrome, loss of pin prick or temperature sensation on one side of the face and/or the other side of the body, intractable hiccups, visual inversion, visual loss, oculopalatal tremor, and mental confusion.

In addition, he performs an *orthostatic blood pressure screening test*, a *dynamic visual acuity test*, and a *headache evaluation as indicated by the presenting history and symptoms*.

Kamran Barin, Ph.D., Assistant Professor, Dept. of Otolaryngology and Dept. of Speech and Hearing Science, The Ohio State University, discussed laboratory vestibular testing. He said that laboratory vestibular tests provide an independent assessment of the peripheral vestibular system, the vestibular nerve, and central vestibular pathways. They detect lesions, differentiate between peripheral and central lesions, and lateralize peripheral lesions or further localize central lesions. They also help with devising treatment plans, monitoring the progress of treatment, and planning for acoustic neuroma, vestibular ablation, and cochlear implantation surgeries.

Dr. Barin described five commonly used laboratory vestibular tests:

1. *ENG/VNG* is a battery of eye movement tests. For decades, we have performed ENG (electronystagmography), in which eye movements are monitored with electrodes placed on the skin around the eyes. In recent years, ENG has been largely supplanted by VNG (videonystagmography), in which eye movements are monitored by infrared video cameras mounted inside lightproof goggles.

The standard ENG/VNG test battery consists of

- a. four oculomotor tests (saccade test, tracking test, optokinetic test, and gaze test with fixation), which detect CNS lesions,
- b. two vestibular tests (static positional test and gaze test without fixation), which detect lesions of the peripheral or central vestibular system,
- c. two tests (Dix-Hallpike maneuver and pressure test), which identify specific etiologies,
- d. the caloric test, which detects and lateralizes lesions of the horizontal semicircular canal or its afferent pathways.

ENG/VNG has more clinical value than any other laboratory vestibular test. Dr. Barin recommends that it be used in the evaluation of all dizzy patients, except that it should be deferred pending treatment outcome in patients with BPPV. *ENG/VNG* detects one or more abnormalities in about 50 % of dizzy patients and about 75 % of these abnormalities specify the site of lesion. Other laboratory tests detect few of these abnormalities. A skilled clinician can detect most of them during physical examination, although physical examination does not permit quantitative analysis or yield a permanent record.

2. *Rotary chair testing* consists of recording horizontal eye movements as the patient is rotated about a vertical axis with the horizontal semicircular canals in the plane of rotation. The patient is usually tested under three conditions: (a) in complete darkness, (b) while viewing an earth-fixed visual surround, and (c) while viewing a head-fixed visual target. Under each of these conditions, the patient undergoes a series of sinusoidal oscillations at frequencies from about 0.01 Hz at octave intervals to about 1 Hz. Phase, gain, and symmetry of eye velocity re head velocity is computed for each test frequency.

Rotary chair testing has only fair clinical value. It is useful in documenting bilateral vestibular loss, although the ice water caloric test, the active head rotation test, and the head thrust test also detect this condition. Otherwise rotary chair abnormalities are nonlocalizing.

3. *Active head rotation* consists of asking the patient to shake his or her head in the horizontal and vertical planes at frequencies from about 0.5 Hz to about 6 Hz while viewing an earth fixed visual target. Head movement is monitored by a head-mounted velocity sensor and eye movement is monitored by electrodes. Phase, gain, and symmetry of eye velocity re head velocity are computed at each test frequency.

Active head rotation has about the same clinical usefulness as rotary chair testing. It costs less and tests both horizontal and vertical vestibulo-ocular responses at higher frequencies, although head and eye movements at high frequencies are difficult to measure accurately.

4. *Computerized dynamic posturography (CDP)* is comprised of two test batteries. The first is the Sensory Organization Test (SOT), in which the patient's postural stability is measured as visual and somatosensory cues are manipulated. The second is the Movement Coordination Test (MCT), in which the patient's postural stability is measured as the supporting surface is tilted or translated.

The diagnostic value of CDP is limited. Malingering patients tend to show an aphysiologic pattern of responses, so the test is sometimes used in medical-legal cases. Some physical therapists use it to design vestibular rehabilitation therapy and monitor its progress.

5. *Vestibular-evoked myogenic potentials (VEMP)* is a new vestibular test. The patient is presented with loud monaural clicks or tone bursts and short-latency EMG responses are recorded from surface electrodes placed over the ipsilateral sternocleidomastoid muscles. These responses are amplified, filtered, and averaged over at least 100 repetitions of the stimulus. Animal studies indicate that they arise from the saccule.

It has been shown that VEMP identifies the symptomatic ear in patients with Tullio phenomenon. Initial reports suggest that it also detects various other pathologies.

Dr. Barin says that he performs laboratory vestibular testing according to the protocol shown in **Fig. 6**. Each new dizzy patient first receives a Dix-Hallpike maneuver and if the test is positive, the patient receives canalith repositioning treatment. If treatment is successful and the patient has no other unexplained

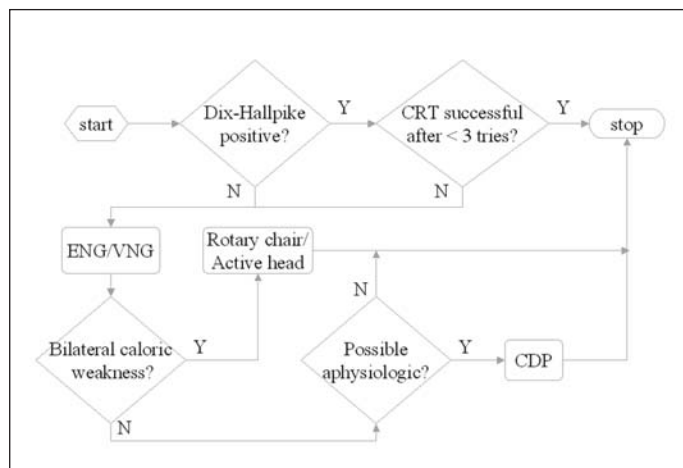


Fig. 6. Protocol for laboratory vestibular testing.

signs or symptoms, the patient receives no further laboratory vestibular testing. If the Dix-Hallpike maneuver is negative or treatment is unsuccessful, the patient receives ENG/VNG. If ENG/VNG shows a bilateral caloric weakness, the patient receives either a rotary chair test or active head movement test to confirm this abnormality. If aphysiologic behavior is suspected, the patient receives CDP to confirm this suspicion. Dr. Barin does not yet perform VEMP in his laboratory, but is following research in this area with interest.

Lorne S. Parnes, M.D., Professor of Otolaryngology and Clinical Neurology, Dept. of Otolaryngology, University of Western Ontario, discussed the diagnosis and treatment of five peripheral vestibular disorders—labyrinthitis/vestibular neuronitis, recurrent vestibulopathy, dehiscence superior semicircular canal syndrome, BPPV, and Meniere's disease.

Labyrinthitis/vestibular neuronitis is often accompanied or preceded by an upper respiratory infection. It is characterized by vertigo lasting for days, nystagmus beating away from the affected ear, and nausea and vomiting. Cochlear symptoms are present with labyrinthitis and absent with vestibular neuronitis; otherwise signs and symptoms are identical. Dr. Parnes treats this disorder symptomatically with antiemetics and vestibular sedatives. He treats with oral steroids if he sees the patient within 72 hours after onset of acute vertigo. There is no evidence that anti-virals are efficacious.

Recurrent vestibulopathy is characterized by Meniere's-like spells of vertigo. There are no cochlear or other localizing symptoms and no diagnostic tests that specify this disorder. A few patients with recurrent vestibulopathy go on to develop typical Meniere's disease. Treatment is symptomatic, and symptoms resolve spontaneously over 2-3 years in most patients.

Dehiscent superior semicircular canal syndrome (DSSCS) is characterized by vertigo and oscillopsia in response to loud sounds (the Tullio phenomenon) or maneuvers that change middle ear or intracranial pressure. Eye movements evoked by these stimuli align with the plane of the dehiscent superior canal. The patient may also have pulsatile tinnitus, sensitivity to body sounds, and hearing loss. Treatments are avoidance of symptom-inducing situations, middle fossa resurfacing of the affected canal, or middle fossa or transmastoid occlusion of the affected canal.

BPPV is usually caused by free-floating particles in the endolymph of the posterior semicircular canal (posterior canalolithiasis). Dr. Parnes performs the Dix-Hallpike maneuver to identify the affected canal and then administers canalith repositioning treatment, as shown in **Fig. 7**. His success rate after a single treatment is 80%. If the patient still has BPPV at the next visit, he repeats the treatment. His success rate after three treatments is 95%.

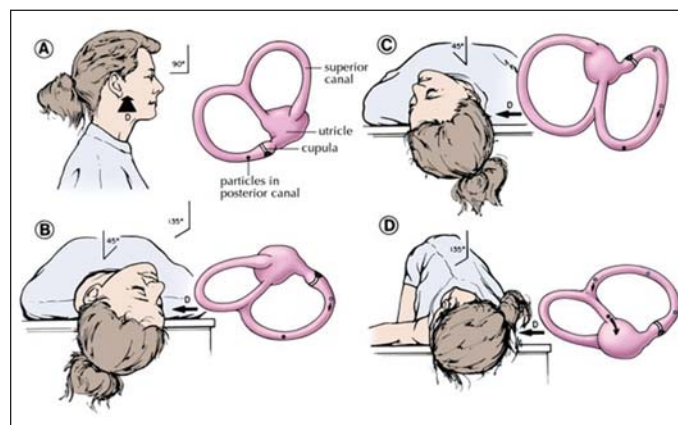


Fig. 7. Diagnosis and treatment of right posterior canalolithiasis. Position of patient and canaliths before right Dix-Hallpike maneuver (A), after right Dix-Hallpike maneuver (B), during canalith repositioning treatment (C), and after canalith repositioning treatment (D).

He treats intractable BPPV with posterior semicircular canal occlusion. He has performed this operation on 46 patients (in both ears of two patients). BPPV was completely relieved in every case. One patient had a hearing loss with vertigo three months after surgery. Six patients had protracted periods of imbalance after surgery and one patient developed horizontal canal BPPV. He has seen free-floating particles in 13 of 40 operated ears.

Dr. Parnes says he rarely sees cases of horizontal canal BPPV and has never seen a case of anterior canal BPPV.

Meniere's disease is characterized by multiple attacks of vertigo (each lasting more than 20 minutes), unilateral fluctuating sensorineural hearing loss, and ipsilateral tinnitus or aural fullness (or both). When one or more of these criteria is missing, the diagnosis is called “probable” or “possible” Meniere’s disease.

Dr. Parnes administers the following medical treatments for Meniere’s disease: low salt diet, avoidance of caffeine, nicotine, and stress, diuretics, benzodiazepines, antihistamines, histamine (betahistine), vasodilators, and corticosteroids.

If medical treatment fails, he treats with intratympanic gentamicin titration. He injects 1 ml of 40 mg/ml stock IV gentamicin solution through a myringotomy once a week. Treatments are discontinued if the audiogram shows a significant hearing drop for two successive weeks, if a new onset of persistent dizziness or imbalance occurs, if a new onset of spontaneous or head-shake nystagmus occurs, or when four treatments have been given. This treatment yields excellent control of vertigo and a low incidence of hearing loss (and no cases of severe hearing loss) and does not preclude further treatment if it fails.

David Solomon, M.D., Ph.D., discussed the diagnosis and management of common neurological vestibular disorders. He finds it useful to distinguish among three types of dizziness—presyncope, vertigo, and dysequilibrium without vertigo.

Presyncope implies insufficient central nervous system blood flow.

Common causes are hyperventilation, orthostatic hypotension, vasovagal attacks, decreased cardiac output (arrhythmia, myocardial infarction, congestive heart failure, aortic stenosis), anxiety or panic disorders, hypoglycemia, and drug toxicity (alcohol, barbiturates, benzodiazepines, anticonvulsants, cardiovascular drugs).

Vertigo implies either peripheral or central nervous system disease. A single attack of vertigo that lasts more than 24 hours may be due to labyrinthitis/vestibular neuronitis, posterior circulation infarction, cerebellar or brainstem hemorrhage, or multiple sclerosis. Recurrent attacks of vertigo that lasts for a few seconds may be due to uncompensated vestibular loss, crisis of Tumarkin, or drop attacks. Recurrent attacks that last for minutes may be due to TIAs. Recurrent attacks that last for hours may be due to Meniere’s disease or migraine. Recurrent attacks that last for days may be due to labyrinthitis/vestibular neuronitis, stroke, or multiple sclerosis. Positional vertigo is usually caused by BPPV (the attacks are severe and brief), but can also be caused by central disorders, such as posterior fossa tumors and infarction, Chiari malformation, cerebellar degeneration, and multiple sclerosis (the attacks are usually mild and persistent).

Disequilibrium without vertigo implies a bilateral vestibular loss (cisplatin or gentamicin), peripheral neuropathy (diabetes), a spinal cord dorsal column lesion (compressive, B12 deficiency, syphilis), cerebellar atrophy, white matter disease, normal pressure hydrocephalus, or an extrapyramidal disorder (Parkinson’s disease, progressive supranuclear palsy).

Central nervous system dysfunction is implied by physical findings of direction changing or purely vertical nystagmus, sustained or non-fatigable positional nystagmus, disconjugate nystagmus, abnormal posture when seated, inability to stand, focal motor deficit, dysarthria, dysphagia, diplopia, limb ataxia, Horner’s syndrome, loss of pin prick or temperature sensation on one side of the face and/or on the other side of the body, or intractable hiccups. ENG findings of defective saccades, pursuit, or gaze holding also imply central nervous system dysfunction. Spontaneous nystagmus with normal calorics suggests (but does not prove) central dysfunction.

Dr. Solomon then discussed specific diseases, as follows:

1. *Vertebrobasilar insufficiency* presents initially as an attack of vertigo in 19% of patients, and 62% of patients experience at least one isolated attack of vertigo at some time during the course of the disease. These attacks are usually accompanied by nausea and vomiting. Patients with vertebrobasilar insufficiency nearly always have other brainstem or visual complaints, such as visual loss, diplopia, drop attacks, unsteadiness, incoordination, extremity weakness, or confusion. When vertebrobasilar insufficiency is first suspected, the patient is treated with daily aspirin and attention to risk factors. If episodes persist, aspirin /dipyridamole or clopidogrel may be substituted. If significant stenosis is found or episodes are frequent and disabling, treatment is anticoagulation with heparin followed by warfarin, titrating to an international normalized ratio of 2-3.
2. *Lateral medullary syndrome* (or Wallenberg’s syndrome) is caused by occlusion of the posterior inferior cerebellar artery (PICA). This artery supplies the dorsal lateral medullary plate and portions of the posterior medial cerebellum. Occlusion of the PICA at its origin causes the full-blown syndrome—vertigo, spontaneous nystagmus, skew deviation, altered subjective visual vertical, ipsilateral limb ataxia, ipsilateral facial hemianesthesia, ipsilateral Horner’s syndrome, ipsilateral vocal cord paresis, ipsilateral gag, ipsilateral palatal weakness, gait ipsipulsion, saccade ipsipulsion, and contralateral body pain and temperature sensory loss. Occlusion of distal branches of PICA can produce a syndrome that mimics a labyrinthine disorder—vertigo, dysequilibrium, and spontaneous nystagmus.
3. *Pontine syndrome* is caused by occlusion of the anterior inferior cerebellar artery (AICA). This artery supplies the lateral pons and part of the middle cerebellar peduncle. It also gives off the labyrinthine artery, which provides exclusive blood supply to the inner ear, as shown in **Fig. 8**. Occlusion of the AICA causes vertigo, nystagmus, ipsilateral tinnitus, ipsilateral hearing

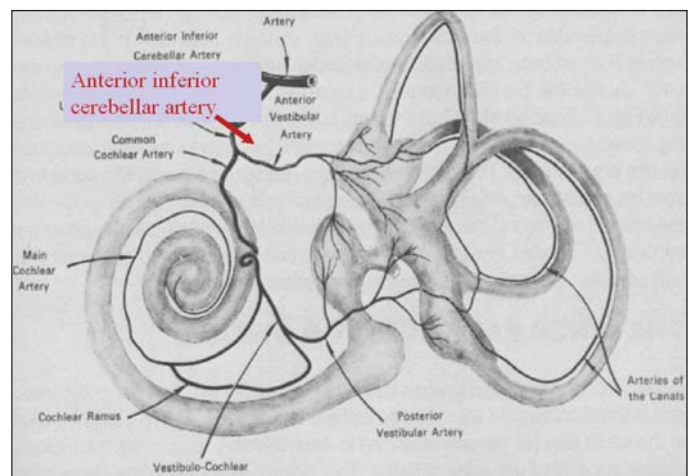


Fig. 8. Labyrinthine arterial supply.

loss, ipsilateral gait and limb ataxia, ipsilateral facial hemianesthesia, ipsilateral facial paralysis, ipsilateral Horner’s syndrome, and contralateral hemibody sensory loss.

4. *Cerebellar infarction* sometimes occurs without brainstem involvement. Since brainstem signs are absent, a mistaken diagnosis of labyrinthine pathology might be made. Key differentiating findings are normal responses on the head thrust test combined with gaze-evoked or vertical nystagmus and/or ataxia. A cerebellar infarction may affect only the inferior and medial cerebellum, causing nystagmus without ataxia, or it may affect only the cerebellar hemispheres, causing ataxia without nystagmus.

5. *Migraine* is present in about 11 million Americans, with 18% of females and 6% of males affected. The highest prevalence is at 30–45 years of age. It has been shown that migraine is undiagnosed in 41% of females and 29% of males who meet strict diagnostic criteria. Most cases of “sinus” headache are migraine. Some patients with migraine also have dizziness, either true vertigo or imbalance and motion sensitivity. Dizziness may occur before or during the headaches or it may occur independently. Dizziness is often accompanied by photophobia, phonophobia, or visual or other auras. Acute attacks usually last for minutes to hours, seldom longer than 24 hours. Migraine may be indistinguishable from Meniere’s disease, except that accompanying hearing loss is uncommon. Treatment is both behavioral and pharmacological. Behavioral treatment includes regular sleep patterns, stress reduction, migraine diet (avoiding chocolate, cheese, red wine), and eliminating caffeine and habitual analgesic use. Pharmacological treatment to abort attacks includes combinations of caffeine, aspirin, acetaminophen and butalbital or a non-steroidal anti-inflammatory (such as ibuprofen or naproxyn sodium). Prophylactic treatments include beta blockers (propranolol), tricyclic antidepressants (nortriptyline), calcium channel blockers, and valproic acid. Acetazolamide and other anticonvulsants have also been used.
6. *Sporadic adult-onset ataxia* can be caused by a variety of disorders, including vitamin deficiency, gluten sensitivity, thyroid disorder, paraneoplastic syndrome, and multiple system atrophy (Shy-Drager syndrome). The diagnostic evaluation includes testing for thyroid function, B12, magnesium and vitamin E levels, antigliadin and antiendomysium antibody, Hashimoto’s antibodies- thyroglobulin and thyroid peroxidase antibodies, antineuronal antibodies, trinucleotide repeats for autosomal dominant spinocerebellar ataxias, anti-GAD antibodies, and TATA-binding protein. In many cases, no cause is found and even if a cause is found, no effective treatment exists.
7. *Multiple sclerosis* typically begins between 20–40 years of age. It usually presents with optic neuritis, but presents with vertigo in 5% of patients. Vertigo is a symptom sometime during the course of the disease in about 50% of patients. Bilateral internuclear ophthalmoplegia is the hallmark of multiple sclerosis, but various types of central nystagmus may also be seen. An attack of multiple sclerosis may mimic a peripheral vestibular lesion with a unilateral caloric weakness. An IV pulse of high-dose steroids may shorten an attack. Acquired pendular nystagmus may respond to gabapentin. Vertical nystagmus may respond to gabapentin or baclofen.
8. *Arnold Chiari malformation (Type 1)* is characterized by unexplained sensorineural hearing loss, headache, vertigo, ataxia, dysequilibrium, dysphagia or other lower cranial nerve dysfunction. Gaze-evoked nystagmus, downbeat nystagmus, and defective pursuit are typical ocular motor findings. Treatment is suboccipital decompression of the foramen magnum.
9. *Neoplastic diseases* can cause dizziness. Infratentorial ependyomas arise from the lining of the fourth ventricle. Protracted nausea and vomiting are often present, and the classical headache is positional, with pain present while supine and relieved by sitting up. Brainstem gliomas may occur at any age, but are most common in children. Cerebellar signs, trigeminal and lower cranial nerve involvement occurs. In children, medulloblastoma may cause non-fatiguing paroxysmal positional nystagmus, which is usually purely vertical and accompanied by vertigo and generalized dysequilibrium. Vestibular schwannomas (or acoustic neuromas) account for 85–90% of all schwannomas. Presentation of vestibular schwannomas is usually insidious, with unilateral progressive hearing loss and vestibular loss (without vertigo). Tinnitus, headache, mastoid pain, facial weakness or otalgia

may be present. Paraneoplastic disease occurs when an immune response is triggered by a tumor that is usually remote from the nervous system. Anti-Yo antibodies cause a loss of Purkinje cells in the cerebellum, resulting in a syndrome of ataxia, dysarthria, and nystagmus. This may be the presenting picture, and when antibodies are detected, a search for the tumor must then begin.

10. *Wernicke’s encephalopathy* is caused by thiamine deficiency and can be brought on by poor nutrition, prolonged vomiting, alcoholism, eating disorders, or chemotherapy. Signs include vertical nystagmus, gaze-evoked nystagmus, and bilateral abducens palsies. Ataxia and mental changes are usually present as well. Signs may reverse within hours of thiamine administration.
11. *Normal pressure hydrocephalus* is characterized by dementia, incontinence, and gait disorder. MRI shows enlarged ventricles out of proportion to atrophy. Ventriculography is not helpful. Response to prolonged CSF drainage is the best predictor of improvement with a shunting procedure.
12. *Epileptic vertigo* is rare. It is characterized by episodes of vertigo lasting minutes, sometimes with associated ictal nystagmus, dysphagia, amnesia, disorientation, and visual field abnormalities.

Finally, I presented management of psychological and psychiatric aspects of dizziness. More than one-third of patients with vestibular dysfunction have anxiety symptoms. These symptoms cause decreased functioning, decreased quality of life, and prolonged recovery from vestibular disorders.

I disagree with the traditional criteria for psychogenic dizziness—vague description of symptoms, exacerbation of symptoms in certain environments, reproduction of symptoms by hyperventilation, and normal physical exam. While dizziness can be a symptom of a psychiatric disorder, dizziness without other psychiatric symptoms is insufficient for the diagnosis of psychiatric disease. Panic disorder may cause lightheadedness, but also causes palpitations and shortness of breath. Panic, general anxiety, acute stress, and post-traumatic stress disorders may cause an “unreal” feeling, but also cause anxiety, numbness, and flashbacks. Depression may cause a vague “swimming” feeling, but also causes poor appetite and insomnia. Conversion disorder may cause imbalance, but also causes tremors and other nonphysiologic behaviors.

Vestibular disorders commonly induce symptoms of anxiety or panic, especially in patients with vulnerable temperaments. Anxiety is part of the response to vestibular dysfunction, just as heart palpitations are part of the response to physical exercise. Concerns about future attacks of vertigo, possible embarrassment, serious medical illness, mental illness, and disability further increase the patient’s anxiety. There is a subset of patients with both panic disorder and vestibular dysfunction. These patients have vestibular symptoms between panic attacks, agoraphobia or height phobia, and discomfort in malls or supermarkets. Patients with chronic dizziness must pay more attention to maintaining their balance and have less time for attention to other tasks. They often complain of “brain fog” or a “spacey” feeling. Patients with chronic dizziness may also have symptoms of depression—trouble concentrating, poor sleep, fatigue, and social withdrawal.

Dismissive behavior by the clinician adversely affects the outcome of treatment. Such behavior includes: (a) failing to acknowledge that there is a problem, (b) minimizing the seriousness of the problem, (c) suggesting that the problem is “mental,” and (d) spending too little time with the patient. Dismissive behavior makes the patient anxious and angry. The opposite of dismissive behavior is validating behavior. Such behavior includes: (a) evaluating

both vestibular and psychiatric symptoms, (b) assessing temperament, (c) avoiding suggestion of psychogenicity, (d) explaining vestibular mechanisms, (e) explaining somatopsychic mechanisms, and (f) identifying sources of secondary anxiety and providing corrective information. Validating behavior usually means spending more time with the patient.

Useful treatments are vestibular rehabilitation therapy and medication (Clonazepam 0.5 mg PO BID is drug of choice). If evaluation reveals a true psychiatric disorder, the patient should be referred to a psychiatric professional who is knowledgeable about vestibular disorders. Such a referral should be made after counseling the patient and should not be made on the first visit.

After hearing these presentations, we broke up into small groups for practical demonstrations of diagnostic and treatment techniques. Dr. Solomon demonstrated the head thrust test; Dr. Parnes demonstrated the Dix-Hallpike and canalith repositioning maneuvers; Dr. Barin demonstrated the interpretation of ENG/VNG tracings; and Timothy C. Hain, M.D., Chicago Dizziness and Balance and Associate Professor of Neurology at Northwestern University, demonstrated the interpretation of video eye movement recordings.

Dr. Parnes wrapped up the course with a presentation of difficult cases.

Summary. Most dizzy patients have benign disorders that can be successfully managed by a family physician, but some have serious disorders that require evaluation and management by a specialist. As specialists who see dizzy patients, we must be able to distinguish among many possible diagnoses, including some outside our own areas of specialty. We must be prepared to take comprehensive histories and perform thorough physical examinations, as outlined in this course. Sometimes our evaluations yield definite diagnoses, but more often they yield lists of possible diagnoses, and to distinguish among them, we must seek information provided by laboratory testing. We rely primarily upon imaging studies and laboratory analyses of blood and other body fluids to help confirm or refute possible diagnoses. Audiometric and vestibular tests are also useful, especially for determining the functional state of the vestibular system, but rarely crucial for diagnosis. Our goal is to make the correct diagnosis and treat accordingly. However, in a subset of patients we need to treat without a definite diagnosis.

It has been a terrific privilege to organize these conferences and interact with our outstanding faculty. Our attendees have been highly motivated learners and active participants. No one left early; in fact, we had to be asked to leave by the hotel staff as they anxiously readied for an evening wedding. For our faculty, and me, the reward is in seeing and feeling the intense attention, interest, and urge to learn of our participants, all with the goal to better care for their patients with dizziness. I invite you to consider joining us for the 3rd such conference planned for June 2006.

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