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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.* Oxford University Press recently published the second edition.

Is there any consensus about the "best way" to evaluate and manage dizziness? Ask any three clinicians about how and why they do what they do and you are likely to get three different, often ardent opinions about the proper way to evaluate and manage dizzy patients. Yet, it is logical to presume that some clinical approaches may be more effective and efficient than others. Perhaps if we look carefully at the methods used by expert clinicians, areas of agreement will surface from the depths of our habits, idiosyncratic training experiences, and biases. Perhaps there are essential elements that should form the foundation of a "best practice" for evaluating and managing dizziness. Understanding these best practices should lead to more accurate and possibly less costly diagnosis and more effective management of dizziness.

To explore this issue, we held a two-day course, "Evaluation and Management of Dizziness," on October 11-12, 2002, in Chicago, Illinois. The course was presented by the University of Colorado Department of Otolaryngology and sponsored by the Office of Continuing Medical Education, University of Colorado School of Medicine. Educational support was provided by ICS Medical, a GN Otometrics company. I served as course director.

The purpose of the course was to examine "best practices" for the evaluation and management of the dizzy patient. I assembled a panel of expert clinicians—a family physician, two neurotologists, two neurologists, an audiologist, a physical therapist, a psychiatrist, and a medical practice manager. I asked each expert to set forth an explicit and systematic version of "best practice" and then to defend it against criticism from other members of the expert panel and the audience. In this manner, I hoped to identify areas of consensus and to hear various viewpoints in areas of controversy.

Philip D. Sloane, M.D., M.P.H., Elizabeth and Oscar Goodwin Distinguished Professor of Family Medicine at the University of North Carolina, provided a perspective on evaluation and management of the dizzy patient by a primary care physician.

Dr. Sloane reported that primary care physicians (FP/GP/IM) see the majority (70-80%) of dizzy patients and manage most of them without hospitalization or referral to a specialist. By comparison, otolaryngologists see about 6% and neurologists 4% of patients seeking care for dizziness. Dizziness is a common complaint, especially among the elderly, but it rarely denotes life-threatening or rapidly progressive disease. While dizziness can result in handicap and diminished quality of life, it is not a good predictor of mortality.

Dr. Sloane believes that there are a few sacred cows that deserve to die. These include: 1)the specialist knows best; 2) you should differentiate between "central" and "peripheral" vertigo; 3) a test battery is useful in working up dizziness; and 4) if you don't know the diagnosis, give Antivert.

Dr. Sloane considers history taking to be the most important part of the workup. He always asks about the character, onset, severity, and duration of the dizziness, precipitating and alleviating factors, associated symptoms, general health, cardiovascular risk factors, and medications. He finds the diagnostic terminology confusing and diagnostic criteria poorly established for all but a few disorders. The high number of possible diagnoses makes decision-making complex. Because of this, he believes that the diagnosis of dizziness is not well suited to empiric algorithms, but often requires diagnosis by intuitive hypothesis generation and testing.

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Dr. Sloane tries to narrow the broad differential diagnosis by distinguishing among four subtypes—presyncope, dysequilibrium, vertigo, and vague dizziness.

Presyncope implies a circulatory disorder. Dr. Sloane asks about anemia, medications, acute illness, cardiac history and risk factors, palpitations, and vasovagal precipitants.

Dysequilibrium implies a neuromuscular disorder. Dr. Sloane asks about vision, proprioception, history of vertigo, and weakness or deconditioning.

Vertigo implies a vestibular disorder. Dr. Sloane asks about previous episodes, duration of episodes, and provocation with position change. Episodic vertigo implies BPPV, Meniere's disease, or TIAs. Continuous vertigo implies a psychophysiologic disorder, stroke, cerebellar atrophy, or neurolabyrinthitis.

Vague dizziness implies a psychologic disorder, particularly if the patient is young and the symptoms are continuous, associated with somatic complaints, or worsened by social or visual stimulation. Dr. Sloane asks about depressive symptoms, anxiety symptoms, and associated symptoms or events. He treats with reassurance and stress reduction.

Dr. Sloane discussed three common specific diagnoses—BPPV, cervical dizziness, and postural dizziness.

BPPV is common and easily treatable. If Dr. Sloane elicits a history of positional vertigo, he performs a Dix-Hallpike maneuver. If the response is positive, he treats immediately with canalith repositioning. He recognizes that primary care physicians need more up-to-date training in BPPV to feel comfortable treating it with repositioning maneuvers.

Cervical dizziness is caused by two types of pathology—cerebral vascular (TIAs of the vertebral artery) and facet joint disease.

Postural dizziness is common, especially in the elderly. Dr. Sloane considers the traditional criterion for orthostatic hypotension to be too stringent. He treats postural dizziness (whether or not orthostatic hypotension has been proven) by first ruling out treatable cerebrovascular disease, blood or other volume loss, and adrenocortical insufficiency. Then he discontinues vasodilators and anticholinergics, hydrates well, and prescribes pressure gradient (Jobst) stockings. He may also medicate with beta blockers, transdermal scopalamine, or fluorocortisone.

Dr. Sloane also discussed management of the dizzy patient by a family physician and presented his six rules of dizziness management:

Rule 1: Make a diagnosis and treat it appropriately. Dr. Sloane reviewed the records of 144 dizzy patients seen by primary care physicians. He found that 34% of these patients had a lab test, 11% had a major test, and 9% were referred to a specialist. Treatment consisted of observation in 72% of patients, reassurance in 42%, medication in 61%, and behavior change in 15%. At follow up six months later, 24% of patients were given a new diagnosis and one patient (a very elderly man) had died.

Rule 2: Be on the lookout for treatable conditions. Dr. Sloane listed sinusitis/otitis media, anxiety/panic disorder, cervical dizziness, postural dizziness (without orthostatic hypotension), cardiac event, intracranial aneurysm/evolving stroke, and acoustic neuroma.

Rule 3: Tincture of time is often the best management strategy. Dr. Sloane reviewed the records of 108 patients seen by primary care physicians. He found that only 63% of them said dizziness had any impact on their daily lives and only 18% of patients said it had a major impact. Three months later only 28% of patients said dizziness had any impact and only 6% said it had a major impact.

Rule 4: Chronic dizziness in older persons is usually multifactorial.

Rule 5: Therapeutic exercise works for selected patients. Dr. Sloane said that vestibular rehabilitation works for patients with chronic dizziness with vestibular involvement. Hip, leg, and abdominal strengthening exercises work for patients with deconditioning, frailty, or fear of falling. Brandt-Daroff exercises work for patients with acute BPPV.

Rule 6: A thoughtful generalist is often better than a specialist. Dr. Sloane doesn't often send patients to a specialist (typical referral rate from PCP to specialist is 5%). When he does, his criteria for referral are (1) a suspicion of progressive or serious disease, (2) a need for specialized treatment, or (3) a difficult, demanding, frustrating and/or non-improving patient.

Steven D. Rauch, M.D., Associate Professor of Otolaryngology at Harvard University, discussed evaluation of the dizzy patient by a neurotologist.

Dr. Rauch also emphasizes history taking. He first tries to distinguish

among four types (or "flavors," as he calls them) of dizziness—near syncope, dysequilibrium, psychogenic dizziness, and true vertigo.

Near syncope (the feeling of impending loss of consciousness) implies inadequate cerebral perfusion. Common causes are carotid stenosis, valvular heart disease or arrhythmia, and postural hypotension. Dr. Rauch sends patients with near syncope to an internist or cardiologist.

Dysequilibrium (the feeling of imbalance or impending fall) can be divided into two subtypes. The first subtype is gait dysequilibrium, or a feeling of imbalance that occurs only while standing or walking. This implies a neuromuscular disorder, such as diabetic peripheral neuropathy or cerebellar dysfunction. Dr. Rauch sends these patients to a neurologist. The second subtype is global dysequilibrium, or imbalance that is always present (not just while standing or walking). These patients may have inner ear or neurologic disease. They receive a neurotologic evaluation.

Psychogenic dizziness (a "spacey" or "disconnected" feeling without sensation of motion or loss of balance) implies a psychiatric disorder. Dr. Rauch sends these patients to a psychiatrist, but they receive a neurotologic evaluation as well, since patients with a psychiatric disorder may also have inner ear disease.

True vertigo (the false sense of motion) implies an inner ear disorder. Dr. Rauch distinguishes four subtypes of true vertigo. The first is episodic vertigo accompanied by a hearing loss, which implies Meniere's disease. The second is episodic vertigo without a hearing loss, which implies BPPV. (Dr. Rauch said he makes the diagnosis of BPPV from the history alone; he does not perform a Dix-Hallpike maneuver.) The third is persistent vertigo accompanied by a hearing loss, which implies labyrinthitis. The fourth is persistent vertigo without a hearing loss, which implies vestibular neuritis. This simple classification system makes the correct diagnosis in 60% of patients with true vertigo.

Dr. Rauch discussed three disorders that don't fit into his classification system—dysequilibrium of aging, migraine-associated dizziness, and cervical vertigo.

The typical patient with *dysequilibrium of aging* is an elderly individual (usually brought in by adult children) with progressive gait instability and advanced multi-system neural deficit.

The typical patient with *migraine-associated dizziness* is a female who usually has a family history of migraine and a past history of childhood or adolescent migraine, chronic or progressive motion intolerance, or anxiety/panic disorder.

The typical patient with *cervical vertigo* has "floating" dysequilibrium with brief episodes of true vertigo provoked by head-on-body movement. There is usually a past history of whiplash, cervical disc disease, degenerative arthritis, or neck injury.

Timothy C. Hain, M.D., Associate Professor of Neurology at Northwestern University, was scheduled to discuss evaluation of the dizzy patient by a neurologist. Unfortunately Dr. Hain was unable to appear due to sudden illness, so I delivered his presentation from the materials he had prepared.

Dr. Hain spends about 45 minutes with each new dizzy patient. He obtains a complete history that comes largely from an eight-page questionnaire filled out by the patient in the waiting room. The questionnaire covers present illness, associations (such as position), otologic history, social context (such as disability issues), full review of systems (especially vascular), family history (especially migraine), medications, and testing to date. Dr. Hain spends about 15 minutes reviewing the questionnaire with the patient.

Dr. Hain does a complete neurologic and neurotologic physical examination, first with the patient standing, then with the patient sitting.

With the patient standing, he does the Romberg test (regular and tandem, eyes open and closed) to detect otologic and neurologic dysfunction and malingering. Then he asks the patient to stand on heels and toes and to perform deep knee bends to detect ataxia due to weakness and neurologic dysfunction. Then he checks standing (and later supine) blood pressure and pulse to detect orthostatic hypotension and positional orthostatic tachycardia.

With the patient sitting, he does an otoscopic examination and tests cranial nerves, upper extremity power, deep tendon reflexes, Babinski signs, vibration at ankle, and cerebellar function to detect gross ear abnormality (such as a tympanic membrane perforation), neurologic dysfunction, and sensory loss. Then he looks for spontaneous, head shaking-induced, and vibration-induced nystagmus with the patient wearing Frenzel's goggles. Then he does the Dix-Hallpike maneuver to detect BPPV.

If the patient has a history of pressure sensitivity or ear trauma, Dr. Hain may perform a fistula test or Valsalva. If the patient has presyn-

cope, he may perform a carotid sinus massage. If the patient has dysequilibrium, he may perform the dynamic illegible 'E' test to detect bilateral vestibular loss.

Dr. Hain divides dizziness diagnoses into six categories, as follows:

Positional vertigo diagnoses include BPPV, central positional vertigo, phobic postural vertigo, low CSF pressure syndrome, orthostatic hypotension, and phobic postural vertigo.

Dizziness and headache diagnoses include migraine-associated vertigo (which afflicts 3.5% of U.S. population and 10% of women of childbearing age), Chiari malformation, and cervical vertigo.

Meniere's disease and related conditions, such as post-traumatic hydrops and autoimmune hydrops.

Unilateral and bilateral vestibular loss diagnoses include vestibular neuritis, labyrinthitis, acoustic neuroma, and gentamicin ototoxicity.

Pressure sensitivity diagnoses include fistula, superior canal dehiscence, and vestibular fibrosis.

Psychiatric disorder diagnoses include anxiety, somatization, post-traumatic stress, and malingering.

Dr. Hain also classifies dizziness diagnoses by duration of the dizziness episodes.

Episodes lasting 1 to 5 seconds (which he calls "quick spins") imply BPPV, superior oblique myokymia, seizure disorder, or Meniere's disease. Multiple bouts of quick spins (which he calls "vestibular paroxysma") imply failed vestibular nerve section or perhaps microvascular compression or viral neuritis.

Episodes lasting seconds to minutes imply BPPV, arrythmia, orthostatic hypotension, or Meniere's disease or its variants.

Episodes lasting minutes to hours imply TIAs, Meniere's disease or its variants, migraine-associated dizziness, or orthostatic hypotension.

Episodes lasting days or longer imply migraine-associated dizziness, stroke, brain tumor, sensory ataxias, vestibular neuritis or labyrinthitis, functional syndromes, or drug reactions.

If history and physical examination fail to yield a diagnosis, Dr. Hain may order laboratory tests, as follows:

Audiological tests, such as a pure tone audiogram (very useful), tympanometry (useful), acoustic reflexes (rarely useful), ECOG (occasionally useful), BAER (rarely useful), and OAE (rarely useful).

Vestibular tests, such as a caloric test (very useful), rotary chair (useful), posturography (occasionally useful), VAT (not useful if rotary chair is available), and VEMP (not sure).

Radiology, such as MRI of brain with gadolinium (often useful), CT of brain (rarely useful), CT of sinus (rarely, if ever, useful), x-ray, CT, or MR of neck (sometimes useful), and CT of temporal bone (rarely useful).

Blood tests, such as CBC, fasting glucose, SMA12, FTA, and TSH (rarely useful), B12 (often useful), anti – heat-shock protein (rarely, if ever, useful), other autoimmune tests, such as ANA or sed rate (rarely useful), and paraneoplastic tests (rarely useful).

Cardiac tests, such as a tilt table test (rarely useful), EKG (rarely useful), and event monitoring (occasionally useful).

Neuropsychiatric tests, such as an MMPI (occasionally useful), malingering battery (often useful), and cognitive tests (rarely, if ever, useful).

Dr. Hain says he fails to diagnose about 15% of dizzy patients.

Neil T. Shepard, Ph.D., Professor of Otolaryngology at the University of Pennsylvania, discussed the role of audiovestibular tests in the evaluation of the dizzy patient.

Dr. Shepard said that some audiovestibular tests detect lesions and others measure functional impairment. These tests rarely make a diagnosis. They provide information that is useful in making certain diagnoses and in monitoring the effects of treatment.

As the director of a Balance Center, Dr. Shepard receives referrals for general "audiovestibular testing," and he decides which particular tests are indicated. The tests that he performs and their indications are as follows:

An *audiometric evaluation* provides information required for making many diagnoses of peripheral vestibular disorders. It is indicated for virtually every dizzy patient.

ABR is useful for detecting brain stem or cerebellopontine angle lesions if MRI is unavailable.

ECOG detects abnormal cochlear mechanics. It is indicated when the diagnosis of Meniere's disease is being considered.

ENG (or VNG) is a battery of eye movement tests that detects and localizes a variety of vestibular and non-vestibular lesions in the peripheral and central nervous system. Most useful is the caloric test, which detects lesions of the horizontal semicircular canal and its afferent pathways. Dix-Hallpike maneuver makes the diagnosis of BPPV. (The torsional component of the BPPV response can be observed using Frenzel's goggles or recorded on video.) Either ENG or VNG is indicated for virtually every dizzy patient.

Clinical Test of Sensory Interaction of Balance (CTSIB), also known as the "foam and dome" test, detects a variety of balance disorders. It is indicated for virtually every dizzy patient.

Rotational chair detects vestibular dysfunction. It is indicated when ENG is normal, when there is a compensated unilateral or bilateral caloric weakness, or when a baseline is needed prior to serial monitoring.

Posturography detects a variety of balance disorders. It is indicated when CTSIB is abnormal, when the patient complains of unsteadiness without vertigo, or when there is known or suspected pathology involving postural control pathways.

Postural evoked responses are indicated when the patient complains of unsteadiness during standing or walking or when response latencies to backward sway on posturography are abnormal.

Vestibular evoked myogenic potentials (VEMP) detect and lateralize lesions of the saccule. This test is indicated when the patient complains of linear vection, when caloric asymmetry is greater than 30%, when there is a bilateral caloric weakness. It is also performed prior to transtympanic gentamicin treatment or surgical removal of CPA mass lesions.

Subjective visual vertical and ocular counter roll detect utricular dysfunction. One or the other of these tests is performed prior to transtympanic gentamicin treatment. The subjective visual vertical test is insensitive to chronic utricular lesions.

Lorne S. Parnes, M.D., Professor of Otolaryngology and Clinical Neurology at the University of Western Ontario, discussed management of the dizzy patient by a neurotologist.

Dr. Parnes sees about 15 new dizzy patients per week in his clinic in London, Ontario. The average waiting period for an initial office visit is six months. All new dizzy patients (except those with the complaint of positional vertigo) receive an audiogram and ENG before he sees them. Dr. Parnes does not have access to rotational chair testing or posturography in his center.

He treats vestibular neuronitis and labyrinthitis symptomatically with antiemetics and vestibular sedatives. Since there is good evidence that oral steroids are effective, he treats with oral steroids if he sees the patient within 72 hours after onset of acute vertigo. (He said there is no evidence that anti-virals are efficacious.)

He provides surgical treatment, if indicated, for BPPV, Meniere's disease, chronic otitis media, cholesteatoma, acoustic neuroma and other temporal bone tumors, perilymph fistula, otosclerosis, vascular compression syndrome, and superior semicircular canal dehiscence.

Dr. Parnes described his treatment for BPPV in detail, as follows:

> Most cases of BPPV arise from free-floating particles in the endolymph of the posterior semicircular canal. Dr. Parnes performs the Dix-Hallpike maneuver to identify the affected canal, then he treats with canalith repositioning. 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%.
>
> Dr. Parnes treats intractable BPPV with posterior semicircular canal occlusion. He has performed this operation on 42 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 11 of 36 operated ears.

Dr. Parnes also described his treatment for Meniere's disease in detail, as follows:

> His treatments for Meniere's disease include low salt diet, avoidance of caffeine, nicotine, and stress, diuretics, benzodiazepines, antihistamines, histamine (betahistine), vasodilators, and corticosteroids.

If these treatments fail, Dr. Parnes 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., Assistant Professor of Neurology at the University of Pennsylvania, discussed management of the dizzy patient by a neurologist.

Dr. Solomon said that he sees dizzy patients with a variety of diseases, neurologic and non-neurologic, so the first step is to determine the cause of dizziness through history, physical examination, and laboratory testing.

The complaint of *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, and cardiovascular drugs). Non-specific dizziness is also part of postconcussion syndrome.

The complaint of *vertigo* implies either peripheral or central nervous system disease. Peripheral disorders, though debilitating, are generally benign, whereas some central disorders require urgent intervention or intensive care unit monitoring. Acute labyrinthine lesions typically present with intense vertigo, nausea, vomiting, and direction fixed horizontal nystagmus that is reduced or eliminated by visual fixation with gradual improvement over a period of three days. Any deviation from this pattern suggests a central lesion. Recurrent attacks of vertigo may be due to Meniere's disease, migraine, or vertebrobasilar transient ischemia (brainstem or labyrinthine). A single attack of vertigo that lasts more than 24 hours may be due to posterior circulation infarction, cerebellar or brainstem hemorrhage, or multiple sclerosis. Positional vertigo due to BPPV is characterized by short latency, duration less than one minute, fatigability, and nystagmus in the opposite direction upon returning to the upright position. Central disorders, such as posterior fossa tumors and infarction, Chiari malformation, cerebellar degeneration, and multiple sclerosis, can also cause positional vertigo, but the vertigo is usually persistent and mild.

The complaint of *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 examination 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 face and/or on the other side of the body, or intractable hiccups.

Central nervous system dysfunction is implied by ENG findings of defective saccades, pursuit, or gaze holding. Spontaneous nystagmus with normal calorics suggests (but does not prove) central dysfunction.

The patient's history may indicate additional testing—cardiac event monitoring and ECG, fingerstick glucose monitoring when symptomatic, ambulatory EEG, tilt table testing, and EMG/nerve conduction studies. A woman with progressive cerebellar symptoms must receive a mammogram and gynecological examination.

An emergent image must be obtained when a patient has acute vertigo and one or more of the following—other brainstem or cerebellar symptoms, direction changing spontaneous nystagmus, acute onset of vertigo accompanied by neck pain, new onset of severe headache, inability to stand or walk, asymmetric or unilateral hearing loss, or stroke risk factors (diabetes, hypertension, history of myocardial infarction).

Cerebellar diseases include infarction, hemorrhage, tumor, Chiari malformation, multiple sclerosis, hereditary and acquired cerebellar degenerations, paraneoplastic syndrome, medications and toxins (alcohol, anticonvulsants, lithium, organic solvents), vitamin E deficiency, and acute cerebellar encephalitis.

Signs of cerebellar disease are gaze-evoked nystagmus, rebound nystagmus, downbeat nystagmus, alternating skew deviation, saccadic dysmetria,

saccadic oscillations (flutter and macro square-wave jerks), positional vertigo (not fitting BPPV), oscillopsia, gait ataxia, positive Romberg with eyes open, defective pursuit, tremor, dysarthria, and limb ataxia.

Hereditary cerebellar ataxia syndromes of various types have been identified and at least 8 different genetic loci have been implicated. Unfortunately, only supportive care is available. Some patients with inherited ataxia have metabolic errors that can be identified and treated.

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 with suboccipital decompression of the foramen magnum.

Migraine is present in about 11 million Americans, with 18% of females and 6% of males affected. Peak age is 30-45 years. In patients meeting strict diagnostic criteria, less than half were given the diagnosis of migraine by a physician. Acute attacks usually last minutes to hours, seldom more than 24 hours. They usually occur immediately before or during the headache, but may occur without headache. 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.

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.

Vertebrobasilar disease is a common cause of dizziness. The vertebrobasilar system supplies not only the brainstem and cerebellum, but also the inner ear.

Vertebrobasilar insufficiency presents initially as attacks of vertigo in 25% of patients and most patients will experience vertigo during an attack at some time. These attacks usually have sudden onset and last for several minutes.Vertigo is nearly always accompanied by other brainstem or visual complaints (visual loss, diplopia, inversion of the environment, drop attacks, limb ataxia, mental status change, dysarthria or focal sensory or motor dysfunction). 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 wayfarin, titrating to an international normalized ratio of 2-3.

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 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 syndome—vertigo, dysequilibrium, and spontaneous nystagmus—that mimics a labyrinthine disorder.

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, as well as giving off the internal auditory artery, which provides exclusive blood supply to the inner ear. Occlusion of the AICA causes vertigo, nystagmus, ipsilateral tinnitus, ipsilateral hearing loss, ipsilateral gait and limb ataxia, ipsilateral facial hemianesthesia, ipsilateral facial paralysis, ipsilateral Horner's syndrome, and contralateral hemibody sensory loss.

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 gaze-evoked or vertical nystagmus and ipsilateral extremity and gait ataxia. A cerebel-

lar 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.

Microvascular compression of the 8th nerve is a controversial diagnosis. Typical symptoms are episodes of vertigo induced by a particular head position, but there are no signs or symptoms that specifically define the disorder.

Vascular malformations occur in 3-7% of the population. They may cause symptoms, including vertigo, due to mass effect or hemorrhage.

Neoplastic diseases include:

Infratentorial ependylmomas 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, medulloblatoma may cause non-fatiguing paroxysmal positional nystagmus, which is usually purely vertical and accompanied by vertigo and generalized dysequilibrium.

Vestibular schwannoma (or acoustic neuroma) 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 be initiated.

Wernicke's encephalopathy is caused by thiamine deficiency. Signs include vertical nystagmus, gaze-evoked nystagmus, and bilateral abducens palsies. Ataxia and mental changes are usually present also. Signs may reverse within hours of thiamine administration.

Normal pressure hydrocephalus is characterized by dementia, incontinence and a gait disorder.

Epileptic vertigo is very uncommon. It is characterized by episodes of vertigo lasting minutes, sometimes with associated ictal nystagmus, dysphagia, amnesia, disorientation, and visual field abnormalities.

Susan L. Whitney, Ph.D., PT, NCS, ATC, Associate Professor of Physical Therapy at the University of Pittsburgh, discussed management of the dizzy patient by a physical therapist.

Dr. Whitney said the goals of vestibular physical therapy are to optimize function, decrease dizziness, improve balance and the ability to walk, decrease fear and anxiety, prevent falls, increase gait speed, decrease stiffness, and improve the patient's ability to perform daily activities.

These goals are accomplished by increasing VOR gain, gait retraining, strengthening muscles, increasing range of motion, improving proprioception, habituation, patient education, and prescribing assistive devices.

Patients are screened for physical therapy with the Dizziness Handicap Inventory, the Activities-specific Balance Confidence Scale, and the sit-to-stand test. These self-report instruments are useful to assess the severity of symptoms and to monitor outcomes.

Vestibular physical therapy should be performed by physical and occupational therapists with special interest and knowledge in vestibular physical therapy. Many of these therapists are members of the Vestibular Special Interest Group in the Neurology Section of the American Physical Therapy Association.

Candidates for vestibular physical therapy are patients with peripheral vestibular disorders, bilateral vestibular disorders, combined peripheral and central vestibular disorders, central vestibular disorders, surgical removal of acoustic neuroma or labyrinthectomy, multisensory dysequilibrium, cerebellar disorders, BPPV, labyrinthine concussion, cervical vertigo, Meniere's disease, migraine, multiple sclerosis, mal de debarquement syndrome, panic disorder, vertebrobasilar insufficiency, fear of movement, or falling.

Patients who have had vestibular physical therapy have more confidence, more awareness of their limitations and abilities, less dizziness, more energy, better balance, ability to walk faster and safer, less fear, fewer falls, and more strength. They feel that they have more control over their lives, that they are not crazy, and that there are others "just like them."

Rolf Jacob, M.D., Professor of Psychiatry and Otolaryngology at the University of Pittsburgh, discussed management of the dizzy patient by a psychiatrist.

Dr. Jacob disagrees 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. He said that while dizziness can be a symptom of panic or anxiety disorder, dizziness without other psychiatric symptoms is insufficient for the diagnosis of psychiatric disease. Vestibular symptoms can exacerbate panic or anxiety disorders. Vestibular symptoms can also cause anxiety, decreased ability to concentrate, depression, social withdrawal, and anger in patients without psychiatric disease.

Dr. Jacob explained that certain anxiety symptoms of psychiatric interest occur in all patients, whereas a subset of symptoms appear to be specific for the patient with unremitting severe dizziness or imbalance, and the neurophysiological correlates of the vestibular dysfunction-anxiety link have only recently been understood.

Anxiety symptoms—both somatic and cognitive—are perhaps the most important psychiatric complication of vestibular dysfunction. There is a high prevalence of anxiety symptoms in vestibular patients. For example, in Dr. Jacob's setting, 1/3 of consecutive (i.e., unselected) patients with vestibular dysfunction had anxiety symptoms qualifying as panic attacks on a questionnaire assessment. In patients with anxiety disorders, vestibular abnormalities have been correlated with the presence of symptoms of agoraphobia, space and motion discomfort and dizziness between panic attacks.

The sources for primary anxiety (somatic symptoms, autonomic symptoms) in vestibular patients include the "hardwired" direct linkage and the immediate somatopsychic response to the somatic and autonomic symptoms. Thus, anxiety is a component of the symptomatic response to vestibular dysfunction just as heart palpitations are part of an integrated response to physical exercise. The sources of secondary anxieties (e.g., "What if….?") are more diverse and include concerns over future attacks of vertigo, dizziness or panic, possible social consequences (e.g., embarrassment), medical illness (e.g., "I have a brain tumor."), mental illness (e.g., "I feel like I am going crazy."), and disability.

Dr. Jacob explained that there is a subset of patients with both panic disorder and vestibular dysfunction. These patients may have symptoms of vestibular dysfunction occurring between panic attacks and are more likely to manifest panic disorder with agoraphobic avoidance or with height phobia. In these patients, as well as patients with solely uncompensated vestibular dysfunction, certain situations (e.g. malls, supermarkets, moving visual scenes) result in increased symptoms. Dr. Jacob refers to this situational pattern of symptoms as space and motion discomfort.

Patients with Unremitting Symptoms. Riding a roller coaster is an experience many individuals choose to have. The situation for the patient with unremitting symptoms, however, can be compared to a roller coaster ride infinite in duration. Besides anxiety and space and motion discomfort, these patients develop psychiatric problems that are related to the disability due to dizziness. Dr. Jacob uses the syndrome of mal de debarquement as a prototype for patients with continuous symptoms. Dr. Jacob told us that among such patients responding to a survey on the Internet, the most common psychiatric symptoms were fatigue, problems concentrating, anxiety, memory problems, and depression. Dr. Jacob explained that patients with chronic dizziness have to deploy attention to maintain balance similar to what is required from normal subjects under demanding balance conditions. This results in difficulty maintaining attention on tasks that require planning, difficulty remembering things, a subjective sense of one's mind seeming foggy ("brain fog"), and feeling spacy. In addition, persistent attentional demands can lead to *fatigue,* a common complaint among these patients.

Depression. Trouble concentrating, poor sleep, and fatigue can also be symptoms of another psychiatric consequence of persistent symptoms—depression. This disorder is also characterized by dysphoric mood, loss of interest in things previously enjoyed, poor appetite, and death wishes that can culminate in suicidal ideation. One source of depression is the very realistic practical restrictions on lifestyle experienced by the patients, restrictions that deprive them from the rewards of everyday life, including employment, household chores and recreational activities.

Like secondary anxiety, depression is in part related to the patient's thinking behavior. The depressed individual engages in ruminative thoughts. Depressive thoughts can be identified by their characteristic "if only" verbal structure, e.g., "If only the doctor could find out why I have these symptoms". Pervading the state of mind of a depressed person is a sense of hopelessness, bitterness, and demoralization. Alternatively, the depressed state can be conceptualized as sadness over the loss of previous function and the change this implies for future possibilities, and the necessary deviation from the patient's anticipated "life story".

Social Withdrawal. Dr. Jacob reported that studies on patients with balance disorders often reveal social anxiety and social phobia to be among the most prevalent complications. For patients with active symptoms of imbalance, social fears provide reasons for avoiding public places in addition to space and motion discomfort already discussed. Besides social anxiety, social withdrawal is the end result of a number of influences. For example, in the case that the patient is unable to maintain employment, the resulting change in social network naturally results in restrictions in social activities.

Anger and Clinician Dismissive Behaviors. In the case of acute illness, the patient's illness behavior is usually complemented by the clinician's treatment behavior, such that the latter leads to a reduction in the former. Treatment behaviors include the clinician showing interest in the problem, performing appropriate assessments, in due course providing physical explanation for the patient's symptoms, and proceeding with appropriate treatment (an approximate index of treatment behavior is the amount of time spent with the patient). When the clinician's behavior does not conform to these norms, the patient will increase his or her illness behavior. This tends to manifest as increased or more persistent symptom report and displays of anger. The clinician behaviors that tend to have this effect can best be described as "clinician's dismissive behaviors."

Dr. Jacob told us that outrage over clinician's dismissive behaviors can be discerned in close to 50% (9/19) of the autobiographical reports published on the Web site for mal de debarquement. One form of dismissive behavior occurs when the patient has been evaluated and no traditional "treatable" cause identified. Associated with the "good news" that no invasive treatments are needed comes the implication that the patient's vestibular problem is minor. Another form is failure to even recognize a problem. Insult is added to injury when suggestions are made that the problem might be "mental."

The opposite of dismissive treatment behaviors is validating treatment behaviors. Such behaviors include: (a) recognizing the patient's symptoms even if they do not fit a predetermined pattern; (b) educating the patients about the manifestations of vestibular dysfunction beyond vertigo, such as those discussed in this course; (c) recognizing that unknown etiology does not imply psychiatric etiology; (d) recognizing that psychiatric symptoms can occur secondarily to vestibular dysfunction. All of this will require increase in the amount of time spent with the patient beyond the usual tight clinical time schedule.

Karen Zupko, a practice management consultant and President of KarenZupko & Associates, Inc., discussed the art and science of getting paid.

Ms. Zupko said that certain audiological tests, known as the "balance testing package," are commonly performed during the evaluation of the dizzy patient (ICD-9 780.4).

> Most insurance carriers cover four tests—comprehensive audiogram (92557), tympanogram (92567), acoustic reflex testing (92568 or 92569), and auditory brainstem response (92585). Documentation requirements include a diagnostic audiological examination report (by a physician, not an audiologist), a physician's order, a consultation report related to hearing problems, a diagnosis, patient history and physical information, and any additional diagnostic studies that support and justify the need for diagnostic audiology tests. These are bilateral CPT codes, which means that if only one ear is tested, the modifier –52 (for reduced services) must be submitted.
>
> ENG (92541-92545) is also covered by most insurance carriers. Caloric irrigation (92543) should be reported four times or with a "4" in the units box.
>
> ECOG (92584) may also be covered.
>
> Officials at HCFA and Medicare are undecided about whether to require Medicare intermediaries to pay for posturography tests (92548). As a consequence, intermediaries in at least 35% of the 50 U.S. states are refusing to pay and private insurance companies have followed suit. The same is true for the sinusoidal rotational chair test (92546).
>
> Patients with balance disorders and dizziness may be referred to physical and occupational therapy. Many of these patients may have developed secondary symptoms, such as decreased strength, loss of range of motion, muscle fatigue, and headaches.
>
> Canalith repositioning can be billed under the unlisted procedure code (92599).

Summary and Conclusions

More than 100 health care professionals—mostly otolaryngologists, neurologists, audiologists, and physical therapists—attended the course. At the end, they made many positive comments and gave us the highest ratings I have ever seen for a CME course. I wish to thank the speakers for their enthusiasm and hard work and ICS Medical for its generous support.

What did we learn from the course? I think we learned that there isn't much controversy about management, but lots of controversy about evaluation. Most dizzy patients have benign disorders that can be successfully managed by the family physician with the "tincture of time," but a few dizzy patients have serious disorders, and distinguishing between benign and serious disorders is sometimes difficult. There appear to be two key decision points in the evaluation process: (1) the decision of the family physician to refer to a specialist, and (2) the decision of the specialist to order laboratory testing.

It is a fact of life that most dizzy patients see a primary care physician first. Dr. Sloane, the family physician on our panel, told us that he would send a dizzy patient to a specialist if (1) he thought the patient needed specialized treatment, (2) he suspected that the patient had serious disease, or (3) the patient made a fuss. These criteria generated lots of discussion. There was no consensus about which clinical observations the primary care physician should be expected to make and which findings should trigger a referral.

Most of us agreed that the specialist who accepts dizzy patient referrals should be prepared to take a comprehensive history and perform a targeted but thorough physical examination, as described by Dr. Hain and Dr. Solomon. The bedside evaluation is essential to determine the physiologic status of both the sensory and motor aspects of the vestibular system. This sometimes yields a definite diagnosis, but more often it yields a list of possible diagnoses, and to distinguish among them, we may need additional information provided by laboratory tests, or an empiric trial of therapy. In the end, a significant number of patients will remain undiagnosed but do need to be treated.

We agreed that it would not be useful to order every test for every patient, but there was no clear consensus about which tests should be ordered for which patients. Basic audiometry studies were deemed valuable and cost-effective, and MRI imaging studies important when CNS abnormalities were suspected based on the history and physical exam. Certainly, the value of vestibular testing was greater for the specialist than the primary care physician, and it was rare for the diagnosis to depend exclusively on the results of vestibular laboratory testing.

What's next? We plan to pursue these issues in a second course in Chicago, June 25-26, 2004. The course will be titled, "Best Practices for the Evaluation and Management of Dizziness: A Workshop with Leading Clinicians." I hope you will join us.

Visit www.bsure4balance.com for information on the June 2004 course.

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