



PRACTITIONER'S GUIDE TO THE DIZZY PATIENT

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ABOUT THE PRACTITIONER'S GUIDE TO THE DIZZY PATIENT

The information in this guide has been reviewed for accuracy by specialists in Audiology, Otolaryngology, Neurology, Physical Therapy and Emergency Medicine

ABOUT THE AUTHOR

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HOW TO MAKE AN APPOINTMENT WITH THE WAKE FOREST BAPTIST HEALTH BALANCE DISORDERS TEAM

Physician referrals can be made through the STAR line at **336-713-STAR (7827)**.

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HOW TO USE THE *PRACTITIONER'S GUIDE TO THE DIZZY PATIENT*

This guide is intended to be used as quick access to information when seeing a patient complaining of dizziness. There are many different sensations, with many different causes, that a patient may describe as “feeling dizzy.” The most common causes of dizziness are covered in this guide, but there are many causes of dizziness that are not covered here. The goal of this guide is to increase efficiency and accuracy, while minimizing time spent gathering pertinent case history information and disseminating patient educational information.

This guide begins with the **typical complaints** of various disorders causing dizziness, imbalance, and/or vertigo on a short, easy access list. This list is also displayed on the inside back cover of this guide. This can be kept by the practitioner's side during the case history interview. As the patient relays their symptoms, the practitioner can flip to the appropriate page to assist in gathering additional relevant information.

For each disorder there is a **Practitioner Review** page and a **Patient Education** page. The information on the Practitioner Review page lists typical complaints associated with each disorder.* Since many patients do not provide a clear description of their symptoms, there are **defining questions** to extract more detailed information. **Medical history questions** may provide a connection between the patient's current complaints and a previously diagnosed medical condition. A short **diagnosis/definition** of the disorder is listed on each practitioner review page, followed by some **management options**.

The **Patient Education** page is designed as a patient handout. These are brief, informative, and written in non-medical terminology.

There are several options included that may be used as intake forms. One option is to copy the inside back cover and have the patient circle the block that best describes their symptoms. There is a two-page traditional **dizziness history questionnaire**, and an eight-question **short form questionnaire** with an **interpretation guide**. The **short form questionnaire** also appears in the accompanying Balance Disorders Program brochure. There is a **fall risk questionnaire** with an interpretation guide to help explore the multiple factors that can lead to increased fall risk. These should be given to the patient to fill out prior to their appointment.

*A patient education page for Ménière's disease is not included, due to the detailed and controversial nature of this condition. A specific patient education handout is recommended in the text of the practitioner review.

TYPICAL COMPLAINTS OF VARIOUS VESTIBULAR AND NON-VESTIBULAR DISORDERS

Benign Paroxysmal Positional Vertigo (BPPV)

PAGES 18–19

"I woke up and the bed was spinning."

"I bent over (rolled over, turned quickly, laid down, sat up from bed, etc.) and everything started spinning around."

"Every time I tried to get up I fell back on to the bed."

Vestibular Neuritis/Labyrinthitis

PAGES 20–21

"I had constant spinning and nausea for about three days."

"After the worst of it (vertigo and nausea) I was okay if I didn't move. If I moved I was off balance and would get nauseous if I moved too much."

Ménière's Disease

PAGES 22–23

"I have had several episodes of severe vertigo with nausea, lasting for hours at a time."

"I feel so much pressure in my head (ear)."

"My ear was roaring."

"After an episode, I need to sleep for several hours."

Vestibular Migraine

PAGES 24–25

"I have episodes of spinning and nausea that come on without warning, but had no ear symptoms."

"I felt a sudden wave come over me, like the floor shifted."

"I can't stand any type of motion. It never bothered me when I was a kid."

Superior Canal Dehiscence

PAGES 26–27

"I get dizzy around loud noise or when I blow my nose."

"My voice sounds like I am in a barrel."

"I hear my pulse or chewing louder than I should."

Anxiety/Hyperventilation

PAGES 28–29

"I felt like my heart was pounding out of my chest."

"I felt like I was dying."

"I couldn't breathe."

Orthostatic Hypotension

PAGES 30–31

"I get dizzy and off balance when I stand up."

"I get up and start to walk and feel like I am going to fall over."

"When I get up quickly, I feel like I could faint."

Multifactorial Dysequilibrium

PAGES 32–33

"I have had several falls."

"I feel very unsteady when I am in a crowd or unfamiliar setting."

"I want to hold on to someone/something when I walk."

Central Dizziness

PAGES 34–35

"I stagger when I walk."

"If I lose my balance, I just go over."

"I have this constant feeling in my head."

STRUCTURE AND FUNCTION OF THE VESTIBULAR SYSTEM: A BRIEF OVERVIEW

The primary role of the balance system is to allow us to interact and maintain contact with our surroundings in a safe and efficient manner. As we move through our environment, information is gathered through our visual, somatosensory and vestibular senses and sent to our brainstem for integration, then finally on to our cortex for perception and processing. Our visual and somatosensory reference information is constantly changing as we move, but our vestibular reference—gravity—is unchanging. As long as the information coming in from these sources is predictable and non-conflicting, our equilibrium is stable and we move about freely with little thought regarding balance. When a sensory conflict occurs, the brainstem must efficiently and quickly (reflexively) adjust the level of priority given to the conflicting information, or a sensation of imbalance may occur. Since the known constant in the mix is gravity, the brainstem tends to rely more on vestibular information for maintenance of balance. When the vestibular system is compromised, the patient may become more dependent of visual and tactile feedback for maintenance of balance and orientation.

The peripheral vestibular apparatus (labyrinth) consists of matched pairs of sensors that are stimulated by any type of head movement, with specific sensors responsible for specific movements. These sensors are known as the cupulas of the semi-circular canals and the otolith structures. The arrangement of the semi-circular canals at right angles to each other causes the inner ear fluid (endolymph) to flow towards or away from the cupula with any head movements. As long as the response registered in each of the matched canals on each side is of the head is the same, balance is maintained. The otolith structures are responsible for sensing translational movement in which the head is steady in relation to the body, but the body as a whole moves. The

sacculle registers primarily vertical movements such as the sensation experienced when moving in an elevator. The utricle senses horizontal movements such as moving forward in a car.

The vestibular-ocular reflex (VOR) can be defined as reflexive eye movement in response to head movement. The role of the VOR is to allow stable gaze or clear, focused vision while the head is moving. It performs this function by causing eye movements that are equal to and opposite of head movement—in effect, visually canceling out head movement. A simple demonstration of the VOR can be done while reading this page. Simply hold this page 18 inches (or so) in front of you, and move your head back and forth at the maximum speed that still allows for clarity and easy reading. Then, with your head stationary, move the page back and forth in front of you at the speed nearly equal to the speed at which you moved your head. A noticeable degradation in visual acuity occurs. It is impossible to voluntarily move the eyes at speeds that are needed to maintain visual acuity during typical head movements. The latency of the response for the VOR is five times faster than the fastest voluntary eye movement, and ten times faster than the typical eye movement.

The VOR is compromised with damage to one or both peripheral vestibular apparatus. In the acute stage, patients complain of vertigo, often accompanied by nausea. Patients with chronic VOR deficit do not typically complain of vertigo, but rather complain of motion provoked dysequilibrium or disorientation, as head movement results in a blurring of their visual field.

Adapted from: Dizziness and Balance Disorders: A Short Course for Primary Care Physicians (Desmond, 2001), available from Micromedical Technologies.

CATEGORIZING THE DIZZY PATIENT

There are three main patient groups that present with complaints of dizziness.

The most common is the group of patients

who report some version of, “I’m not dizzy now, but I get dizzy at times.” Those patients with episodic symptoms require questioning about associated symptoms, timing and triggers. For example, “How long does it last? Is it provoked by movement or position change? Are there any changes in hearing noticed?” See page 6 for a timing and triggers checklist.

A second group presents with some version of, “I’m dizzy right now.” Many of these patients show up in the emergency room if they are acutely dizzy, ataxic (inability to walk) or vertiginous. These patients require a physical examination, including inspection for nystagmus and screening exams to investigate for symptoms and clinical signs that may be the result of a stroke. If stroke is suspected, neuro-imaging is indicated, and the patient should be followed closely. Many patients can be more accurately diagnosed by examination than imaging in the first 48 hours. On page 7, see the checklist for screening the patient with acute vertigo, and page 8 for a review of the HINTS protocol.

The third group says, “I am not dizzy. I am unsteady, off balance, afraid of falling.” These patients require a different line of questioning regarding risk factors for falling. The Desmond Fall Risk Questionnaire on page 10 is designed to help primary care physicians quickly determine if a patient may be at risk for falling, and to decide which specialist to consult (if any) for further

evaluation. The questionnaire may be filled out by the patient as part of their pre-appointment paperwork, and may be reviewed by nursing staff before the patient is seen. Each question is intended to obtain information regarding various known risk factors for falling, and which risk factors may be present in a particular patient.

The questions are worded so that a “Yes” response indicates the need for further questioning or examination. The questions are blocked according to each known risk factor: Previous history of falling or fear of falling (questions 1 and 2), vestibular dysfunction (3, 4 and 5), proprioceptive (somatosensory) loss (5 and 6), visual deficit (7), vascular disease (8), medication effect (9 and 10), cerebellar disorder (11 and 12), sedentary lifestyle (13), anxiety or psychological effect (14) and motivation for rehabilitation (15).



The questionnaire has two parts: 1) the questionnaire itself, which is intended to be copied and given to the patient, and 2) the Physician’s Guide. The Physician’s Guide offers possible explanations for positive responses to each question, as well as some suggestions for management.

TYPICAL TIMING AND TRIGGERS FOR COMMON DISORDERS CAUSING VERTIGO AND DIZZINESS

Pathology	Timing	Triggers
BPPV Transient vertigo	Less than one minute	Change in head position relative to gravity (e.g. tilting, lying down)
Orthostatic hypotension	Less than one minute	Rising from sitting or supine
Ménière's disease (vertigo, unilateral tinnitus)	Hours	Salt, caffeine, tobacco, stress, alcohol
Vestibular migraine (vertigo, motion intolerance)	Hours	Chocolate, cheese, red wine, hormonal changes, stress, bright/flickering light, atmospheric pressure change, disruption of sleep pattern, caffeine withdrawal
Vestibular neuritis/ Labyrinthitis (prolonged vertigo)	Hours to days with gradual decrease	Prior viral symptoms, idiopathic
Cerebellar stroke (Ataxia, vertigo)	Sudden onset persistent symptoms	Stroke risk factors
Anxiety/Hyperventilation (lightheaded, parasthesias)	Varies	Situational

INITIAL EXAMINATION CHECKLIST FOR ACUTE VERTIGO:
PERIPHERAL VERSUS CENTRAL

Name _____ Date _____

Equivocal	Peripheral	Central
No nystagmus	Direction fixed nystagmus Nystagmus decrease w/fixation Ambulates unassisted Positive head thrust exam Headshake nystagmus Transient positional nystagmus (positive Dix-Hallpike)	Direction changing nystagmus No decrease w/fixation Ataxia—unable to walk unassisted Ocular misalignment—vertical skew deviation Focal neurologic deficit (hemiplegia, dysarthria, limb ataxia) New onset severe headache
	 Refer for vestibular exam	 Refer for neuro-imaging Refer for neurologic consult

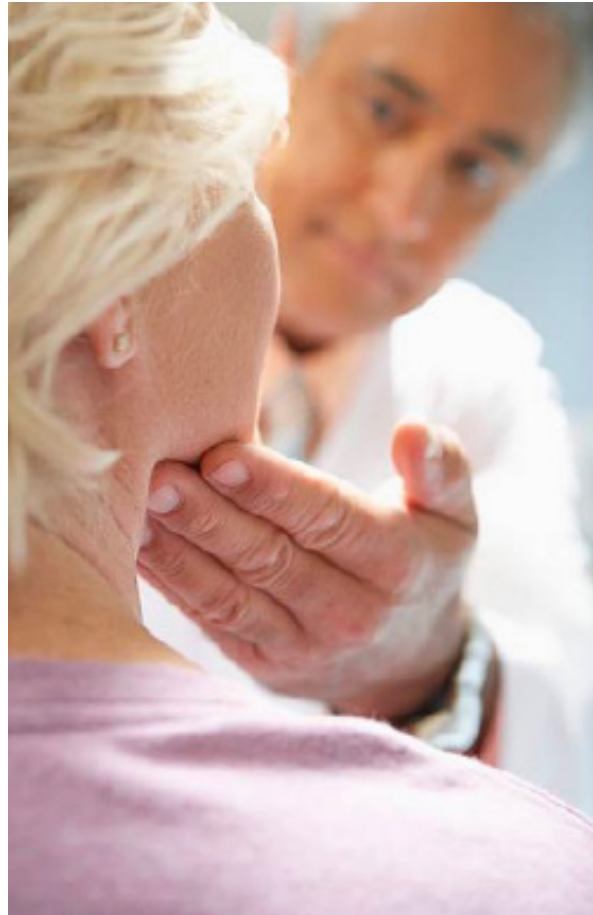
DIAGNOSING ACUTE VERTIGO

Imaging

Patients presenting with dizziness and vertigo are often referred for Computerized Tomographic (CT) scan of the brain. CT scans are frequently normal in the first few hours following acute ischemic stroke, therefore, a normal CT scan cannot rule out cerebellar/brainstem stroke (CBS). As many as 50 to 74 percent of CBS patients may be missed if the diagnosis is dependent on CT scanning (Simmons et al, 1986; Chalela et al., 2007). CT studies are particularly poor for ruling out brainstem stroke as that area is often poorly visualized due to surrounding bone structures. The American College of Radiology recommends “MRI of the head without and with contrast” as the appropriate test for the complaint of vertigo with no hearing loss (ACR, 1996). Although MRI has significantly higher sensitivity than CT, the examiner must not rely totally on MRI findings to identify or rule out CBS. On initial presentation, 12 percent of CBS patients had normal MRI exams, with abnormal exams a few days later (Kattah et al. 2009). Similarly, Chalela et al. (2007) report that 17 percent of patients diagnosed with acute stroke had normal MRI exams on initial presentation, commenting that there is a higher likelihood of a false-negative MRI exam when the stroke is located in the brainstem. So the old adage, “Let’s get an MRI just to be sure,” isn’t such a sure bet.

Is There a Better Way?

Kattah et al. (2009) describe a bedside eye movement exam thought to be very sensitive in differentiating acute vertigo patients with CBS from those with peripheral vestibular disorders.



The brief exam includes a combination of head impulse (head thrust) testing as described below, a review of nystagmus pattern, and examination for ocular misalignment (vertical skew deviation)

using the cross cover test. The cross cover test involves having the patient look at an object in the distance, then alternately covering each eye. If there is a consistent eye movement to regain fixation on the object, then ocular misalignment is suspected.

This combination of eye exams, described as **HINTS** (Head Impulse – Nystagmus – Test of Skew) is reported to be more sensitive than MRI in early identification of CBS.

Head thrust testing is almost always positive in patients with acute vertigo of labyrinthine origin, and almost always (approximately 90 percent) negative in patients suffering vertigo related to CBS. Direction changing horizontal nystagmus is sometimes (approximately 20 percent) present with CBS, but nystagmus is almost always direction fixed in acute labyrinthine disorders. Vertical skew deviation (ocular misalignment) is present in some (25 percent) of CBS patients, but very rare (4 percent) in labyrinthine patients. When a patient presents with the combination of: 1) normal head thrust exam, 2) direction changing horizontal nystagmus, and 3) positive skew deviation, there is a high probability (100 percent in the recent Kattah et al study) of brain or brainstem abnormality. Conversely, when this

combination of exams is considered benign (e.g., positive head thrust, no nystagmus or direction fixed nystagmus, and negative test for skew deviation) there is a very small chance (4 percent) of central involvement. This exam has significantly better sensitivity (100 versus 72 percent), and comparable specificity (96 versus 100 percent) when compared to immediate MRI. Because this exam can be done in one or two minutes and requires minimal equipment, there is great promise in more cost effective techniques to evaluate acute vertigo.

American College of Radiology. (1996). ACR Appropriateness Criteria.

Chalela et al (2007) Magnetic resonance imaging and computed tomography in emergency assessment of patients with suspected acute stroke: a prospective comparison. *Lancet*, Jan 27;36

Kattah, et al (2009). HINTS to diagnose stroke in the acute vestibular syndrome: three-step bedside oculomotor examination more sensitive than early MRI diffusion-weighted imaging. *Stroke*, 3504-3510.

Simmons, et al. (1986). Cerebellar infarction: comparison of computed tomography and magnetic resonance imaging. *Ann Neurol*, 19, 291-293.

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QUESTIONNAIRE

DESMOND FALL RISK QUESTIONNAIRE

Please answer all questions.

Name _____ Date _____

YES NO

1. ☐ ☐ Have you had a fall or near fall in the past year?
2. ☐ ☐ Do you have a fear of falling that restricts your activity?
3. ☐ ☐ Do you experience dizziness or a sensation of spinning when you lie down, tilt your head back, or roll over in bed?
4. ☐ ☐ Do you feel uneasy or unsteady when walking down the aisle of a supermarket or in an area congested with other people?
5. ☐ ☐ Do you have difficulty walking in the dark or on uneven surfaces such as gravel or a sloped sidewalk?
6. ☐ ☐ Do your feet or toes frequently feel unusually hot or cold, numb or tingly?
7. ☐ ☐ Do you wear bifocal or trifocal glasses, or is your vision notably better in one eye?
8. ☐ ☐ Do you experience loss of balance or a lightheaded/faint feeling when you stand up?
9. ☐ ☐ Do you take medication for depression, anxiety, nerves, sleep or pain?
10. ☐ ☐ Do you take four or more prescription medications daily?
11. ☐ ☐ Do you feel like your feet just won't go where you want them to go?
12. ☐ ☐ Do you feel like you can't walk a straight line or are pulled to the side while walking?
13. ☐ ☐ Has it been longer than six months since you participated in a regular exercise program?
14. ☐ ☐ Do you feel that no one really understands how much dizziness and balance problems affect your quality of life?
15. ☐ ☐ Are you interested in improving your balance and mobility?

PHYSICIAN'S GUIDE TO THE DESMOND FALL RISK QUESTIONNAIRE

Questions 1 and 2: A previous fall may indicate increased risk for future falls. Inquire as to the circumstances of the fall. Fear of falling can lead to restricted activity.

Questions 3, 4 and 5: A positive response to any of these questions indicates the possibility of a vestibular disorder. Patients with benign paroxysmal positional vertigo (BPPV) are at risk for falling if they tilt their head back. Patients with vestibular disorders tend to be more reliant on vision for postural control. When the visual feedback is unreliable (moving visual scene) or unavailable (dark), they are at risk for loss of balance and falling. Vestibular evaluation may be indicated (e.g., VNG, posturography, rotary test).

Questions 5 and 6: The sense of touch is an important contributor to balance and orientation. The stretch receptors in the legs, fingertips and soles of the feet all provide sensory feedback for balance. An assessment for peripheral neuropathy may be indicated.

Question 7: Vision plays an important role in balance, and patients with visual deficits have greater risk for falls. Visual problems associated with decreased postural stability include: 1) visual acuity less than 20/50; 2) asymmetric vision impairing binocular vision and depth perception; 3) slow pupillary reaction causing increased adaptation time when going from a lighted to a dark room and vice versa; and 4) impaired peripheral vision.

Multifocal glasses have been shown to increase the risk of falling (Lord et al, 2002). Ophthalmology evaluation may be indicated.

Question 8: Orthostatic hypotension may result in an increased risk of falling when assuming the upright position. Diabetes and many medications used to regulate heart rate and blood pressure can lead to orthostatic hypotension.

Questions 9 and 10: The use of four or more daily prescription medications or the use of tricyclic anti-depressants and/or benzodiazepine is associated with increased risk for falls (Tinetti et al, 1994).

Questions 11 and 12: Poor motor control is a sign of possible cerebellar dysfunction. The integration of vestibular, visual and somatosensory information takes place in the cerebellum. Cerebellar dysfunction can result in slow or inappropriate reaction to self movement or external visual stimuli.

Question 13: Inactive patients may have accelerated decrease in muscle mass and prolonged reaction time when faced with a possible fall.

Question 14: Physicians often underestimate (compared to the patient) the impact that a balance problem has on the patient's quality of life (Honrubia et al, 1996).

Question 15: Therapy for improved balance requires motivation and commitment. Patient compliance is important to a successful fall prevention program.

Lord SR, Dayhew J, Howland A. (2002) Multifocal glasses impair edge contrast sensitivity and depth perception and increase the risk of falling in older people. *J Am Geriatr Soc*, Nov;50(11):1760-6

Tinetti, et al. (1994) A multifactorial intervention to reduce the risk of falling among elderly people living in the community. *N Engl J Med*, 331(13):821.

Honrubia et al (1996) Quantitative evaluation of dizziness characteristics and impact on quality of life. *Am J Otol*, 17(4), 95-102 Physicians

QUESTIONNAIRE

DETAILED DIZZINESS HISTORY QUESTIONNAIRE

Name: _____ Age: _____ Date: _____

WHEN was the first time you ever had dizziness? _____

WHAT were the circumstances? _____

WHEN was the last time you experienced dizziness? _____

WHAT were the circumstances? _____

Currently, your dizziness...

- ☐ is constant.
- ☐ is always there, but changes in intensity.
- ☐ comes in episodes.

If it comes and goes:

How long does it typically last? _____ seconds / minutes / hours (Circle ONE)

How often does it typically occur? _____ times per: hour / day / week / month / year

Your dizziness mostly consists of... (check ALL that apply)

- ☐ spells of spinning with nausea.
- ☐ off-balance sensation.
- ☐ a light-headed or near faint sensation.
- ☐ other. Please explain _____

Between episodes you feel... (check ONE)

- ☐ dizzy or off balance all the time.
- ☐ normal.
- ☐ other. Please explain _____

Episodes occur... (check ALL that apply)

- ☐ spontaneously. Nothing seems to bring them on or turn them off.
- ☐ only when standing or walking.
- ☐ in relation to any head motion.
- ☐ only in certain head positions. Please describe _____

When you roll over in bed... (check ONE)

- ☐ nothing unusual happens.
- ☐ the room seems to spin sometimes.

Is there anything you can do to make your dizziness go away? (sit, lay down, close eyes)

Explain: _____

Circle all that apply:

Difficulty hearing Right Left Both
 Ringing or other sounds Right Left Both
 Ear fullness Right Left Both
 Ear surgery Right Left Both

Circle YES or NO

Did you have cold, flu or virus type symptoms shortly before the onset of your dizziness? . . . YES / NO
 Did you cough, lift, sneeze, fly in a plane, swim under water or have a head trauma shortly before the onset of your dizziness? YES / NO
 Were you exposed to any irritating fumes, paints, etc. at the onset of your dizziness? YES / NO
 Do you get dizzy when you have not eaten for a long time? YES / NO
 Is your dizziness connected with your menstrual period? YES / NO
 Did you get new glasses recently? YES / NO
 Do you consider yourself to be an anxious or tense type of person? YES / NO

In the past year, have you had: (Check ALL that apply)

<input type="checkbox"/> loss of consciousness	<input type="checkbox"/> double vision	<input type="checkbox"/> palpitations of the heartbeat
<input type="checkbox"/> seizures or convulsions	<input type="checkbox"/> spots before the eyes	<input type="checkbox"/> tingling around mouth
<input type="checkbox"/> slurring of speech	<input type="checkbox"/> occasional loss of vision	<input type="checkbox"/> tendency to fall
<input type="checkbox"/> difficulty swallowing	<input type="checkbox"/> severe pounding headache or migraine	<input type="checkbox"/> loss of balance when walking
<input type="checkbox"/> weakness in one hand, arm or leg		

Do you or have you had: (Check ALL that apply)

<input type="checkbox"/> diabetes	<input type="checkbox"/> stroke	<input type="checkbox"/> high blood pressure
<input type="checkbox"/> migraine headaches	<input type="checkbox"/> arthritis	<input type="checkbox"/> a neck and/or back injury
<input type="checkbox"/> irregular heartbeat	<input type="checkbox"/> allergies	

Please check below for any MEDICATIONS you have tried for DIZZINESS or are currently taking:

	TAKEN IN PAST	TAKING NOW	HELPS
Antivert® (Meclizine)	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Valium® (Diazepam)	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Dyazide® "water pills"	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>

Have you ever been previously evaluated for dizziness?

Where? When? _____

QUESTIONNAIRE

SHORT FORM QUESTIONNAIRE

Name: _____ Age: _____ Date: _____

Please circle the number that **BEST** describes your symptoms. Circle **ONLY ONE** number.

1. The room spins for less than one minute when I lie down, roll over in bed, or tilt my head back.
2. I get temporarily dizzy, light-headed or off-balance when I stand up, worse if I stand quickly.
3. I have had several episodes of severe vertigo with nausea lasting for hours at a time, with fullness and noise in one ear that increases when I am dizzy.
4. I have had several episodes of severe vertigo lasting hours at a time, sometimes accompanied by headache and/or sensitivity to light.
5. I had an episode of constant spinning vertigo and nausea for one to three days, and I feel like I have not recovered back to normal yet.
6. I am unsteady whenever and as long as I am on my feet. I am fine while sitting or lying down.
7. I don't really feel dizzy or off balance, but I am afraid of falling (or have already fallen).
8. None of these describe my symptoms.

If you have circled more than one number, please go back and correct.

On a scale of 1 to 10, with 1 being mildly annoying, and 10 being debilitating and life-altering, how would you rate the severity of your dizziness or imbalance? _____

SHORT FORM QUESTIONNAIRE GUIDE

This guide corresponds to the Short Form Questionnaire on the previous page. You may copy the previous page and ask the patient to circle the ONE description that BEST fits their symptoms. In 2017-18, we performed a validation study of this intake form, and found it to be predictive of final diagnosis in 62% of patients, but 80% accurately predictive of orthostatic hypotension and 90% accurately predictive of Ménière's disease. This allows quick reduction in likely differential diagnosis, and helps determine which patients may benefit from vestibular evaluation.

Most likely diagnosis:

- 1. Benign Paroxysmal Positional Vertigo.** Perform Dix-Hallpike exam, treat or refer (pages 18–19).
- 2. Orthostatic Hypotension.** Record blood pressures, supine; then immediately after standing; and at one-minute intervals for three to five minutes (pages 30–31).
- 3. Ménière's Disease.** Refer for audiometric and vestibular function testing (pages 22–23).
- 4. Vestibular Migraine.** Inquire about history or family history of migraine and complaints of self-motion or visual motion intolerance. Treat or refer for neurology consult (pages 24–25).
- 5. Acute Vestibular Syndrome (aka: Vestibular Neuritis).** Perform head impulse test and refer for vestibular function testing. If head impulse negative, consider infarct (pages 20–21).
- 6. Postural or Gait Instability.** Inquire about strength, sensation and stability of the lower extremities and/or assess nerve conduction.
- 7. Multifactorial Dysequilibrium.** Have patient complete fall risk questionnaire, assess neurologic status and refer as indicated (pages 32–33).
- 8. Non-specific.** Ask patient to describe symptoms.

We find that the majority of patients will choose the description that best fits their final diagnosis determined after extensive interview and vestibular function testing.

Physician referrals can be made through the STAR line at **336-713-STAR (7827)**.

VESTIBULAR FUNCTION TESTS

Vestibular function tests help identify deficits in labyrinthine response to rotational movement, impulsive head movement, caloric stimulation, and position change, as well as screen for abnormalities in ocular motility. Abnormal eye movements associated with vestibular pathology (nystagmus) is often suppressed when examined in a lighted room. With vestibular function testing, the eye movements are recorded with infra-red assisted video goggles. These tests have been shown to provide additional diagnostic information when the cause of dizziness is not apparent through clinical examination. Common vestibular function tests include:

VNG (Videonystagmography)

Many inner ear disorders cause nystagmus. This nystagmus can be recorded and analyzed through VNG testing. Stimulation of the inner ear for this test includes rapid position changes of the head and body, and caloric (temperature) stimulation of the inner ear through irrigating the ear canal with different temperatures of water or air. The goal is to determine if both inner ears are functioning and responding equally to the stimulation. Additionally, tests of voluntary eye movement serve as a screening for possible neurologic disorders.

Audiometry

Audiometric testing (hearing evaluation) provides information regarding the health and integrity of the eardrums, may identify middle ear fluid that can affect balance, and detect auditory asymmetry associated with inner ear or auditory/vestibular nerve pathology.

Less commonly available vestibular function tests include:

ROTATIONAL CHAIR

Rotational chair testing stimulates the horizontal semi-circular canal and avoids stimulating any

other part of the balance system. It is a very sensitive test of inner ear abnormality. The patient is placed in a motorized rotating chair and eye movements are recorded and analyzed. The patient's ability to enhance or suppress the VOR also provides information about the central processing of labyrinthine responses.

vHIT (Video Head Impulse Test)

So that we may maintain visual stability and focus on objects while moving our head, nature provides us with a vestibular ocular reflex (VOR). An impairment of this reflex action can cause imbalance or visual blurring with head movement. vHIT evaluates the patient's ability to use their VOR efficiently to maintain visual stability at fast speeds of head movement.

CDP (Computerized Dynamic Posturography)

CDP allows us to evaluate a patient's reaction to a variety of difficult balance situations. This is done by having the patient stand on a platform that measures center of gravity and any sway from the center. Visual and tactile information are systematically denied or distorted. By analyzing the patient's response, we can determine what type of situation is most likely to result in a fall.

VEMP (Vestibular Evoked Myogenic Potentials)

The cervical VEMP test is a test of otolith function, particularly the saccule. The ocular VEMP is a test of the utricle. Whereas most vestibular tests examine the horizontal semi-circular canals and superior vestibular nerves, the VEMP test examines the function of the inferior vestibular nerve.

ABR (Auditory Brainstem Response)

The ABR test evaluates the integrity of the auditory/vestibular nerve and serves as a cost effective screening for acoustic neuroma/vestibular schwannoma. When a significant hearing asymmetry exists, cranial MRI is the preferred test.

MOTT (MOST OF THE TIME) LIST

General rules to aid in diagnosis.

- A complaint of vertigo **MOTT** indicates a peripheral vestibular asymmetry, but can mean migraine or infarct.
- A complaint of lightheadedness or faintness **MOTT** is not vestibular.
- Vertigo of less than one minute duration when lying down or tilting head **MOTT** indicates BPPV.
- Lightheadedness / presyncope with transient loss of balance of less than one minute after rising **MOTT** indicates orthostatic hypotension.
- Recurrent vertigo with light sensitivity **MOTT** indicates migraine.
- Recurrent vertigo with associated symptoms such as unilateral tinnitus and/or hearing loss, particularly at the time of an episode of dizziness, **MOTT** indicates Ménière's syndrome.
- Symptoms that increase with eyes closed or with a change in head position **MOTT** indicates vestibular etiology.
- Symptoms noted only while standing **MOTT** are related to vascular or orthopedic disease.
- Symptoms such as syncope, numbness, tingling, confusion and slurred speech **MOTT** indicate CNS disease.
- Nystagmus that diminishes with visual fixation, and/or is direction-fixed **MOTT** is of peripheral vestibular origin.
- Nystagmus that is vertical or direction changing without change in head position is **MOTT** due to CNS disease.
- Nystagmus that increases when gaze is directed toward the fast phase, and decreases when gaze is directed toward the slow phase is **MOTT** a sign of acute peripheral vestibular asymmetry.

BENIGN PAROXYSMAL POSITIONAL VERTIGO (BPPV)

Typical Complaints

"I woke up and the bed was spinning."

"I bent over (rolled over, turned quickly, laid down, sat up from bed, etc.) and everything started spinning around."

"Every time I tried to get up I fell back into the bed."

Defining Questions

- If you lay perfectly still would the dizziness pass until you moved again? (Should)
- Was the dizziness constant even if you didn't move? (Should not)
- If you forced yourself to get up and move around, would it ease up throughout the day until you laid back down? (Should)

Many patients will tell you they were dizzy for hours or all day. Upon detailed questioning, BPPV patients should tell you that the dizziness occurred in short but intense spurts, but was not constant or non-stop for several hours. Many will state that each episode lasted for several minutes, when in fact it lasted less than one minute. It is a pretty awful minute.

Medical History Questions

- Have you ever had anything like this before? (BPPV has a high incidence of recurrence.)
- Do you have a history of head trauma, either significant in the past, or minor in recent weeks/months? (Head trauma is the most frequent cause in patients under age 50.)

Definition

"BPPV is by far the most common cause of episodic vertigo. Patients typically report brief episodes (less than one minute) of intense vertigo, usually brought on by lying down, rolling over in bed, or tilting the head back. BPPV is a mechanical dysfunction of the inner ear, and

does not usually represent an ongoing disease process. It is typically diagnosed with the Dix-Hallpike maneuver (DHM), but a negative DHM does not rule out BPPV. BPPV does not respond to medication, but rather is most effectively treated by canalith repositioning procedures. The typical pattern of BPPV is one of intermittent episodes. The vertigo (spinning sensation) may occur frequently for weeks at a time, disappear for months, then reappear with no warning.

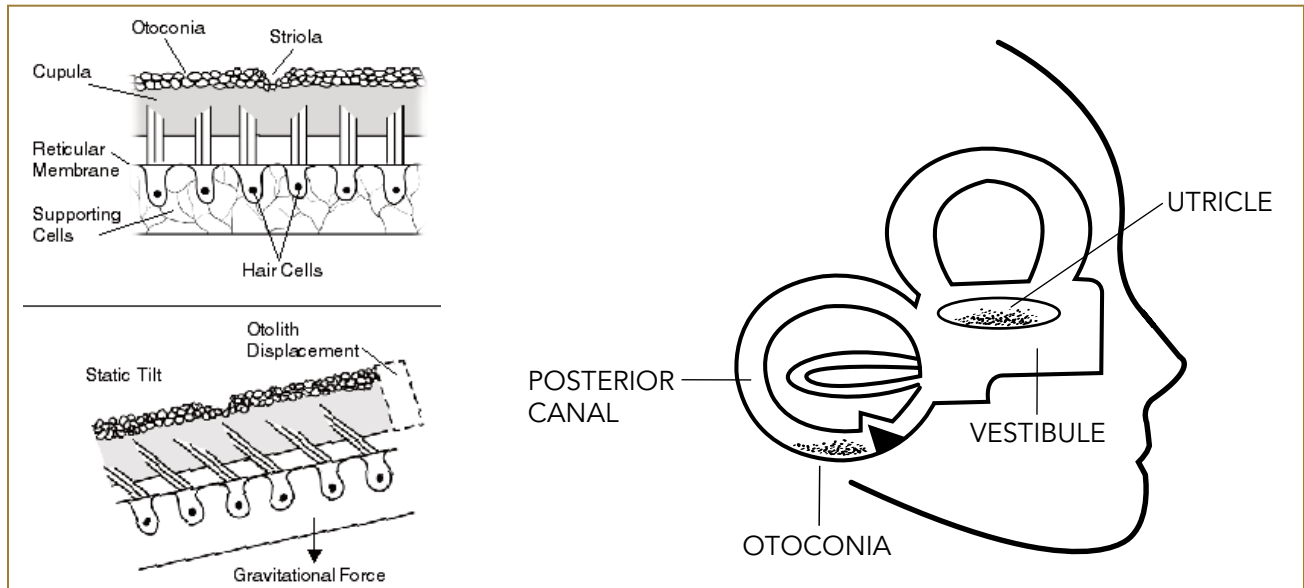
BPPV is believed to be a result of a plug of calcium carbonate and protein crystals (otoconia), which have become dislodged from the utricle, settling most frequently in the posterior semicircular canal. Otoconia cause no problem until the patient moves in a manner stimulating the offending semicircular canal. The otoconia then begin moving, causing abnormal stimulation of the motion sensor in the affected ear. While the otoconia are in motion (typically 15 to 45 seconds), the patient is experiencing conflicting signals from the two labyrinths of the inner ear, resulting in a brief but intense period of vertigo."

(Excerpt from Vestibular Function: Evaluation and Treatment, Desmond, 2004)

Management Options: Canalith repositioning (aka, Epley maneuver) is the recommended treatment for posterior canal BPPV. In some cases, home-based repositioning exercises may be appropriate. It is important that the patient be examined with the DHM prior to recommending home exercises. If home exercises are recommended, the home Epley maneuver has been shown to be significantly more effective than Brandt-Daroff exercises.

Occasionally, nystagmus inconsistent with posterior canal BPPV will be noted, rendering the Epley maneuver inappropriate. If CRP or exercises are contra-indicated, a period of observation may be warranted. Vestibular suppressant medications such as meclizine or diazepam are not appropriate treatments for BPPV.

BENIGN PAROXYSMAL POSITIONAL VERTIGO (BPPV)



BPPV is by far the most common cause of episodic vertigo. BPPV causes brief (less than one minute) episodes of intense vertigo, triggered by certain movements such as lying down or rolling over in bed, or tilting the head. It occurs as a result of some small but heavy particles called otoconia coming loose from the utricle and settling into one of the semi-circular canals (SCCs). The most common cause of this is head trauma, followed by aging and degeneration of the inner ear structures.

Normal Function

The SCC's are C shaped tubes, made of bone, full of inner ear fluid. You have three SCCs in each ear, and they are at different angles so that with any head tilt, at least one SCC on each side is stimulated. As you move or tilt your head, the fluid moves in the canals, and small sensors called the cupula (membrane barrier) register that fluid movement and send signals to the brain regarding movement. As long as both ears register the same movement, no symptoms will occur.

BPPV occurs as a result of the otoconia debris settling into a SCC while you are still for a while.

When you change head orientation (tilting, rolling over, lying down), the otoconia debris moves through the SCC causing a temporary fluid disturbance, much like the wake from a speeding boat. Until that fluid comes to rest (typically 15 to 45 seconds), the two inner ear structures are sending conflicting signals to the brain. The result is brief, but intense, vertigo.

This condition is easily diagnosed when the symptoms are active at the time of examination. Occasionally, the symptoms can not be reproduced on examination. This does not mean you do not have BPPV, but you may require additional examination or a diagnostic trial of home exercises known to promote recovery from BPPV.

Once the condition has been diagnosed, a simple procedure known as canalith repositioning resolves the symptoms quickly, with over 90 percent effectiveness for the most common variant. BPPV is known to reoccur in up to 50 percent of patients diagnosed with this condition. There is no way to predict or prevent recurrences. However, treatment can be provided for quick relief.

ACUTE VESTIBULAR SYNDROME: VESTIBULAR NEURITIS / LABYRINTHITIS

Typical Complaints*:

"The room just started spinning and wouldn't stop."

"I had constant spinning and nausea for about three days."

"I was in the hospital for about 3 days, and they couldn't find anything wrong with me."

"After the worst of it (vertigo and nausea) I was okay if I didn't move. If I moved I was off balance and would get nauseous if I moved too much."

* With labyrinthitis, there will also be the complaint of sudden onset of unilateral hearing loss and tinnitus.

Defining Questions:

- Did you have any viral type symptoms (fever, chills, diarrhea, etc.) in the few weeks prior to the onset of vertigo? (Common)
- Do you get cold sores around your lips? (A reactivation of herpes virus is thought to be a causative factor.)

Diagnosis/Definition

"Acute vestibular syndrome is most often the result of viral inflammation of the vestibular nerve— known as vestibular neuritis—but can also be the result of a stroke involving the arteries close to the inner ear. There is no way to be absolutely certain of the source of the vertigo in the early stages, but vestibular testing is at least as sensitive as imaging studies.

Vestibular neuritis* is characterized by a sudden onset of vertigo without any associated auditory symptoms. Vertigo is usually restricted to one attack, but a minority of patients may have repeated attacks. The vertigo is usually prolonged—lasting twenty-four hours or

*With labyrinthitis, there are unilateral complaints of tinnitus and decreased hearing.

more—and is accompanied by nausea, vomiting, spontaneous nystagmus and postural instability. Vestibular neuritis is thought to be the result of a viral inflammation of the VIIIth nerve causing dysfunction of the superior division of the vestibular nerve. No specific virus has been deemed responsible; however, serology has demonstrated increased viral antibody titers to several viruses including herpes simplex, Epstein-Barr, rubella, influenza and cytomegalovirus. Acute symptoms can be reduced through the use of vestibular suppressant and/or anti-emetic medications; however, gradual reduction of sedating medication and increased activity to promote central compensation is recommended. Vestibular evaluation will usually demonstrate a caloric hypofunction on the affected side. Depending on the level of central compensation, direction fixed nystagmus that reduces with visual fixation, and reduced VOR gain on rotary tests may be noted."

(Excerpt from Vestibular Function: Evaluation and Management, Desmond, 2004)

Management Options:

Acute vertigo and nausea may be relieved by vestibular suppressant or anti-emetic medication. These medications should be withdrawn once the acute vertigo has subsided (typically three to five days). Some practitioners also use a short course of oral steroids unless medically contraindicated. Vestibular evaluation can identify the location and extent of labyrinthine injury. Vestibular rehabilitation exercises should begin once the acute phase has passed. Extended use of vestibular suppressant medication inhibits recovery. Sudden onset sensori-neural hearing loss is a medical emergency and should be treated accordingly.

ACUTE VESTIBULAR SYNDROME: VESTIBULAR NEURITIS / LABYRINTHITIS

Acute vestibular syndrome (AVS)/vestibular neuritis (VN) is typically characterized by sudden onset vertigo, frequently accompanied by nausea and vomiting. These acute symptoms usually last for several hours, but gradually decrease over a period of three to five days (faster with vestibular suppressant medications). Once the acute symptoms have passed, you may be fairly comfortable while lying or sitting still. When you feel good enough to get up and walk around, you may notice that you are unsteady on your feet, have blurred vision if you move your head quickly, and can become nauseous if you push yourself too much.

AVS is most often caused by inflammation of the nerve that connects the inner ear to the brain, usually the result of a virus, but can also be the result of a small stroke in the blood vessels near the inner ear. When the nerve becomes inflamed, the information from the inner ear on that side is reduced suddenly, and sometimes drastically. The severity of AVS/VN can range from mild temporary inflammation that resolves in a few days with no lingering abnormalities, up to total and permanent destruction of the nerve pathway. Typically, it falls somewhere in between these two extremes. Labyrinthitis additionally involves the auditory nerve connected to the cochlea (the hearing portion of the inner ear). In addition to vertigo, labyrinthitis is characterized by sudden loss of hearing and a roaring noise in the affected ear.

From a functional standpoint, when the nerve function on one side is disrupted, you develop a sense of rotation because the brain is not receiving equal signals from the two inner ears. Because the inner ears are sending a signal to the

brain that you are moving, while the rest of your body knows that you are not moving, this creates a conflict within your balance system. This conflict causes symptoms of nausea (possibly vomiting) and pallor (paleness).

The brain tries to correct this conflict by shutting down its sensitivity to both inner ears, a process called “cerebellar clamp.” This process typically takes two to three days. Once this process is completed, there is less conflict between the inner ears and the rest of the body—until you start moving. Now, the rest of the body senses movement (because you are moving), but the inner ears are not registering the same amount of movement because the brain has “shut down” its connection to them. So now, your body knows it is moving, but the inner ears are not keeping up. This creates a similar conflict causing motion-provoked nausea, visual instability and disorientation.

This conflict won’t go away simply by waiting. You must retrain your brain to use the information provided by the inner ears through rehabilitation exercises. These exercises work more effectively if you are not taking any type of sedating medication such as Antivert® (Meclizine). Your vestibular specialist will guide you on these exercises.

MÉNIÈRE'S DISEASE

Typical Complaints

"I have had several episodes of severe vertigo with nausea, lasting for hours at a time."

"It didn't matter if I moved or not."

"I feel so much pressure in my ear (head)."

"My ear was roaring."

"After an episode, I need to sleep for several hours."

"I was drenched in sweat."

Defining Questions

- Is the roaring and ear pressure something that occurs only on the same day as the vertigo, or is it with you most of the time?
- Is there a change in the tinnitus or pressure when you have an episode? (Should notice a change around the time of the episode.)
- Which ear is it that bothers you? (Most Ménière's patients can quickly tell you which ear.)
- Do you have any family members with similar problems? (There is a one in three chance of a close relative having Ménière's.)



Ménière's disease is the most controversial and potentially debilitating inner ear disorder. There is general agreement that there is a relationship between endolymphatic hydrops (a buildup of endolymph volume and pressure

within the labyrinth) and Ménière's disease, but there is disagreement as to why this happens. Plausible theories include obstruction of the endolymphatic or reuniting duct, inability of the endolymphatic sac to absorb excess endolymph, an overproduction of endolymph, migraine variant or auto-immune disorder.

Our experience has been that Ménière's disease is over-diagnosed at both the primary care and specialist level. Many patients who have been on a restricted diet for years are found to have no history or audio-vestibular findings consistent with Ménière's disease.

Definition

"Ménière's disease is a disorder of the inner ear, which in its classic presentation is characterized by episodes of vertigo, unilateral decreased hearing, increased tinnitus and aural fullness on the affected side. The cause of Ménière's disease is unknown, but is thought to be related to an inability of the ear to regulate endolymph, resulting in an episodic buildup of pressure in the membranous labyrinth. The Committee on Hearing and Equilibrium of the American Academy of Otolaryngology defines Ménière's disease as an 'idiopathic syndrome of endolymphatic hydrops.' Endolymphatic hydrops varies greatly in symptomology, duration and frequency of episodes. In the early stages of the disease, examination between episodes can be completely normal. As the disease progresses, decreased hearing and reduced vestibular sensitivity are typically noted in the affected ear. Variations of the disease have been described as 'cochlear' or 'vestibular' hydrops. In these conditions it is hypothesized that the isolated symptoms (fluctuating hearing OR episodic vertigo) are related to hydrops restricted to certain portions of the labyrinth."

(Excerpt from Vestibular Function: Evaluation and Treatment, Desmond, 2004)

Diagnosis of Ménière's disease is primarily based on the patient's report of repeated episodes of the above group of symptoms. Audiometric evaluation may reveal an asymmetrical, typically low frequency sensorineural hearing loss. Inspection for nystagmus during an attack may reveal spontaneous nystagmus following Alexander's law. Unlike other vestibular disorders, the nystagmus may beat (fast phase) towards the lesioned side. This is felt to be the result of increased vestibular output during the inflammatory process of a hydrops attack. Vestibular evaluation will usually reveal a caloric hypofunction on the affected side in patients with lengthy histories of episodic vertigo. In the early stages of the disease, caloric testing may be normal, or may even reveal a stronger caloric response in the pathologic ear due to inflammation of the labyrinth.

A consensus statement produced by the Barany Society has set specific criteria for definite and probable Ménière's disease. The criteria for definite Ménière's disease includes: two or more spontaneous episodes of vertigo, duration between 20 minutes and 12 hours, documented low- to mid-frequency sensori-neural hearing loss on the suspected side, fluctuating hearing loss, tinnitus and fullness in the suspected ear, not better accounted for by another vestibular diagnosis. The criteria for probable Ménière's disease is similar, but without documented hearing loss.

Management Options:

Treatment is best managed by an otolaryngologist, otologist or neuro-otologist. Treatment options include a salt-restricted diet and/or use of a diuretic (both to reduce fluid retention), vestibular suppressant medications (to reduce the intensity of vertigo and nausea during an attack), surgery and chemical ablative procedures. Low-dose intra-tympanic gentamicin or intra-tympanic steroids have provided relief without ablation.

For a detailed review and excellent pictorials, please visit the Cochlear Fluids Lab at Washington University (<http://oto.wustl.edu>) or Dr. Timothy Hain's site at Northwestern University (www.dizziness-and-balance.com).

For a patient handout, the 16-page booklet on Ménière's disease available from Krames (1-800-333-3032) is recommended.

Lopez-Escamez (2015) Diagnostic Criteria for Ménière's Disease. Jnl Vest Rs 25

Note: In the past, many patients who were diagnosed with vestibular hydrops are now suspected of having vestibular migraine.

VESTIBULAR MIGRAINE

Typical Complaints

"I have episodes of spinning and nausea that come on without warning, but have no ear symptoms."

"The episodes are sometimes accompanied by headache or sensitivity to light."

"I felt a sudden wave come over me."

"I can't stand any type of motion. It never bothered me when I was a kid."

"I just feel kind of 'spaced out' a few days a month."

Note: The symptoms of vestibular migraine are so varied, the above represents only a small sample of related complaints.

Defining Questions

- Do the episodes seem to occur on a regular monthly basis? (Migraine is more common in women, and symptoms tend to be worse around the menstrual cycle.)
- Have you ever had classic migraines with headache and sensitivity to light and sound? (Migraine symptoms can change over time, particularly after a hormonal event like pregnancy, childbirth or menopause.)
- Do you have any fullness or tinnitus in one ear at the time of an episode of vertigo? (Generally, episodic vertigo of migraine etiology is not accompanied by any unilateral auditory symptoms.)

Medical History Questions

- Is there a family history of migraine? (There is a higher likelihood of migraine when one or both parents have a history of migraine-type headaches.)

Diagnosis/Definition:

The diagnosis of vestibular migraine (as well as classic migraine) is accomplished largely by clinical history, negative examinations for other causes of vestibular symptoms and a positive response to migraine treatment.

A Joint Committee of the International Headache Society and the Barany Society has developed specific criteria for the diagnosis of vestibular migraine. These include: 1) At least five episodes of vestibular symptoms; 2) Current or previous history of migraine; 3) Headache, photophobia, phonophobia or visual aura with at least 50 percent of episodes; and 4) Not better accounted by another vestibular diagnosis.

Lempert et al (2012) Vestibular Migraine: Diagnostic Criteria. Jnl Vest Rs. 22

Management Options:

Vestibular evaluation may identify or effectively rule out labyrinthine dysfunction. A neurologist familiar with the characteristics and treatment of vestibular migraine may be consulted.

VESTIBULAR MIGRAINE

Most people associate “migraine” with “headache.” While it is true that headache is a common symptom of migraine, it is not always part of the symptom complex. Migraine can cause episodes of vertigo (spinning), motion intolerance, nausea and spatial disorientation. Many patients with migraine are mistakenly diagnosed with sinus headaches or Ménière’s disease (an inner ear condition responsible for episodic vertigo).

Vestibular migraine symptoms

There are two typical presentations (and many atypical presentations) of vestibular migraine: 1) episodes of vertigo lasting minutes to hours, often (but not always) accompanied by headache, visual disturbance, sensitivity to light or sound; and 2) Intolerance to self-motion and/or external visual motion intolerance, frequently beginning in adulthood.

Migraine triggers

While the exact mechanism of migraines may be uncertain, there are known triggers that may lead to a migraine attack. Hormonal changes; certain foods such as cheese, chocolate and red wine; stress; bright or flickering light; changes in atmospheric pressure; caffeine withdrawal and disruption of sleep patterns have all been associated with triggering migraine symptoms.

Treatment

Migraine symptoms are typically not treated by ENT or audiology specialists. If vestibular migraine is suspected, you may be referred to a neurologist familiar with the variants and current treatments for migraine. The treatment is generally similar to treatment for classic migraine.



SUPERIOR CANAL DEHISCENCE SYNDROME (SCDS)

Typical Complaints

"I get dizzy around loud noise, when I blow my nose, lift something heavy."

"My own voice, chewing, eye movements, seem too loud."

"I hear my heartbeat in my ear(s)."

Defining Questions

- Did your symptoms start after a plane flight, deep water diving, or incident of head trauma? (Sudden changes in intra-tympanic air pressure or mild head trauma can cause thin bone to open leading to a canal dehiscence.)
- Have you had previous vestibular evaluation? (SCDS is often missed or misdiagnosed through standard vestibular testing such as videonystagmography.)

Definition/Description

Superior canal dehiscence syndrome (SCDS) causes patients to experience vertigo and oscillopsia in response to loud noises or changes in middle ear pressure. Auditory complaints and findings may include autophony, hypersensitivity to bone-conducted sound, and a small, low-frequency air-bone gap on audiometric testing.

SCDS occurs when there is an abnormal opening (dehiscence) in the bony wall of the superior semi-circular canal. (Note: Audiologists often refer to this same canal as the posterior canal.) SCDS is thought to be developmental, but can be triggered by trauma in patients with very thin bone surrounding the superior canal.

Physiologically, SCDS brings on transient vertigo and oscillopsia (visual blurring) as a result of asymmetric labyrinthine fluid response to sound and pressure. The dehiscence acts as a "third window" in the labyrinth. The oval window is

associated with instigating fluid motion in the cochlea, and the round window allows that fluid motion to dissipate. The "third window" alters the fluid dynamics within the labyrinth.

Clinical signs of SCDS include torsional nystagmus associated with induced sound or pressure changes in the affected ear. SCDS is diagnosed by high resolution CT scanning using a very specific technique. CT scanning using 10 mm slices may detect only half of patients with SCDS, while 5 mm slices will detect over 90 percent (Belden, Weg, Minor, & Zinreich, 2003).

"The standard battery of vestibular testing would not demonstrate any abnormality associated with SCDS as they focus on the horizontal canals. Clinical findings consistent with SCDS include: 1) Vestibular evoked myogenic potentials (VEMPs) may be identified at lower stimulus levels, or may have a higher amplitude response to equal stimulus than the non SCDS ear; and 2) audiometric evaluation may demonstrate a low frequency conductive loss with a normal tympanogram. SCDS has been mistakenly diagnosed as otosclerosis. The examiner should be careful to view acoustic reflexes, as these remain present in SCDS and would be absent in most patients with otosclerosis."

(Excerpt from Vestibular Function: Clinical and Practice Management, Desmond, 2011)

Management Options:

Once the diagnosis is made, some patients may choose to employ an avoidance strategy (avoiding loud sounds, wearing an earplug in the affected ear in some settings, avoiding heavy lifting/straining). Others may benefit from surgery, which may include repair of the dehiscence, or plugging of the affected canal.

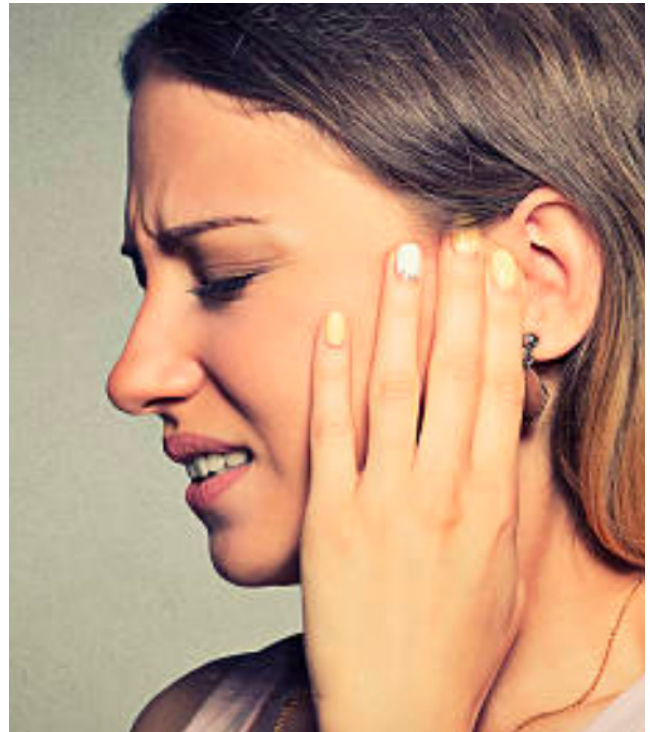
SUPERIOR CANAL DEHISCENCE SYNDROME (SCDS)

Superior canal dehiscence is an abnormal opening in the inner ear inside the skull. There is a bony plate that separates the inner ear from the brain. Some people are born with a defect where this bone is very thin and at risk for breaking through, leaving an abnormal opening between the superior semi-circular canal of the inner ear and the brain. Others develop the defect after an incident involving sudden pressure change, such as flying or swimming under water, or a bump on the head.

When the bone gives way, the fluid dynamics of that inner ear are altered. This presents in two main ways. Sounds generated internally (such as chewing, talking, even your pulse) have an opening for direct transmission into the affected inner ear, making these sounds seem too loud. When the fluid in the inner ear is stimulated by things such as loud sounds, blowing your nose, or physical strain from lifting something heavy, the difference in fluid response between the two inner ears can make you feel dizzy or cause transient visual disturbance.

SCDS has historically been misdiagnosed because this condition was discovered only 20 years ago, and routine testing for most disorders causing “dizziness” will not detect a canal dehiscence. Unless your doctor suspects SCDS based on your complaints, the special tests required to identify this condition may not be ordered.

For some people, SCDS can be managed by avoiding things that make you dizzy (such as wearing an ear plug when exposed to loud noise). For others, the bony defect can be surgically repaired with potential for immediate resolution of symptoms.



ANXIETY/HYPERVENTILATION

Typical Complaints:

"I felt like my heart was pounding out of my chest."

"I felt like I was standing outside my body."

"I felt like I was dying."

"I couldn't breathe."

"I just have this strange feeling in my head."

Defining Questions:

- Do you feel that stress increases your symptoms? (Frequently)
- When you are dizzy, do you notice: tingling around the lips? Tightness in the chest? Lump in your throat? Shortness of breath? (These symptoms are associated with stress-related dizziness.)

Medical History Questions:

- Do you take any medications for stress and/or anxiety? (You may review patient's medications.)

Definition:

"Hyperventilation is more of an anxiety disorder than a vestibular disorder; however, many of these patients' primary complaints are of dizziness or lightheadedness, which they feel is unprovoked. Many of these patients are unaware of any co-existing symptoms until questioned by a clinician. Hyperventilation occurs when "ventilatory effort exceeds metabolic need," and causes hypocapnia (low level of arterial P_{CO_2}) leading to constriction of the cerebral blood vessels and reduction of cerebral blood flow. Typical signs and symptoms include complaints of lightheadedness, frequent sighing, chest pain, numbness and tingling around

the mouth and hands. Some of these symptoms can be reproduced during examination in affected individuals."

(Excerpt: Vestibular Function: Evaluation and Treatment, Desmond, 2004)

Note: Reproducing symptoms of dizziness by having the patient voluntarily over-breathe for 60 to 90 seconds does not necessarily mean the patient is suffering from hyperventilation syndrome. It does give you some insight as to whether the patient's complaints are likely vestibular or non-vestibular in nature. The dizziness brought on by over-breathing is the result of decreased blood flow to the brain. If the patient's symptoms are similar to what they experience at home, it indicates their complaints are most likely non-vestibular. View the eyes under goggles while the patient is over-breathing. Some disorders of the vestibular nerve can result in dizziness and nystagmus from over-breathing.

Management Options:

Counseling the patient on the connection between anxiety and dizziness may break the cycle and bring some relief. Vestibular evaluation may identify nystagmus associated with over-breathing, and may identify vestibular dysfunction leading to situational disorientation and anxiety.

ANXIETY/HYPERVENTILATION

Anxiety and dizziness are commonly associated complaints. Whether dizziness leads to anxiety, or anxiety leads to dizziness is open to speculation. The truth is both can occur.

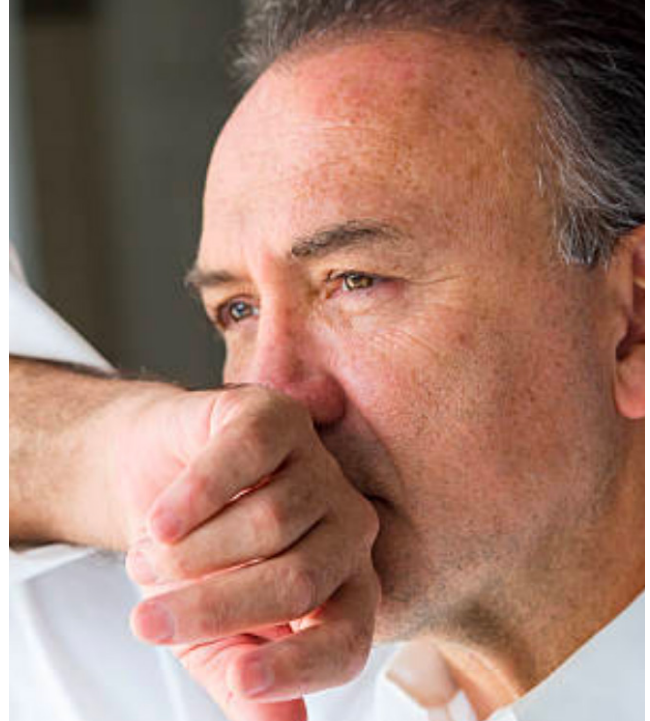
Anxiety leading to dizziness

Anxiety, stress or panic creates chemical changes in the body. We all have a “fight or flight” response to stress. This surge of adrenaline gives our body what it needs to either “fight” the source of danger (anxiety), or run away from the danger: “flight.” When adrenaline is released into our bodies, our heart rate increases and our blood pressure rises. A secondary effect is that our respirations increase. Picture yourself running around a track. Your heart rate and respirations are up, but at the same time, through exercise, you are burning off the extra oxygen that is being pumped into the blood. This exchange of bringing in more oxygen and burning off more oxygen keeps things in balance.

If you have elevated respirations and heart rate as a result of anxiety, you are bringing in more oxygen without burning it off sufficiently. The oxygen level gets too high in the blood stream, and the relative carbon dioxide level becomes too low. This causes a constriction of the cerebral blood vessels leading to a sensation of dizziness or lightheadedness. Other symptoms often associated with this condition include tingling around the lips and fingertips, shortness of breath and tightness in the chest. These symptoms can add further anxiety, exacerbating the condition.

Dizziness leading to anxiety

Patients with chronic, undiagnosed vestibular (inner ear) disorders are subject to a variety of anxiety reactions. The effects of vestibular disorders on quality of life are often underestimated, both by family and by physicians. First, there is frustration at not having a firm diagnosis



for such bothersome symptoms. Second, vestibular disorders are more bothersome when exposed to unfamiliar, unreliable or moving visual settings. An example of an unreliable visual setting would be a busy grocery store or market. Patients with vestibular disorders often become overly reliant on visual information for balance. In the busy market setting, all the movement, lights and colors make the visual information unreliable. This can make these types of environments very unsettling. Since it is not always easy to make the connection between the visual surrounds and the discomfort, many vestibular patients find themselves uncomfortable whenever they leave their own house. This describes an anxiety condition known as agoraphobia, which can severely limit one's enjoyment of life.

ORTHOSTATIC HYPOTENSION

Typical Complaints:

"I get dizzy and off balance when I stand up."

"I get up and start to walk and feel like I am going to fall over."

"When I get up quickly, I feel like I could faint."

Defining Questions:

- Is it worse in the first few seconds or minutes you are on your feet, but then improves after you have been up for a bit? (Should improve after seconds to minutes)
- Does it improve if you sit back down? (Should improve)
- Is it worse if you get up quickly? (Can be worse with rising quickly.)
- Is it more of a lightheaded/faint feeling, or more of a spinning feeling? (Should be more lightheaded/faint, but some describe vertigo.)

Medical History Questions:

- Do you take any medications for hypertension or diabetes?
- Do you take any diuretics?
- Are you careful to make sure you drink enough water?

Definition

"Orthostatic hypotension (OH) is a reduction of systolic blood pressure of at least 20 mm Hg or diastolic blood pressure of at least 10 mm Hg within three minutes of standing. It is a physical sign and not a disease. An acceptable alternative to standing is the demonstration of a similar drop in blood pressure within three minutes, using a tilt table in the head up position, at an angle of at least 60 degrees. Confounding variables to be considered when reaching a

diagnosis should include: food ingestion, time of day, state of hydration, ambient temperature, recent recumbency, postural deconditioning, hypertension, medications, gender and age. Orthostatic hypotension may be symptomatic or asymptomatic. Symptoms of OH are those that develop on assuming the erect posture or following head-up tilt and usually resolve on resuming the recumbent position. They may include lightheadedness, dizziness, blurred vision, weakness, fatigue, cognitive impairment, nausea, palpitations, tremulousness, headache and neck ache. If the patient has symptoms suggestive of, but does not have documented, orthostatic hypotension, repeated measurements of blood pressure should be performed. Occasionally, patients may not manifest significant falls in blood pressure until they stand for at least ten minutes."

(Consensus Statement, American Academy of Neurology, 1996)

Orthostatic hypotension can be evaluated by having the patient lie supine for five to ten minutes, then check blood pressure while still in the supine position. The examiner then asks the patient to stand up quickly, and rechecks the blood pressure immediately and again after about one minute, and three minutes. Be sure to hold the patient steady when they first stand up. Record as follows:

BP Supine BP Standing ASAP BP 1 minute BP 3 minutes

________ ________ ________ ________

Management Options:

A review of medications, including dosage and time of ingestion, is recommended. Increased hydration, brief exercise before rising and the use of support hose have been associated with reducing symptoms of orthostatic hypotension. See page 40 for a blood pressure log.

ORTHOSTATIC HYPOTENSION

Orthostatic hypotension (OH) is the most common source of the complaint of lightheadedness and loss of balance when standing. Orthostatic hypotension is defined as a temporary lowering of blood pressure (hypotension), usually due to suddenly standing up (orthostatic). Orthostatic hypotension may be experienced when rising quickly from a chair, especially after a meal, or when sitting up from bed in the morning. The change in position causes a temporary reduction in blood flow to the head and therefore a shortage of oxygen to the brain. This leads to lightheadedness and sometimes a “dizzy” episode. Occasionally, it may lead to a loss of consciousness.

Symptoms include dizziness, lightheadedness, feeling faint, loss of balance and visual disturbance, all due to insufficient blood flow to the brain. The symptoms are typically worse when standing and improve with lying down.

Regulation of blood pressure is part of the body’s autonomic nervous system, which can be compromised by diabetes and/or certain medications. Typically, in healthy individuals, the carotid sinus reflex assists in this regulation by causing constriction of the blood vessels in the lower body in response to standing. This vasoconstriction forces blood up towards the brain during the first minute or so after rising. During this time, there may be a measurable increase in blood pressure in the arm (since the arm is part of the lower body). After a couple of minutes, the vasoconstriction ceases and the blood pressure returns to its previous level. This temporary vasoconstriction in the lower body keeps a steady supply of blood to the brain when first standing.

Orthostatic hypotension can be the result medications used to control blood pressure, heart rate, bladder problems or anxiety. Many medical conditions can be the cause of OH—the most common being diabetes mellitus. There



are several variables that can affect OH, so the symptoms can vary throughout the day. Symptoms of OH tend to be more bothersome if the patient is dehydrated, as this can lead to decreased blood volume. Brief exercise before standing, such as crossing the legs several times or clenching fists, may minimize symptoms.

OH may be identified by a drop of 20 mmHg or greater in systolic blood pressure, or a drop of 10 mmHg or greater in diastolic blood pressure within three minutes of standing up. OH can not be ruled out based on a single negative blood pressure exam, and the test may need to be repeated at various times throughout the day. Patients with a home blood pressure monitor can keep track of changes in blood pressure sitting versus standing, and then report those findings to the primary care physician.

MULTIFACTORIAL DYSEQUILIBRIUM

Typical Complaints

"I have fallen more than once."

"I am afraid to go out because I am afraid of falling."

"I just lose my balance when I walk/turn around."

Defining Questions:

See Fall Risk Questionnaire and Physician's Guide on pages 10 and 11.

RISK FACTORS FOR FALLING

1. Vestibular pathology. An impairment of the vestibular system can cause the patient to become dizzy or off balance associated with certain movements and certain visual environments.

2. Polypharmacy. The use of four or more prescription medications or the initiation of a new medication or dosage has been associated with an increased risk of falling.

3. Use of tricyclic antidepressants or benzodiazepines. These medications are associated with increased risk of falls. SSRI antidepressants may have fewer side effects, but it is not clear that they result in a reduced risk of falling compared to tricyclics and benzodiazepines.

4. Orthostatic (postural) hypotension. Postural pre-syncope or lightheadedness associated with orthostatic hypotension may result in an increased risk for falling when assuming the upright position.

5. Impaired proprioception (somatosensation). The sense of touch is an important contributor to balance and orientation. The stretch receptors in the legs, the finger tips, and the soles of the feet, all provide feedback for balance.

6. Cerebellar dysfunction. The integration of vestibular, visual and proprioceptive information takes place in the cerebellum. Cerebellar dysfunction can result in slow or inappropriate reaction to self movement or external visual stimuli.

7. Impaired vision. Vision plays an important role in balance, and patients with visual deficits have greater risk for falls. Visual problems associated with decreased postural stability include: visual acuity less than 20/50, asymmetric vision impairing binocular vision and depth perception, slow pupillary reaction causing increased adaptation time when going from a lighted to a dark room and vice versa, impaired peripheral vision.

8. Depression. Depressed patients may be more internally (therefore less externally) aware. The use of antidepressants and anxiolytics increases the risk of falling.

9. Impaired cognition. Patients with impaired cognition may be less aware of their surroundings or more likely to engage in risky activities.

10. Impaired reaction time. Many fall avoidance strategies are dependent on reaction time when postural stability is challenged. Slower reaction time may increase the risk of falls when the patient's limits of stability are exceeded.

Management Options:

Typically, there is no magic bullet or single intervention that will make a significant impact. Identified risk factors may be treated, medications may be reviewed and adjusted, balance retraining therapy can improve postural and gait stability, living quarters can be modified to reduce exposure to fall hazards, and the patient can be educated to avoid fall risk situations.

MULTIFACTORIAL DYSEQUILIBRIUM

Poor balance and instability in the elderly have been described as a geriatric syndrome because the specific cause of these complaints is often not obvious to the examiner. This is primarily because poor balance in the elderly is most often multifactorial, with no single clinical abnormality responsible. The balance system has built-in redundancy. This means that there are fallback systems in place if one system fails. For example, when walking in the dark, you may feel more stable if you can run your fingers along the wall, increasing tactile feedback. In this situation, your sense of touch is substituting for your loss of visual information. The basis of multifactorial dysequilibrium is that when one system fails, the backup system also fails and you lose your balance. Over 50 percent of those who fall have four or five risk factors. The risk factors for increased likelihood of falling have been identified, and intervention for these risk factors can significantly reduce the risk of falling. Obviously, intervention cannot eliminate the possibility of an injurious fall, but research indicates that systematic evaluation and intervention can dramatically reduce the likelihood of a fall.

Common Risk Factors for Falling

- 1. Vestibular (inner ear) disorders.** Vestibular disorders can cause dizziness, vertigo or loss of balance with certain movements and in certain environments.
- 2. The use of four or more prescription medications.** When using four or more prescription medications, the chance of an adverse reaction increases, and the ability to predict interactions between medications decreases.
- 3. The use of anti-depressant or anti-anxiety medications.** These medications can affect reaction time, which is crucial when faced with a potential fall.



4. A drop in blood pressure when standing.

Some medical conditions such as diabetes and many medications used to treat high blood pressure, irregular heart rate and bladder problems can cause a sudden drop in blood pressure when standing. This causes temporary lightheadedness and loss of balance.

5. Weakness or numbness in the feet or lower legs. Numbness, tingling, or feet that feel unusually hot or cold may be a sign of decreased nerve sensation in the feet and legs, a condition known as peripheral neuropathy.

6. Degeneration of the cerebellum. The cerebellum is the part of the brain mostly responsible for balance and coordination. Stroke, medications or aging can compromise the efficiency of the cerebellum, and therefore, the efficiency of the balance system in general.

7. Visual disorders. Vision is an important contributor to balance, so any visual compromise can increase the risk of falling.

8. Depression. The use of anti-depressants increases the risk of falling.

9. Impaired cognition. Lack of awareness of surroundings can increase risk for falling.

Comprehensive assessment for risk of falling may require examination by more than one specialist. Your primary care doctor should coordinate any assessment or treatment plan.

CENTRAL DIZZINESS

(aka Vertebro-Basilar Insufficiency, Cerebellar Dysfunction)

Typical Complaints:

"I just can't walk."

"I can't put my feet where I want them to go."

"I feel like anytime I lose my balance, I am going to fall. I react too slowly."

"I stagger when I walk."

"I have this constant feeling in my head."

Defining Questions:

- Were you completely coherent during the episode? (Vestibular vertigo does not cause confusion or memory loss.)
- Are you more unsteady when in a group of people? (Patients with decreased efficiency of the cerebellum have more difficulty ignoring visual stimulation.)

Medical History Questions:

- Do you have a history of vascular disease? (Central dizziness is more common in patients with atherosclerosis, coronary or peripheral artery disease, hypertension, hyperlipidemia or a history of smoking.)

Diagnosis/Definition

Dizziness, vertigo or dysequilibrium arising from abnormalities at or medial to the vestibular nuclei are often termed central dizziness, while symptoms occurring as a result of labyrinthine or auditory/vestibular nerve pathology are frequently termed peripheral dizziness.

Vertebro-basilar insufficiency is characterized by chronically poor or acutely disrupted blood flow to the brainstem and cerebellum. Symptoms of chronically poor blood flow, to some degree, mimic those of cerebellar degeneration. Patients may exhibit an ataxic gait, poor coordination, and decrease in efficient reaction to challenging balance situations.

Symptoms of acutely disrupted blood flow may include sudden onset vertigo, ataxia, double vision or loss of vision, slurred speech, dysphagia, confusion and numbness. Acute vertebro-basilar insufficiency can be transient (TIA) or permanent (CVA) as a result of blockage of the vertebral, basilar or cerebellar arteries. Screening tests for central dizziness include tests of cerebellar function (past pointing, finger to nose, rapid alternating hand movements, and heel-to-toe walking), as well as standard cranial nerve exam and testing of reflexes for possible stroke.

Management Options:

The American College of Radiology recommends cranial MRI (with and without contrast) as the best imaging study for the complaint of dizziness. Neurologic exam may identify or rule out brainstem pathology. The HINTS exam described on pages 8 and 9 is more sensitive than imaging in the first 48 hours.

CENTRAL DIZZINESS

Good balance and stability require the efficient interaction of information provided by the eyes (visual), the inner ears (vestibular) and the muscles and joints (proprioception) every time we move. All this information converges in a part of the brain called the cerebellum. The visual and proprioceptive information is constantly changing as a function of movement, but the vestibular reference—which is gravity—is unchanging.

Since gravity is constant, humans tend to rely more on vestibular information for maintenance of dynamic balance. When the peripheral vestibular system is damaged, this may change. As long as the information arriving from the three sources is predictable and non-conflicting, equilibrium is maintained, and there is little thought regarding balance.

The cerebellum is responsible for responding quickly and effectively if any of the information received from the sensory inputs is considered conflicting. A sensory conflict might occur when a person is sitting in a car, stopped at a light, and a large truck next to the person moves suddenly. Frequently, the person will experience a sensation of movement and step on the brake and grip the wheel. In this particular situation, vestibular and proprioceptive input did not detect movement, but the visual input detected motion. The result is a fleeting, inaccurate illusion of movement, or loss of balance. If there is any deficiency in the cerebellum, those fleeting symptoms may be of longer duration and occur in more situations.

Central dizziness (also known as vertebro-basilar Insufficiency, or cerebellar dysfunction) is related to dysfunction of the cerebellum or brainstem.



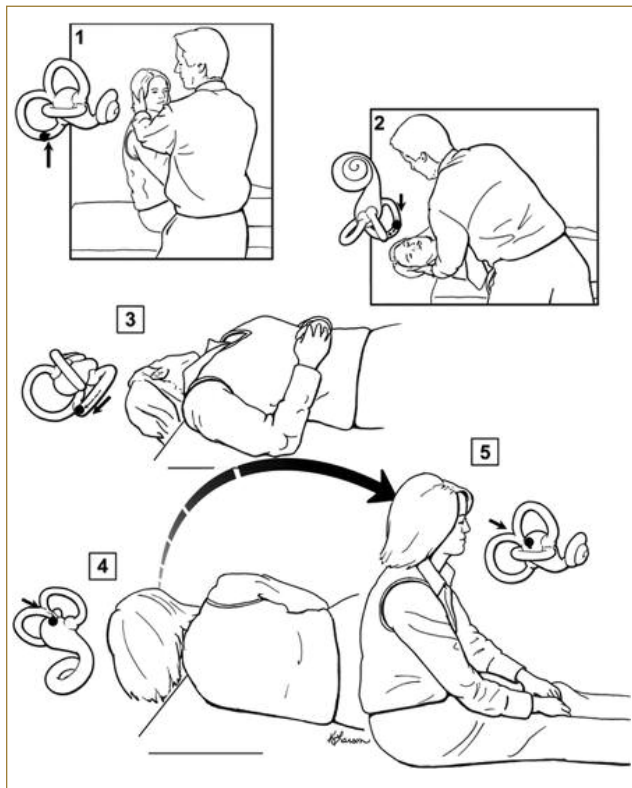
This may be due to vascular problems such as vertebro-basilar insufficiency or other factors such as medications. Vertebro-basilar insufficiency may result from arterial blockages such as atherosclerosis (hardening of the arteries) that can lead to decreased blood flow to the brain causing problems such as transient ischemic attacks (mini-strokes) or strokes.

Central dizziness is the most difficult type of dizziness to treat as most balance therapy is designed to train the brain to use balance-related information more effectively. In central dizziness, the brain is simply less trainable. Balance therapy does help, but the benefits are less predictable. Depending on the extent of dysfunction, modifications to your living environment to reduce the risk of falling may be recommended. Medical intervention may focus on medications, procedures or lifestyle changes to reduce the risk of ischemia.

CANALITH REPOSITIONING (CRP)

Canalith repositioning (CRP) has been proven safe and effective for the treatment of BPPV. BPPV occurs in the posterior semi-circular canal about 90 percent of the time. The CRP is a modification (evolution really) of the Epley maneuver described by John Epley, MD, in the early 90s. The Epley Maneuver was intended specifically for posterior canal BPPV. There are CRP techniques suggested for both the horizontal and anterior canals.

CRP is best performed immediately following a positive Dix-Hallpike test. In fact, the first step of CRP is the Dix-Hallpike test.



CRP for Right Posterior Canal BPPV

Dix-Hallpike test The patient is seated on the exam table, head turned towards the test ear (step 1). The patient is then lowered to a supine head hanging position beyond the edge of the exam table. This position should be maintained for one minute (step 2). The examiner should observe for nystagmus. Both figures on this page are for right-sided posterior canal BPPV.

If the Dix-Hallpike is positive for posterior canal BPPV (typical transient upbeat/rotary nystagmus), CRP can be completed. After a period of 30 to 60 seconds (or until the transient nystagmus abates), the patient's head only is rolled to the contralateral side (step 3). The head must not be lifted during the transition from the step 2 to the step 3 position.

After 30 to 60 seconds, the patient is rolled on to the side opposite the affected ear with the nose pointing directly to the floor if possible (step 4). After 30 to 60 seconds, the patient may be raised up to a sitting position (step 5).

CRP Suggestions:

- Explain the purpose and procedure before starting.
- Inquire about neck/back problems before starting.
- Never take your hands off the patient's head.
- Multiple CRP's on the same day increases success rate.

CRP Cautions:

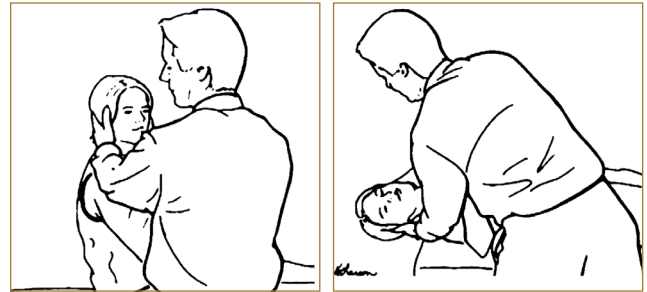
- View eyes for change in direction/pattern of nystagmus. Otoconia may reverse course in the posterior canal or enter the horizontal canal.
- Hold patient firmly on rising in step 5 as they may experience a brief, intense falling sensation.

CANALITH REPOSITIONING (CRP)

Canalith repositioning (CRP) is an effective, simple treatment for benign paroxysmal positional vertigo (BPPV) of the posterior canal. The diagnosis of BPPV is made by inducing an episode of vertigo during examination. This is typically accomplished by performing the Dix-Hallpike maneuver, where you are moved quickly from the sitting to supine (lying down) position with your head slightly extended beyond the end of the examination chair or table. If this movement triggers vertigo, by looking at your eye movements, the examiner can determine: 1) if the vertigo is a result of BPPV, and 2) where in the inner ear the offending otoconia debris is located. Once this has been accomplished, treatment can begin.

When the examiner knows where the otoconia debris is located, it is simply a matter of using gravity to our advantage to move the debris from its present vertigo-inducing location to a part of the inner ear where the debris is harmlessly dissolved. This is accomplished through a series of specific, timed head movements, based on the examiner's ability to predict the angles of the affected semi-circular canal. The examiner may view your eyes throughout the procedure, as certain patterns of eye movement help determine whether the debris is being moved to the desired location.

The most commonly used form of CRP begins with the Dix-Hallpike maneuver (see above). If this triggers a period of vertigo, the examiner will wait for the vertigo to subside (typically less than one minute). Rather than sit you back up and causing



the debris to go back into the affected canal, the examiner will roll your head to the opposite side. Rarely, you might experience brief vertigo in this position. The next step is to roll you onto your side (the side opposite the affected ear), with the goal of getting your nose pointing directly toward the floor. Frequently, this position will induce some brief vertigo. Finally, the examiner will bring you back to a sitting position. It is this fourth and final position when the otoconia debris typically come out of the affected canal and fall back into the vestibule. Sometimes, this can cause brief but intense dizziness as the debris settles into the vestibule. This only lasts a few seconds, and the examiner will make sure you are safe by holding you firmly.

You may feel unsteady or mildly nauseous after the treatment. This usually passes quickly, but you may wish to have someone drive you home. There are no specific post-treatment restrictions that have been proven to affect the outcome of this treatment, but we suggest avoiding lying flat on your back for a few hours after treatment to reduce the possibility of the debris re-entering the semi-circular canals.

VESTIBULAR AND BALANCE EXERCISES

Instructions to give patient.

VOR (Vestibular Ocular Reflex) Exercises

The goal of these exercises is to enhance the communication between your inner ears and your eyes. The brain uses the inner ear information to determine how much eye movement is needed to allow your eye to stay fixed on an object as you move around. If you move fast enough that the brain perceives difficulty staying visually fixed and focused, the brain may increase the signal from the inner ear to adjust. Over time you will be able to move faster and still maintain visual stability. Make sure you mix up the speed and direction of head movements, because that will best simulate real life situations. These exercises help you recover as quickly and as much as possible from most inner ear disorders. When you finish an exercise session, you should feel a little uneasy. That means you have pushed the system sufficiently to trigger progress. If you feel perfectly fine, you are probably moving too slowly. If you feel very nauseous, you are probably moving too fast.

Balance Exercises*

Ankle sways provide feedback from the muscles, joints and nerves in the lower legs, while at the same time, the eyes and the inner ears are responding. By performing ankle sways, you:

1. Find your limits of stability (LOS)—how far you can lean before needing to step or touch something
2. Increase your LOS by strengthening the lower legs
3. Flood the brain with information from all senses related to balance

Walking with knees touching hands forces you to shift your weight. By bringing each leg up to waist height, you are on one leg long enough that you must shift your weight or you will lose balance. By doing this repetitively, your body learns when and how to shift your weight efficiently.*

Both of these exercises can be modified to work a specific balance input. By closing your eyes, your brain is forced to make better use of vestibular and tactile information. By standing on a folded towel, your brain is forced to use visual and vestibular information more efficiently. Sometimes after a vestibular injury, the goal is to teach your brain that there is still residual useful vestibular information. To force the brain to use that vestibular information, you may need to reduce dependence on visual and tactile information.

Ball toss is simply an exercise to increase your reaction time and keep you moving. It enhances your hand-head-eye coordination.

Summary

By improving your reaction time, teaching you how to shift your weight efficiently, increasing your limits of stability, and having faster warning when you might fall, you should be more stable and safe when walking or faced with challenging balance situations.

* Patients with multi-factorial dysequilibrium or with postural or gait instability not associated with a pure vestibular disorder should not attempt these balance exercises at home. They may require supervised therapy working with a physical therapist.

VESTIBULAR AND BALANCE EXERCISES

Name _____ Date _____

VESTIBULAR OCULAR REFLEX (VOR) EXERCISES

Sitting Exercises

1. (x1 viewing) Take an item to read, hold it in front of you, move your head back and forth, up and down, around in circles both directions, as fast as you can and still be able to read the words on the page. Do each direction for about one minute.

2. (x2 viewing) Perform exercise #1, but instead of holding the reading material stationary, move it up and down and side to side, opposite and along with head movement.

You should spend about five minutes, two to three times a day performing the above exercises.

BALANCE EXERCISES

Standing Exercises

1. Ankle sways. Stand next to something you can hold to steady yourself or have someone assist you. Sway side-to-side and front-to-back by swaying with your ankles. Do this with everything stiff except your ankles (knees locked, hips locked). After a few days, progress to swaying in circles, both directions.

2. Walking. Start walking in place. Once you are comfortable (typically after a few days) put your hands up and bring your knees to your hands. Gradually raise the height of your hands to waist height.

3. Ball toss. Take a tennis ball and bounce it and toss it around.

Each exercise should be performed non-stop for about one minute. Exercises 1 and 2: start in front of a mirror. After a couple of weeks do them in front of a wall. When comfortable, try with your eyes closed. This forces you to reduce your dependence on vision for balance.

Complete 2–3 times a day. Each exercise should be performed for about one minute.

Be sure to have someone assist to steady you in case you lose your balance.

BLOOD PRESSURE (BP) LOG

Name _____ Start Date _____

This log allows you to discuss with your physician any noted changes in blood pressure over time and in different positions. You should check your blood pressure as described below, over several days, at different times during the day (for example, before lunch one day, after lunch the next – before you take your medications one day, after taking medications the next, etc.)

BP Supine: Lay back on a bed or recliner chair with head back, feet elevated, for at least five minutes. Check your blood pressure BEFORE sitting or standing up.

BP Standing ASAP: Stand quickly (have someone to help stabilize you, if needed), and check your blood pressure as soon as possible upon standing.

BP Standing One and Three Minutes: Stay standing for one to three minutes. Check your blood pressure after one and three minutes. Record the numbers below.

DAY	TIME	BP SUPINE	BP STANDING ASAP	BP STANDING ONE MINUTE	BP STANDING THREE MINUTES
_____	_____	____/____	____/____	____/____	____/____
_____	_____	____/____	____/____	____/____	____/____
_____	_____	____/____	____/____	____/____	____/____
_____	_____	____/____	____/____	____/____	____/____
_____	_____	____/____	____/____	____/____	____/____
_____	_____	____/____	____/____	____/____	____/____
_____	_____	____/____	____/____	____/____	____/____
_____	_____	____/____	____/____	____/____	____/____
_____	_____	____/____	____/____	____/____	____/____
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_____	_____	____/____	____/____	____/____	____/____
_____	_____	____/____	____/____	____/____	____/____
_____	_____	____/____	____/____	____/____	____/____
_____	_____	____/____	____/____	____/____	____/____

TYPICAL COMPLAINTS OF VARIOUS VESTIBULAR AND NON-VESTIBULAR DISORDERS

Benign Paroxysmal Positional Vertigo (BPPV)

PAGES 18–19

"I woke up and the bed was spinning."

"I bent over (rolled over, turned quickly, laid down, sat up from bed, etc.) and everything started spinning around."

"Every time I tried to get up I fell back on to the bed."

Vestibular Neuritis/Labyrinthitis

PAGES 20–21

"I had constant spinning and nausea for about three days."

"After the worst of it (vertigo and nausea) I was okay if I didn't move. If I moved I was off balance and would get nauseous if I moved too much."

Meniere's Disease

PAGES 22–23

"I have had several episodes of severe vertigo with nausea, lasting for hours at a time."

"I feel so much pressure in my head (ear)."

"My ear was roaring."

"After an episode, I need to sleep for several hours."

Vestibular Migraine

PAGES 24–25

"I have episodes of spinning and nausea that come on without warning, but had no ear symptoms."

"I felt a sudden wave come over me, like the floor shifted."

"I can't stand any type of motion. It never bothered me when I was a kid."

Superior Canal Dehiscence

PAGES 26–27

"I get dizzy around loud noise or when I blow my nose."

"My voice sounds like I am in a barrel."

"I hear my pulse or chewing louder than I should."

Anxiety/Hyperventilation

PAGES 28–29

"I felt like my heart was pounding out of my chest."

"I felt like I was dying."

"I couldn't breathe."

Orthostatic Hypotension

PAGES 30–31

"I get dizzy and off balance when I stand up."

"I get up and start to walk and feel like I am going to fall over."

"When I get up quickly, I feel like I could faint."

Multifactorial Dysequilibrium

PAGES 32–33

"I have had several falls."

"I feel very unsteady when I am in a crowd or unfamiliar setting."

"I want to hold on to someone/something when I walk."

Central Dizziness

PAGES 34–35

"I stagger when I walk."

"If I lose my balance, I just go over."

"I have this constant feeling in my head."

**HOW TO MAKE AN APPOINTMENT WITH THE
WAKE FOREST BAPTIST HEALTH BALANCE DISORDERS TEAM**

Physician referrals can be made through the STAR line at **336-713-STAR (7827)**.



Otolaryngology
Medical Center Boulevard
Winston-Salem, NC 27157