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Review

Otogenic versus neurogenic dizziness: The neurotology perspective

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ABSTRACT

Symptoms of dizziness are very common, and they can be quite frustrating and debilitating. As the underlying presentation and etiology can be quite variable from patient to patient, deciphering the most appropriate evaluation steps can be a rather daunting task even for the experienced physician. The primary objectives of this review are to 1) present a relatively concise yet comprehensive overview of the multitude of signs, symptoms, and causes of different forms of dizziness, and 2) provide type-specific clinical and diagnostic guidelines for the evaluation and treatment of this complex condition. The diagnostic utility of various evaluation techniques and procedures, including audiometry, electronystagmography (ENG), vestibular evoked myogenic potential (VEMP), electrocochleography (ECOG), rotary chair testing, posturography, blood analyses, and radiographic imaging are reviewed for the workup of otogenic vertigo. The importance of professional referral to the appropriate medical subspecialist for the workup of neurogenic and cardiogenic dizziness is also discussed. Evaluation and treatment algorithms are integrated into the narrative to facilitate all discussions and for future reference.

KEYWORDS: dizziness, vertigo, disequilibrium, dysequilibrium, lightheadedness, presyncope, vestibular therapy

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1. Introduction

As many as 30% of adults over the age of 65 years experience persistent or intermittent symptoms of dizziness that require further investigation [1]. Children and younger adults do not suffer from this disorder nearly as often. In most cases the symptoms of dizziness diminish over time. This fact is largely the result of accommodative or compensatory interactions between the brain, skin, muscles, joints, vestibulo-ocular, and vestibulo-spinal reflex circuitry. In general, dizziness occurs as a result of conflicting signals between these normally synergistic central and peripheral balance sensing components and pathways. Of these, perhaps the two most important for bodily balance control and positional orientation are the visual and vestibular mechanisms. Vision provides feedback about body position and motion in relationship to all physical surroundings, without which balance stability would substantially suffer. The semicircular canals and neighboring saccule and utricle form the labyrinth or vestibular apparatus of the inner ear. These bilateral structures contain specialized cells that are sensitive to bodily motion and position alterations. Malfunction of this interconnected system can send false signals to the brain that motion has been detected when in fact no such action has been initiated by the patient. If such pseudo signals persist and conflict with other neurologic inputs from the visual and peripheral sensory balance pathways, symptoms of dizziness may occur. The purpose of this tutorial review is to provide a succinct overview

of various populations who initially present to otolaryngologists, neurologists, or primary care physicians for evaluation and treatment of dizziness. The signs, symptoms, etiologies, and testing procedures for this multivaried disorder will be discussed and assembled in the form of clinically practical charts and algorithms. Although alternative treatments for these patient subtypes will be addressed in a forthcoming companion tutorial, in this paper a chart overview of management options will be presented for completeness.

For simplification purposes, there are 3 primary conditions that have been classified as forms of dizziness: 1) vertigo, 2) lightheadedness, and 3) imbalance or disequilibrium. These descriptive terms require differential definitions inasmuch as they each represent a unique set of specific signs and symptoms with multivaried etiologies and treatments. In contradistinction to sensations of true dizziness, erroneous feelings of intermittent dizziness are commonly reported by patients with chronic states of mental and physical malaise secondary to anxiety or depression, fatigue syndrome, and fibromyalgia [2, 3]. Because dizziness can be representative of a serious medical problem and because it can lead to injuries secondary to falls or syncope, physicians who treat patients with complaints of dizziness must understand the inherent complexity and risks of this condition. Older patients (>65 years) who generically complain of feeling dizzy often present with complicated medical histories and multiple comorbidities [4, 5]. It is often difficult for them to describe precisely their disturbing sensations. When this happens, the examining physician should prompt a focused description of the problem by offering them dizziness classification options, as described below. Familiarity with such nomenclature may enable the physician to develop a differential diagnosis and appreciate when it is necessary to refer the patient to the appropriate medical subspecialist for diagnostic testing and treatment. This referral should be based on whether the most likely etiology is central (neurogenic) or peripheral (otogenic or proprioceptive) in origin. However, distinguishing between the two is not always straightforward and many patients have overlapping symptoms of both subtypes.

1.1. Vertigo

This term should be applied when the patient reports an illusion of motion when there is no actual motion occurring at the time of such sensations. Some patients complain that they themselves are actually spinning, tilting, or swaying around uncontrollably. Others report that they feel as if they are being pulled in one direction or another and that the environment itself seems like it is whirling. Patients experience associated nausea and vomiting typically with acute presentations, which are exacerbated by moving the position of the head [6, 7]. These symptoms may occur as a single short-lived event, or they may be episodic or chronic, depending upon the underlying cause. Clinically significant vertigo is more commonly a peripheral or otogenic phenomenon due to an inner ear abnormality, and may variably occur in patients with benign paroxysmal positional vertigo (BPPV), vestibular neuronitis, labyrinthitis, or Meniere's disease. Less commonly, vertigo may result from inner ear viral pathogens, direct ear trauma, cholesteatoma, superior semicircular canal dehiscence, traumatic brain injury, migraine headaches, cerebrovascular accident (CVA), vertebrobasilar insufficiency, benign or metastatic neoplasms involving the oculovestibular balancing pathways, Chiari malformation, ototoxic pharmacologic sequelae, and in patients with histories of abusive alcohol consumption or hallucinogenic drug usage [6]. Vertigo is often self-limiting owing to its natural history and a multiplicity of compensatory mechanisms. Restoration of peripheral vestibular function commonly occurs due to the natural recovery of a high proportion of primary lesions, such as BPPV and vestibular neuronitis [8]. Central and peripheral adaptation processes allow for the restoration of static balance of vestibular tone and dynamic recovery of vestibulo-ocular reflex (VOR) gain through the plasticity of brainstem and cerebellar pathways [9, 10, 11]. When this proves not to be the case, such as with coexisting limb weakness, speech disturbances, or visual acuity changes, immediate medical attention is necessary for differential diagnosis and type-specific treatments. Figure 1 is an algorithm demonstrating the interrelated and distinct characteristics of vertigo and its corresponding correlates.

1.2. Lightheadedness

This condition is also referred to as near syncope or presyncope. Patients who are lightheaded often complain of a sense of fainting (vasovagal syncope)

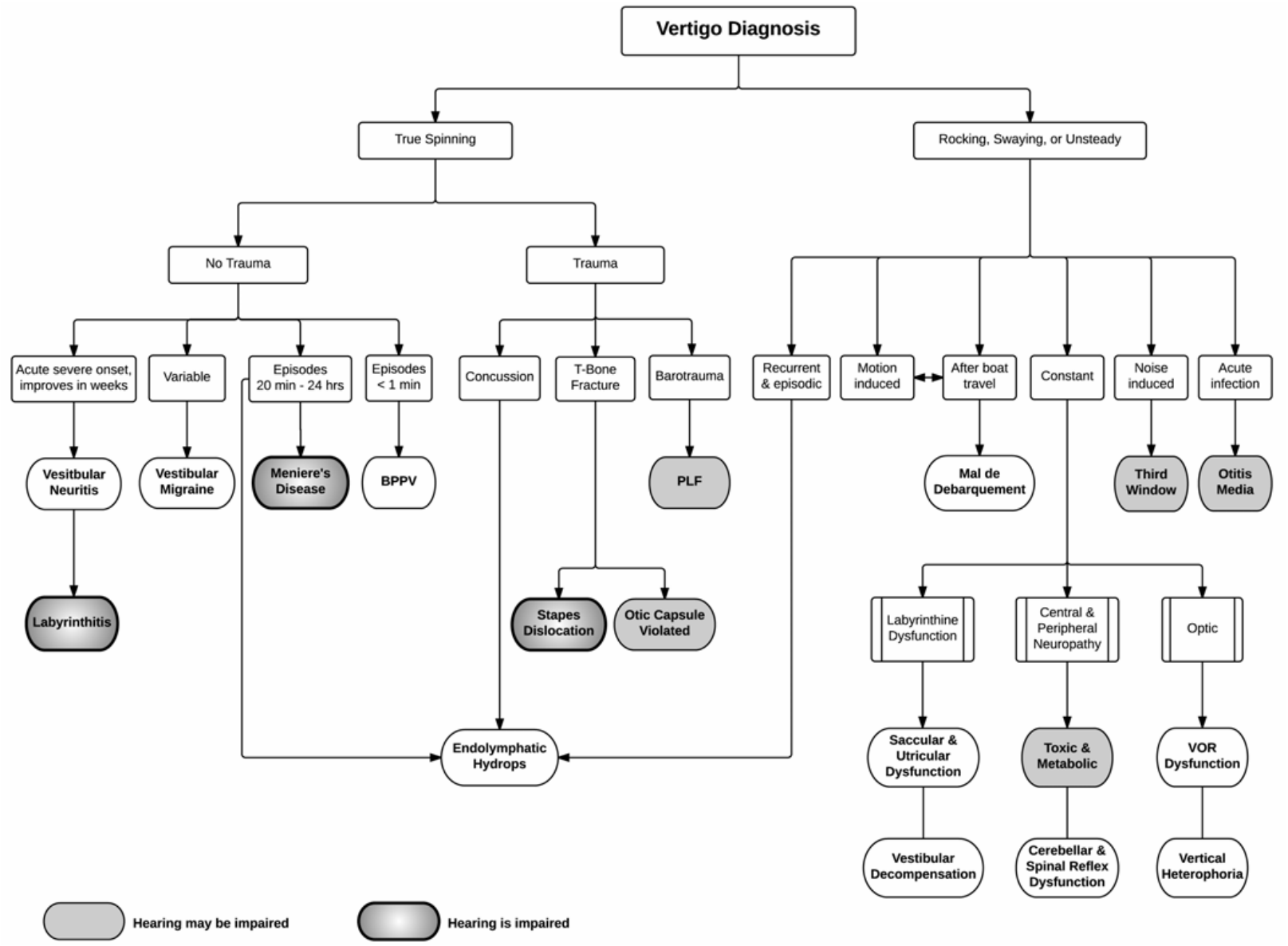

Figure 1. Vertigo diagnosis. Key: BPPV (benign paroxysmal positional vertigo), PLF (perilymphatic fistula), T-Bone (temporal bone), VOR (vestibulo-ocular reflex).

or passing out. Not infrequently these disabling sensations are triggered when the patient stands up too quickly or breaths deeply and rapidly for at least one minute (hyperventilation). Lightheaded patients may have a feeling of dizziness, but they do not usually report that they or their surroundings seem to be moving or whirling. Symptoms often improve when the patient lays down on a flat surface. Brief episodes are not uncommon and are typically not symbolic of a serious medical problem. Rather, the condition is usually caused by a momentary drop in blood pressure and transitory impairment of adequate blood flow to the brain known as orthostatic hypotension. Factors such as 1) high body temperature secondary to an influenza viral infection, 2) an illness that results in dehydration, 3) emotional anxiety and stress, 4) heavy menstruation, 5) hyperventilation, 6) anemia, 7) hypertension, 8) hypoglycemia, 9) excessive alcohol consumption, and 10) use of certain blood pressure or antidepressant medications are common causes of lightheadedness. More serious causes include transient ischemic attack, CVA, atrial fibrillation (or other cardiac arrhythmia), excessive bleeding, and congestive heart failure. Whereas some of these causes require prompt medical evaluation and management, others do not because they are often self-limiting.

1.3. Disequilibrium

Occurring frequently in the elderly population, this form of dizziness is usually the result of multi-level sensory acuity degeneration. In many cases imbalance sensations are directly attributable to diminished joint sensitivity of the lower limbs and concurrent sensory degradation of the vestibulocerebellar pathways. If vision is blocked, the sensation of disequilibrium worsens. Of diagnostic significance is the fact that such symptoms improve almost immediately when the patient holds onto a stationary object for balance via support of the upper limb sensory control system; a cane or walker is also quite helpful. Neurogenic explanations may include motor control disturbances, secondary to abnormalities in limb-trunk muscle tone, strength, range and speed of motion, and coordination. Patients with histories of cerebellar pathology, 4th ventricle lesions, Parkinson's disease, cervical spondylosis, peripheral neuropathies, long tract degeneration or lesions, or diabetes commonly present with vague signs and symptoms of disequilibrium or imbalance due to deconditioning [8]. These factors explain the need to include both sensory and motor testing when evaluating patients with this chief complaint.

Figure 2 provides a pictorial synopsis of the various types of dizziness and their possible causes, as discussed above.

2. Testing options: Evaluating "dizzy" patients

Obtaining an accurate and detailed description of the dizziness complaint is critical to the differential diagnosis. It is vital to appraise the degree to which this condition has adversely impacted the patient's personal and vocational quality of life. As always, the history interviewing process should include a comprehensive review of the present illness, all systems, past medical and surgical histories, results of head imaging studies, any audiometric test findings including dizziness assessments, social history, family history, all current medications, and drug allergies.

2.1. Onset and triggers

The examiner should ask the patient to describe when the first episode occurred, exactly what was experienced at that time, and the duration of the episode. It is important to ascertain whether the patient experienced vertigo, lightheadedness, or disequilibrium symptoms, as described earlier. If the dizziness complaint is due to an inner ear condition the patient will often describe a sudden onset with vivid recall of the factors leading up to the event. For example, the patient may report that when awakening from sleep he or she often experiences a spinning sensation when turning the head; this feeling weakens rather quickly when the head is held still. This description should strongly influence the examiner to consider BPPV as the underlying cause. However, if quick resolution of this sensation is not reported by the patient and in fact the spinning continues for some time despite head adjustments, then the examiner should consider labyrinthitis/vestibular neuronitis or a form of endolymphatic hydrops for an explanation given a congruent history. If the patient reports that the dizziness symptoms usually occur during weight lifting or straining activities, then the examiner should consider superior canal dehiscence or another third window condition as the cause of the complaint. In contrast, if the patient describes dizziness when

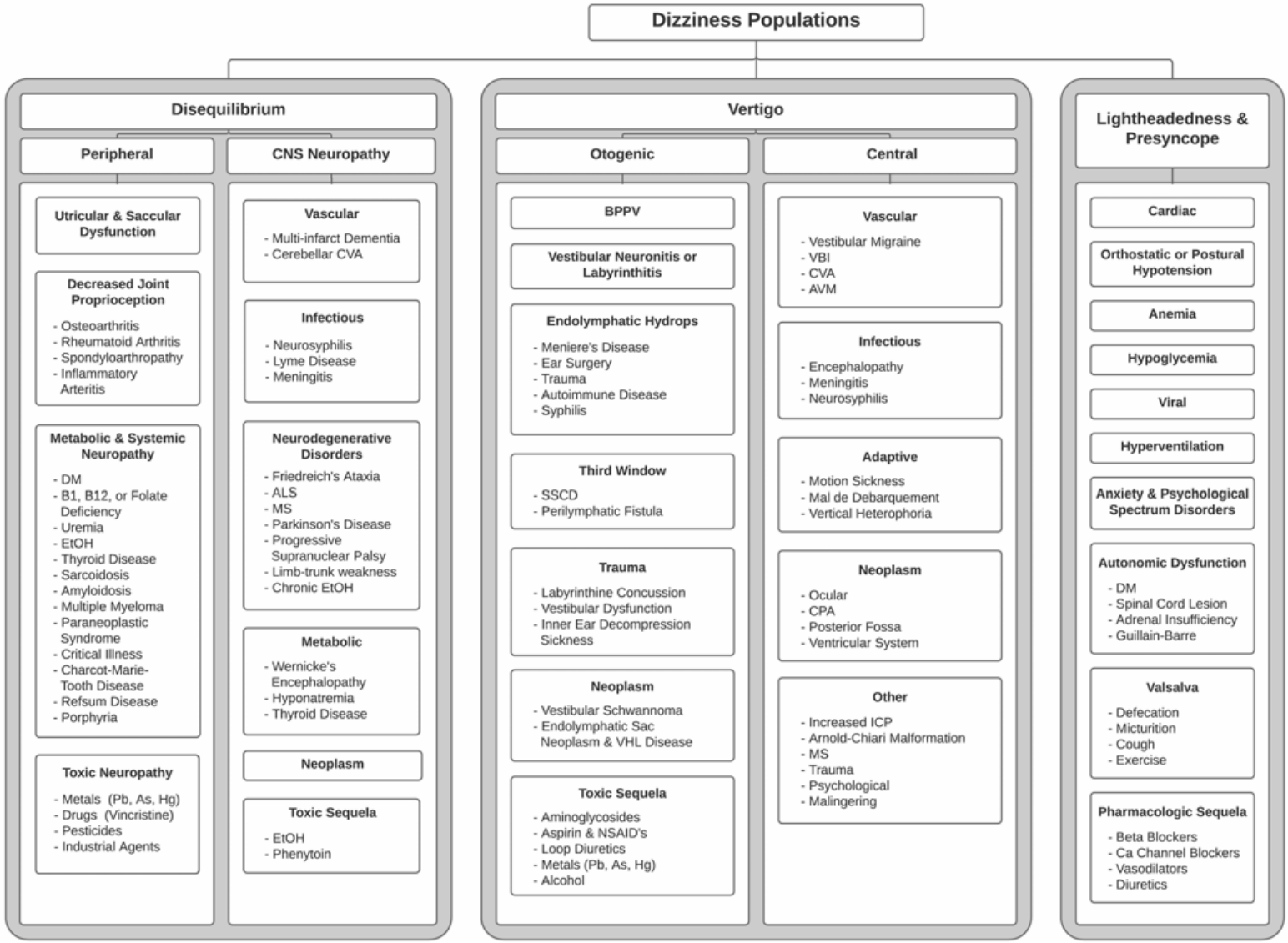

Figure 2. Dizziness populations. Key: As (arsenic), ALS (amyotrophic lateral sclerosis), AVM (arteriovenous malformation), BPPV (benign paroxysmal positional vertigo), CPA (cerebellopontine angle), CVA (cerebrovascular accident), DM (diabetes mellitus), ETOH (alcohol), Hg (mercury), ICP (intracranial pressure), MS (multiple sclerosis), Pb (lead), SSCD (superior semicircular canal dehiscence), VBI (vertebrobasilar insufficiency), VHL (Von Hippel-Lindau).

bending over or when the head is held in an upward looking direction, as may occur during star gazing, the examiner should consider BPPV or vertebrobasilar insufficiency as the cause. Figure 2 illustrates that many metabolic conditions, systemic and toxic neuropathies, and brainstem or cerebellar CVAs often contribute to chronic symptoms of dizziness. In such cases, patients do not always recall a specific onset event. As important to the differential diagnosis is the fact that, in addition to contributing to long-lasting dizziness symptoms, these underlying etiologies can cause progressive worsening of the problem over time. If dizziness onset is induced when the patient rolls out of bed, the examiner should determine to which side the patient normally rolls out of bed. The answer to this question can help establish the ear of origin and may help differentiate positional vertigo from other subtypes of dizziness less directly affected by positional variations.

2.2. Duration and side effects

It is important to inquire about whether additional dizziness spells have occurred since the first episode and the general duration of each event; that is, are they typically episodic and non-predictable in their occurrence or chronic? The time frame of the experience can be of extreme diagnostic significance. Was it brief, as is usually the case with BPPV, or did it continue for hours as may occur with Meniere's disease, or even days as with labyrinthitis? The patient should be instructed to describe any associated symptoms, such as nausea and vomiting, photosensitivity, or headache. Otologic symptoms, including pressure or fullness in the ears, reduced hearing acuity, new-onset tinnitus, or an increase in the intensity of tinnitus should be explored with the patient, because these complaints often are suggestive of a causally related inner ear pathology. For definitive diagnosis and comprehensive treatment considerations, it is important to determine laterality of these symptoms; do they occur primarily on the right side, left side, or both? Ultimately, the patient's premorbid medical history often facilitates the decision about the types of evaluation techniques that should be employed and the preliminary diagnosis. Such factors as a preceding illness history or vascular impairment, an antecedent ear infection, a recent head injury, or an ear trauma should be comprehensively explored with the patient. Neurological symptoms should then be explored, such as change in visual acuity, facial weakness or paralysis, limb weakness, limb numbness, gait incoordination, or speech and language difficulties. These types of problems often point to central nervous system explanations for the dizziness complaints. If the reported dizziness was not an isolated episode, the next step is to characterize the frequency of episodes and determine any aggravating or alleviating factors, such as visual obstruction or darkness, certain dietary triggers (sodium and caffeine intake), abrupt bodily activity or head position change, rising suddenly from a seated or supine position, and bending down suddenly. A personal or family history of Meniere's disease should be delineated, as there can be a familial tendency with this condition in up to 20% of patients [12]. A description of visual aura, photosensitivity, or a headache during or separate from the dizzy episode can support the presence of a vestibular migraine component. A life-long history of motion sensitivity should also raise the concern for a migraine variant. Previous medical treatment for dizziness should be documented along with the outcomes. Finally, it is important to note if the dizziness has ever responded positively to the use of corticosteroids. If the answer to this inquiry is affirmative, this information may prove helpful in the final analysis of various treatment options.

2.3. The physical examination

After obtaining a detailed history, a thorough head and neck physical exam, including neurological and vestibular clinical examinations, should be performed. The external auditory canals and tympanic membranes should be evaluated under otomicroscopy. Signs of pathology should be recorded, including otitis externa, cerumen impaction, perforation, otitis media with or without effusion, ossicular chain abnormalities, or cholesteatoma. Pneumatic otoscopy should be performed to evaluate the compliance of the tympanic membranes, abnormal presence of middle ear fluid, and overall middle ear aeration and functioning of the Eustachian tubes in response to induced intra-aural pressure variations. If available, Frenzel goggles should be used to evaluate for peripheral nystagmus. By magnifying the patient's eyes and removing visual fixation, clinical vestibular testing can be done with greater detection, such as fistula testing when

pressure is introduced to the ear canal and translated to the middle ear space. Nystagmus and vertigo are positive findings and should alert the examiner to the possibility of a semicircular canal dehiscence or perilymphatic fistula. It should be noted whether the patient exhibits spontaneous or gaze-evoked nystagmus with Frenzel goggles. This should be followed by an examination for post head shake nystagmus and catch-up saccades with head thrust. The Dix-Hallpike maneuver may then be performed to evaluate for benign paroxysmal positional vertigo. The patient should then be asked to stand for a variety of stationary and active tests. Gait with ambulation may be examined for ataxia. Romberg testing may be done to assess proprioceptive function reflective of posterior spinocerebellar tract integrity, and pronator drift will provide information regarding general cerebellar and upper motor neuron functioning. The Fukuda Step Test may further identify a unilateral vestibular deficit or asymmetry; drift greater than 45 degrees from midline after 30 seconds of marching in place with eyes closed is often indicative of pathology of the ipsilateral labyrinth [13]. Specific cerebellar testing may also include appraisal of alternating finger-to-nose and finger-to-finger manual gestures to identify past-pointing phenomena (dysmetria), and rapid, rhythmic, and repetitive hand tapping tasks to reveal dysdiadochokinesia.

3. Formal laboratory testing alternatives

3.1. Audiometry

Standard air and bone conduction audiometric testing with speech audiometry, including reception thresholds and discrimination scores, should be routinely performed on all dizzy patients to decipher hearing status and any corollary conductive or sensorineural auditory system impairment. Results may help corroborate the differential diagnosis of the dizziness complaint. Patients with classic Meniere's disease often present with a low frequency sensorineural hearing loss. Those with acoustic neuromas not infrequently suffer from high frequency sensorineural hearing loss or rollover word discrimination score patterns classic of retrocochlear pathology, and patients with superior canal dehiscence may have low frequency conductive hearing loss and supra-threshold bone conduction curves [14].

3.2. Vestibular system examinations

As previously described, the vestibular system facilitates balance, visual fixation, posture, and limb-trunk control via the receptor organs within the cochlea, utricle, saccule, and semicircular canals of the inner ear. Whereas the cochlea's principal function is sound transmission for hearing, the utricle and saccule are sensory organs for linear acceleration in all horizontal and vertical directions of head motion, respectively, and the perpendicularly oriented semicircular canals help detect head angular acceleration in all three dimensional planes. This complex vestibular apparatus is lined by nearly 100,000 neurosensory hair cells, found within each of the aforementioned anatomical components. These dispersed ciliated hair cell organs are embedded in a local gelatinous matrix and become interconnected *via* a fluid medium known as endolymph. Collectively, these vestibular components transmit information with hair cell motion that occurs with all head movement patterns. In humans, these five vestibular sense organs in each labyrinth are innervated by approximately 18,000 bipolar neurons with ganglionic cells located in the internal auditory canal, known as Scarpa's ganglion [15, 16]. Various tests have been developed to evaluate the integrity of the vestibular system.

a. Electronystagmography (ENG) records eye movements during various visual tracking tasks, with and without various head movement adjustments (up/down, left/right) and after warm and cold water (or air) applications to the ear canals. Ocular movements are recorded indirectly with the use of periorbital electrodes. Videonystagmography (VNG) uses infrared camera-goggles for direct recordings of eye movement patterns and is now used more freqently. It requires that the eyes remain wide open; make-up is prohibited because it reduces overall signal resolution. This method eliminates the issue of muscle artifact or active baseline that can be seen with ENG. Both modalities may help determine whether complaints of vertigo are causally related to abnormalities of the peripheral vestibular apparatus, the central vestibular pathways, or both. Four primary tests are utilized in both VNG and ENG. Occular mobility examines how well the patient visually tracks moving objects. Slowness or inaccuracies may reflect neurological problems, possibly involving pathways connecting the vestibular

apparatus to the visual cortex. Optokinetic nystagmus is evaluated as the patient visually tracks a large continuously moving image. Unusual involuntary, rapid shimmying or shaking of the eyes during this task often indicates central neurological abnormality, often involving vestibulo-ocular reflex pathways. Positional nystagmus is measured by abrupt and various passive movements of the patient's head to detect inappropriately induced rapid movements of the eyes. If identified, such nystagmus often suggests that otoliths (small calcium carbonate particles) are inappropriately suspended in the endolymph of one or more of the semicircular canals, causing a disturbance to normal fluid flow. This is usually observed in patients with BPPV, most commonly in the posterior semicircular canal. Caloric testing involves stimulation of each ear canal separately with warm and then cold air to determine that both ears are reactive *via* specific eye movement patterns. Results can help demonstrate unilateral versus bilateral vestibular system dysfunction or weakness, with particular sensitivity evaluating function of the horizontal semicircular canals.

b. Vestibular evoked myogenic potential (VEMP) is a test that can be used to evaluate the functional status of the utricle and saccule. These two structures contain a region called the macula, the neuroepithelial end organ of the vestibular nerve. It is composed of a three part structure with hair cells and an overlying viscous gelatinous medium embedded with calcium carbonate particles known as otoliths. When these particles are activated through variable linear head movements they deflect the underlying hair cells and induce central sensory neural transmission of associated motion. More than 50 years ago it was shown that certain neck muscles exhibited electromyographic activity in response to loud clicks. Such muscle reactivity was believed to be mediated by otolithic activity. More recently, peripheral inhibitory responses of the sternocleidomastoid muscles to such clicks have been demonstrated ipsilateral to the ear stimulated [17]. This is known as cervical VEMP (cVEMP) testing and it has been employed in the diagnosis of superior semicircular canal dehiscence, which is known to induce clinical symptoms of vestibular activation by loud sounds. In this clinical population, there is normally a pathologically lowered threshold and increased amplitude response in the click-evoked cVEMP test. [17]. Additionally, this test has been used successfully, either to confirm or refute the diagnosis of vestibular neuronitis and more variably with Meniere's disease. These patients may have higher thresholds and lower amplitudes in response to VEMP testing due to decreased vestibular input. Similar reflexes have been demonstrated in the contralateral inferior oblique occular muscle with click stimuli (oVEMP). The results of this test procedure are ordinarily quite reliable and reproducible, with some studies suggesting superiority over cVEMP [18-20]. High correlations between cVEMP test results and inferior vestibular nerve pathology have been demonstrated; conversely, strong causal relationships have been demonstrated between oVEMP test results and isolated superior vestibular nerve pathology [21-23].

c. Electrocochleography (ECOG) measures electrical potentials of the cochlea. It is a controversial test, as it is highly specific but with low sensitivity, dependent on the established threshold. Tone bursts or clicks are presented to the cochlea resulting in an action potential (AP) and a summating potentionl (SP). Endolymphatic hydrops or Meniere's disease should be suspected in cases with an SP/AP ratio greater than 0.4 to 0.5 when extratympanic techniques are used [24]. Although the more invasive transtympanic measurement approach has been considered more reliable by some clinical researchers, sufficient evaluation is often achieved through electrode placement on the tympanic membrane, characterisitc of the extratympanic approach [24].

d. Rotational chair testing is particularly useful in the assessment of bilateral vestibular dysfunction. It is considered a valuable adjunct to ENG because the procedure specifically enables close examination of the vestibular occular reflex (VOR); that is, head movement in one direction normally induces involuntary eye movements in the opposite direction. Close inspection of this involuntary physiologic phenomenon is the prinicple basis for the development of the variable speed rotary chair test. Many individuals with bilateral vestibular deficiency or cerebellar occular motor abnormalities are accurately diagnosed using this chair spinning procedure, often reflected with decreased gain and increased phase test outcome measures.

e. Posturography provides a quantitative assessment of all processes used to maintain balance, including vestibular, visual, and somatosensory. The patient is strapped in a harness and stands on a computer-controlled movable platform surrounded by a movable visual scene. Three functional test protocols exist: sensory organization test, motor control test, and the adaptation test. Patient sway is detected by pressure sensitive gauges in the platform. The patient's score is demonstrated on a graph and compared to age-corrected normative values. This test is especially useful when all other testing is inconclusive and in cases where malingering is suspected. These individuals parodoxically fail the "easier" testing modules, but manage to pass the more difficult ones.

3.3. Hematologic laboratory testing

Blood appraisal is not critical in the evaluation of every dizzy patient, but should be considered and employed for those whose diagnosis is less straightforward. Specific studies may be chosen based on the patient's symptomatology and past medical history. The following is a list of frequently prescribed blood tests to rule out various possible underlying etiologies: 1) Complete blood count, folate, and vitamin B12 levels for anemia, 2) Basic metabolic panel and HgbA1c for diabetes mellitus, 3) Thyroid stimulating hormone for thyroid function, 4) Lyme (*Borrelia burgdorferi*) and syphilis titers, 5) Antinuclear antibody and rheumatoid factor for autoimmune disease, and 6) Erythrocyte sedimentation rate and C-reactive protein for inflammatory disorders. In rare cases, where symptoms of vertigo are progressive and other neurological findings are noted, a lumbar puncture may be indicated. Not only will this procedure produce a biochemical analysis of the cerebrospinal fluid for monoclonal antibodies or infections, opening pressure may be recorded to evaluate the degree of intracranial hypertension.

3.4. Radiologic imaging

Imaging studies may be prescribed for patients with atypical dizziness features, or for those whose symptoms may be of neurologic origin. A computed tomography (CT) of the head may be obtained without contrast in the acute setting to rule out an acute cerebrovascular event. CT is widely available, may be obtained quickly, and is useful to delineate bony anatomy. Bony dehiscence of the superior semicircular canal may be noted on Stenver or Pöschl views of the temporal bone.

Magnetic Resonance Imaging (MRI) is more widely used to evaluate soft tissue and allows differentiation between the gray and white matter of the brain. In patients suspected of having a vestibular schwannoma, an MRI of the brain and internal auditory canal (IAC) should be obtained with and without gadolinium contrast. An enhancing lesion in the IAC or the cerebellopontine angle on post contrast T1-weighted images is consistent with a vestibular schwannoma. This abnormality can be identified with highest sensitivity using MRI of the IAC, detecting lesions as small as 3 mm, versus 10-15 mm using high-resolution CT imaging [25]. Such technology may also be of adjunctive value in the diagnosis of multiple sclerosis (MS) and Parkinson disease, two neurodegenerative conditions that often result in disequilibrium and imbalance. Finally, vertebrobasilar insufficiency or other vascular etiologies may be evaluated with MR or CT angiography.

Figure 3 illustrates suggested sequences of examination and testing steps for the differential diagnosis of vertigo, disequilibrium, and lightheadedness. For completeness, figure 4 is a graphic display of alternative treatment strategies for different forms of dizziness. Information in this flow chart will be addressed in detail in our forthcoming companion paper.

4. Summary

Dizziness is a very common generic complaint. As described in this paper, this condition often represents a spectrum of various possible sensations, including true vertigo, disequilibrium, and lightheadedness. Appropriate diagnostic and treatment strategies are not always straightforward because the underlying medical history of dizziness is ordinarily quite variable across these different patient populations. The clinical and laboratory workup of dizziness often includes a multitude of tests, especially for patients who present atypical symptom profiles or have difficulty in accurately describing their symptoms. Thorough history taking is perhaps the most important component of the differential diagnostic battery. Results of such comprehensive review of the patient's medical

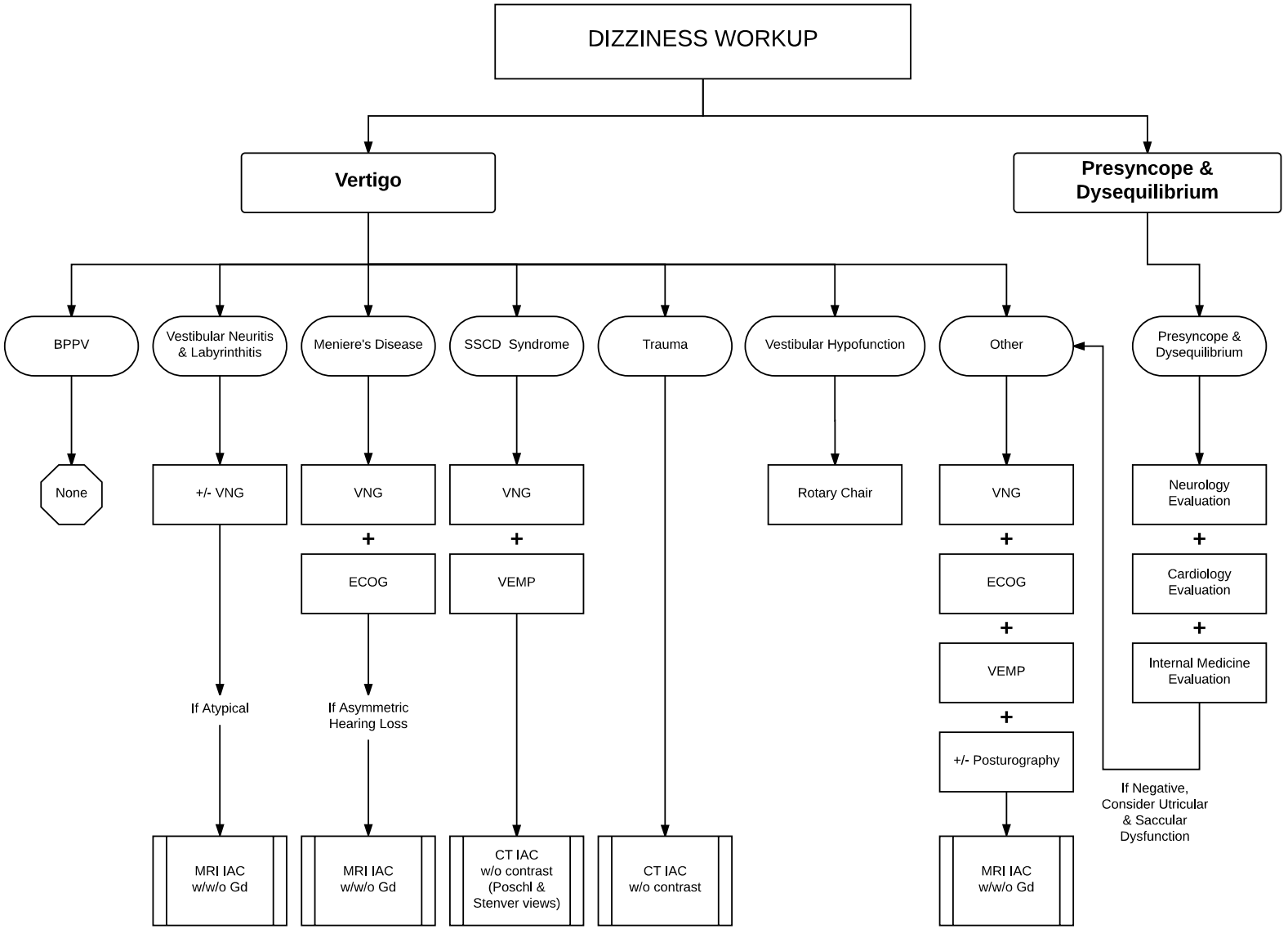

Figure 3. Dizziness workup. Key: BPPV (benign paroxysmal positional vertigo), ECOG (electrocochleography), IAC (internal auditory canal), SSCD (superior semicircular canal dehiscence), VEMP (vestibular evoked myogenic potential), VNG (videonystagmography).

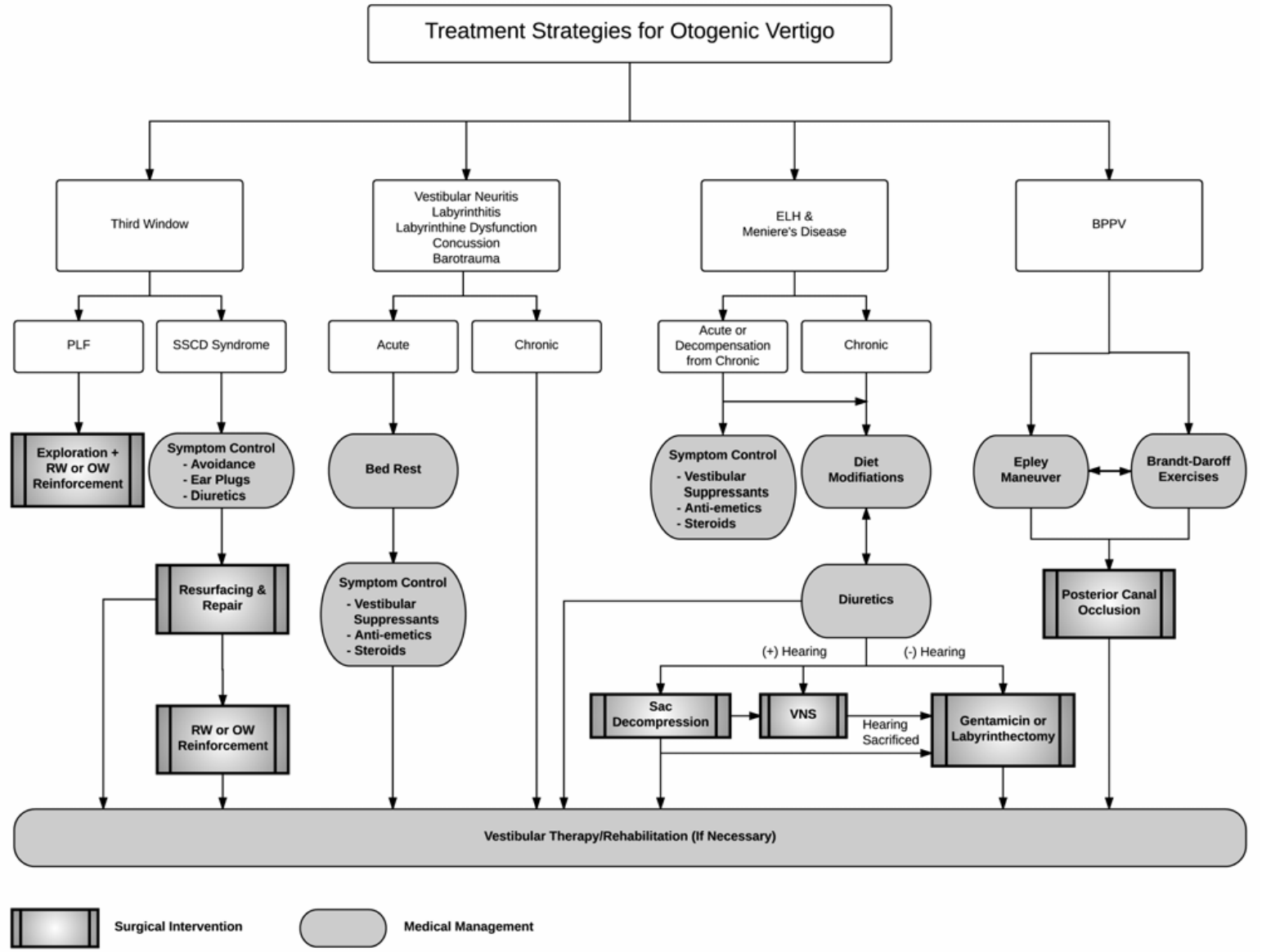

Figure 4. Treatment strategies for otogenic dizziness. Key: BPPV (benign paroxysmal positional vertigo), ELH (endolymphatic hydrops), OW (oval window), PLF (perilymphatic fistula), RW (round window), SSCD (superior semicircular canal dehiscence), VNS (vestibular nerve section).

background, onset characteristics, possible antecedent events, and chief signs and symptoms often lead the astute examiner to a consolidated list of formal tests. Findings from these evaluations either lead to the differential diagnosis and treatment recommendations or the need for additional testing before any conclusions can be drawn. The illustrative flowcharts presented in this paper were designed to facilitate these diagnostic and management decision processes.

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CONFLICT OF INTEREST STATEMENT

There are no conflicts of interest to disclose.

REFERENCES

1. Gassmann, K. G. and Rupprecht, R. 2009, J. Nutr. Health Aging, 13(3), 278-82.
2. Yardley, L., Owen, N., Nazareth, I. and Luxon, L. 2001, J. Nerv. Ment. Dis., 189, 321.
3. Rosenhall, U., Johansson, G. and Orndahl, G. 1996, Scand. J. Rehabil. Med., 28, 225.
4. Sloane, P. D. 1996, Clin. Geriatr. Med., 12, 785.
5. Maarsingh, O. R., Dros, J., Schellevis, F. G., van Weert, H. C., van Der Windt, D. A., Ter Riet, G. and van Der Horst, H. E. 2010, Ann. Fam. Med., 8, 196.
6. Baloh, R. W., Jacobson, K. M., Beykirch, K. and Honrubia, V. 1998, Arch. Neurol., 55(5), 649.
7. Stanton, V. A., Hsieh, Y. H., Camargo Jr., C. A., Edlow, J. A., Lovett, P. B., Goldstein, J. N., Abbuhl, S., Lin, M., Chanmugam, A., Rothman, R. E. and Newman-Toker, D. E. 2007, Mayo Clin. Proc., 82, 1319.
8. Lacour, M. 1989, Vestibular Compensation: Facts, Theories, and Clinical Perspectives. Elsevier, Amsterdam.
9. Vidal, P. P., De Waele, C., Vibert, N. and Muhlethaler, M. 1998, Otolaryngol. Head Neck Surg., 119, 34-42.
10. McCabe, B. F., Ryu, J. H. and Sekitani, T. 1972, Laryngoscope, 82, 381-96.
11. Zee, D. S. 1994, Vestibular Adaptation in Vestibular Rehabilitation, S. J. Herdman (Ed.), F. A. Davis Company, 68-79.
12. Eaton, D. A. and Roland, P. S. 2003, Geriatrics, 58, 46, 49-52.
13. Fukuda, T. 1958, Acta Otolaryngol. (Stockh), 50, 95.
14. Lee, G. S., Zhou, G., Poe, D., Kenna, M., Amin, M., Ohlms, L. and Gopen, Q. 2011, Laryngoscope, 121(10), 2256-261.
15. Rasmussen, A. T. 1940, Laryngoscope, 50, 67-83.
16. Jackler, R. K. and Brackmann, D. E. 2005, Neurotology. Philadelphia, PA, Mosby.
17. Milojcic, R., Guinan, J. J., Rauch, S. D. and Herrmann, B. S. 2013, Otol. Neurotol., 34(2), 360-67.
18. Nguyen, K. D., Welgampola, M. S. and Carey, J. P. 2010, Otol. Neurotol., 31(5), 793-802.
19. Welgampola, M. S., Myrie, O. A., Minor, L. B. and Carey, J. P. 2008, Neurology, 70(6), 464-72.
20. Zuniga, M. G., Janky, K. L., Nguyen, K. D., Welgampola, M. S. and Carey, J. P. 2013, Otol. Neurotol., 34(1), 121-26.
21. Murofushi, T., Nakahara, H., Yoshimura, E. and Tsuda, Y. 2011, Acta Otolaryngol., 131(9), 945-50.
22. Shin, B. S., Oh, S. Y., Kim, J. S., Kim, T. W., Seo, M. W., Lee, H. and Park, Y. A. 2012, Clin. Neurophysiol., 123(2), 369-75.
23. Walther, L. E. and Blödow, A. 2013, Otol. Neurotol., 34(6), 1084-089.
24. Wuyts, F. L., van De Heyning, P. H., van Spaendonck, M. P. and Molenberghs, G. 1997, Acta Otolaryngol., 117(Suppl. 526), 14-20.
25. Lin, D., Hegarty, J. L., Fischbein, N. J. and Jackler, R. K. 2005, Arch. Otolaryngol. Head Neck Surg., 131(3), 241-4.