Chapter 11: Ear, Nose, Throat, and Related Structures

Introduction

This chapter provides criteria for evaluating permanent impairments resulting from principal dysfunction of the ear, nose, throat, and related structures. It assesses permanent impairment ratings of these structures by evaluating losses in structure or the following functions: hearing; equilibrium; facial motion; respiration; mastication, deglutition, olfaction, taste and smell; speech and voice; and the effect of these losses on the ability to perform activities of daily living (ADLs). Mental health of the subject also may be affected and can be evaluated as described in Chapter 14. Impairment criteria, listed in earlier editions of the *Guides,* were adapted from the American Academy of Otolaryngology–Head and Neck Surgery.[1] Abbreviations and their definitions are listed in the Glossary.

For the Sixth Edition and subsequent online revisions thereof, all sections have been reviewed and revised. The principles used in the Sixth Edition table organization are consistent with other chapters and the philosophy established in Chapter 1. Some changes have been made in the impairment percentages to provide clear selection of a specific number and to accommodate additional ratings in complex cases, which may be combined with ratings in other chapters. Ratings also have been changed slightly to make them more consistent with impairment ratings in other chapters. Thus, the reader should anticipate that some ratings might deviate slightly from previous editions.

11.1 —Principles of Assessment

Before using the information in this chapter, the *AMA Guides* user should become familiar with Chapters 1 and 2 and the Glossary. Chapters 1 and 2 discuss the *AMA Guides*' purpose, applications, and methods for performing and reporting impairment evaluations. The Glossary provides definitions of common terms used by many specialties in impairment evaluations.

While hearing sensitivity may be tested with and without an assistive device, use of an assistive device will give a false impression of a subject's hearing sensitivity. Only the non-assisted measurement should be used to determine the impairment rating so that the need for hearing conservation and other indicated measures can be evaluated accurately.

11.1a –Interpretation of Symptoms and Signs

The history of specific symptoms ~~and their severity, duration,~~ including timing and manner of onset, duration, progression, triggers, and severity, along with the physical examination and diagnostic studies, will establish the diagnosis and will guide the impairment rating

process. Since the ear, nose, throat, and related structures have distinct functions, disorders of each system will be covered separately in this chapter. Permanent impairments of each system that have no overlapping functional losses are evaluated separately and then combined.

Some impairment classes refer to limitations in the ability to perform daily activities. When this information is subjective and possibly misinterpreted, it should not serve as the sole criterion on which decisions about impairment are made. ~~Rather, obtain objective~~Objective data about the severity of the findings and the limitations~~,~~ should be obtained and ~~integrate the findings~~integrated with the subjective data to estimate the degree of permanent impairment.

11.1b –Description of Clinical Studies

Multiple and diverse tests are used to investigate the ear, nose, throat, and related structures. Some of these tests are discussed in the relevant organ system section.

11.2 —Hearing and Tinnitus

The ear consists of the auricle, the external auditory canal, the tympanic membrane, the ossicles, the middle ear, the eustachian tube, the mastoid, the inner ear, and the internal auditory canal. The auditory and vestibular systems include the ear, sensory apparatus, eighth cranial nerve, and central nervous system pathways.

The ear provides sensorineural input critical to the senses of hearing, spatial perception, and balance. Hearing enables contact with environmental cues (e.g., those that alert) and enables ~~us to communicate socially. Balance contributes~~social communication. Spatial perception and balance contribute to maintenance of equilibrium in relation to the environment. ~~Balance function is~~ These functions are mediated by dynamically monitoring and integrating information about the position of the head, eyes, trunk, and joints at rest and ~~with~~during activity. Hearing and ~~some components of~~certain aspects of spatial misperception and balance disturbances can be measured objectively.

Chronic otorrhea and other conditions such as otalgia and tinnitus that are subjective[2] should be noted based on the individual's self-reports, but they cannot be measured objectively.

Permanent hearing impairment is a reduced hearing sensitivity, outside the range of normal for healthy young adults.[3] Hearing should be evaluated after maximum rehabilitation has been achieved and when the impairment is no longer accelerating beyond an age-

appropriate rate. Evaluation of hearing impairment should be based on the individual's binaural hearing, determined from the pure tone audiometry.[4]

11.2a Evaluation of Hearing Impairment

Hearing loss may be sensorineural, conductive, mixed, and/or central. Sensorineural hearing impairment is caused by pathologic processes taking place in the cochlea, the acoustic nerve, or the brain stem. There are many causes of sensorineural hearing impairment, including excessive noise exposure, ototoxic medications, childhood diseases, hereditary hearing loss (which may begin at any age), presbycusis, meningitis, tumors, infections, and head injuries, among others.[2,5] Conductive hearing impairment is due to abnormalities in the external or middle ear, including but not limited to otosclerosis, otitis media with or without eustachian tube dysfunction, congenital deformities, otitis externa, and impacted cerumen. Mixed hearing impairment occurs when there is combined sensorineural and conductive pathology. Examples include advanced otosclerosis and chronic otitis media. Central "hearing loss" involves the inability to process auditory signals; it may be seen with multiple sclerosis, head trauma, brain tumors and other conditions. It is different from peripheral (inner ear or eighth nerve) hearing loss in that it is a form of brain dysfunction, cannot be quantified easily; and is excluded from consideration for impairment rating in this chapter.

The human ear has a frequency range from about 20 to 20,000 Hz. It is also extremely sensitive in detecting sounds of low intensity. ~~Pure tone~~ Hearing threshold measurements are made with an instrument called an audiometer. Earphones are placed over the ears or in the ear canal, and tones are controlled for intensity and frequency to determine the hearing threshold, which is the lowest sound pressure level that can be heard by the individual. Routine audiometry requires a voluntary response such as raising a finger or hand or pushing a button. Hearing is usually measured with pure tone signals at 250, 500, 1000, 2000, 3000, 4000, 6000, and 8000 Hz. Intensity is measured in dBHL (decibels hearing level). In this chapter, dB will refer to dBHL unless otherwise specified.

Air conduction tests measure the status of the external, middle, and inner ear, including the cochlea, acoustic nerve, brain stem, and cortex. Bone conduction tests measure sensorineural function more directly, bypassing the external and middle ear. Speech reception and discrimination are tested by using spondee (2 stressed syllables) and phonetically balanced words.

There are more sophisticated and specialized tests, such as brain-stem evoked response audiometry, also called auditory brain-stem response or auditory evoked potential; electrocochleography; otoacoustic emission tests; and middle ear impedance

measurement. These tests, along with other medical evaluations (blood tests, imaging studies, and other tests), are used by otologists to help determine the nature and specific cause of hearing impairment in selected individuals.

11.2b –Tinnitus

Tinnitus is a term used to describe perceived sounds that originate within a person, rather than in the outside world. Although nearly everyone has mild tinnitus momentarily at some point in life, continuous tinnitus is abnormal.

In 2022, Jarach et al published a systematic review on tinnitus globally.[6] They reviewed 767 publications on tinnitus and identified 113 that met the criteria for review. The papers had been published from 1972 through 2021. 83 articles provided information on prevalence, and 12 articles provided the information on tinnitus. The pooled prevalence of any tinnitus among adults was 14.4%. However, prevalence was affected by age and was present in 9.7% of adults aged 18-44, 13.7% of those aged 45 – 64, and 23.6% of adults aged 65 or older. The pooled prevalence of severe tinnitus was 2.3%, and the pooled prevalence of chronic tinnitus was 9.8%. The pooled incidence rate of any tinnitus was 1,164 per 100,000 person-years. Their data are similar to those reported in 2014 in the National Health Interview Survey, the most recent year that includes tinnitus data. That study showed that in the United States, 11.2% of the population (about 27 million people) had tinnitus in 2014 of whom 41.2% had tinnitus all the time, and 28.3% had had tinnitus for 15 years or more.

~~The National Center for Health Statistics has reported that about 32% of all adults in the United States acknowledge having had tinnitus at some time.[6] Approximately 6.4% of the affected individuals characterize their tinnitus as debilitating or severe.~~ The prevalence of tinnitus increases up until approximately age 70 years and declines thereafter.[2(pp411–440)(428-441)] This symptom is more common in people with otologic problems, although tinnitus also can occur in otologically normal patients.

It has been speculated that tinnitus may be the result of a continuous stream of discharges along the auditory nerve to the brain caused by abnormal irritation in the sensorineural pathway. Although no sound is reaching the ear, the spontaneous nerve discharge may cause the patient to experience a false sensation of sound. This theory sounds logical, but there is no scientific proof of its validity.

Thus, tinnitus is not a disease but rather is a symptom that may be the result of disease or injury. However, tinnitus is so common that establishing causation is frequently difficult. The principal reason for the increasing interest in tinnitus within the context of a discussion of impairment is its effect on the daily activities of those individuals who have it. The major problem with evaluating tinnitus is that it is primarily a subjective phenomenon.

Consequently, it is frequently difficult to verify even the presence of tinnitus, let alone its consequences. Nonetheless, if the tinnitus interferes with ADLs, including sleep, reading (and other tasks requiring concentration), enjoyment of quiet recreation, and emotional well-being, up to 5% may be added to a measurable binaural hearing impairment. There is currently no way to scientifically evaluate tinnitus, although validated instruments such as the Tinnitus Handicap Inventory have been used.[7] Consequently, because physicians are often required to rate tinnitus, a variety of individually devised systems have been created using reasonable data sources. However, these are not standardized ~~or~~, nor are they generally accepted by any official medical organization, such as the American Academy of Otolaryngology–Head and Neck Surgery or the American Medical Association. As an example, tinnitus may be scaled as slight, mild, mild-moderate, moderate, or severe.[8] Verification of the presence of tinnitus through techniques matching loudness and pitch is fraught with pitfalls and not recommended.

11.2c Criteria for Rating Impairment due to Hearing Loss

Criteria for evaluating hearing impairment are established through hearing threshold testing, which serves as the most reproducible of the measures of hearing. Hearing impairment is measured by evaluating hearing in each ear separately and both ears together using audiometry. The binaural hearing impairment percentage is based on the severity of the hearing loss, which accounts for changes in the ability to perform ADLs.

In the calculation of a hearing impairment rating, no correction for presbycusis should be made because: (1) the method in Section 11.2d calculates the degree of hearing and assigns a rating regardless of cause (e.g., age, injury, or noise exposure) and (2) age correction would result in a reduced binaural impairment score that would thus underestimate the true magnitude of the hearing impairment. The estimation of the relative contributions of different causes of hearing impairment is an apportionment process, as described in Chapter 2.

11.2d Audiometric Measurements to Determine Hearing Impairment

Hearing levels are determined according to American National Standards Institute (ANSI) Standard S3.6-1996.[4] In the determination of impairments, the following steps should be taken:

1. Test each ear separately with a pure tone audiometer and record the hearing levels at 500, 1000, 2000, and 3000 Hz. The following rules apply for extreme values:
    a. If the hearing level at a given frequency is greater than 100 dB, the level should be taken as 100 dB.

b. If the hearing level for a given frequency has a negative value ~~(eg, 25 dB),~~, the level should be taken as 0 dB.

2. Add the 4 hearing levels (dB) for each ear separately.
3. For monaural impairment, see Section 11.2a and consult Table 11-1 to determine the percentages of monaural hearing impairment for each ear.
4. For binaural impairment, see Section 11.2b and consult Table 11-2 to convert the monaural hearing impairment percentages to a binaural hearing impairment rating.
5. Consult Table 11-3 to determine the impairment of the whole person.

Table 11-1 **Monaural Hearing Loss and Impairment**[a] (Content was not changed. Table was combined into a single page for ease of reading)

DSHL[b]	%	DSHL[b]	%	DSHL[b]	%	DSHL[b]	%
100	0	170	26.2	240	52.5	310	78.8
105	1.9	175	28.1	245	54.4	315	80.6
110	3.8	180	30.0	250	56.2	320	82.5
115	5.6	185	31.9	255	58.1	325	84.4
120	7.5	190	33.8	260	60.0	330	86.2
125	9.4	195	35.6	265	61.9	335	88.1
130	11.2	200	37.5	270	63.8	340	90.0
135	13.1	205	39.4	275	65.6	345	91.9
140	15.0	210	41.2	280	67.5	350	93.8
145	16.9	215	43.1	285	69.3	355	95.6
150	18.8	220	45.0	290	71.2	360	97.5
155	20.6	225	46.9	295	73.1	365	99.4
160	22.5	230	48.8	300	75.0	≥370	100.0
165	24.4	235	50.6	305	76.9		

[a] Audiometers are calibrated to ANSI Standard S3.6-1996 reference levels.[4]

[b] Decibel sum of the hearing threshold levels at 500, 1000, 2000, and 3000 Hz.

Table 11-2 (not printed due to size limitations)

Table 11-3 **Relationship of Binaural Hearing Impairment to Impairment of the Whole Person** Content was not changed. Table was combined into a single page for ease of reading

% Binaural Hearing Impairment	% Impairment of the Whole Person	% Binaural Hearing Impairment	% Impairment of the Whole Person
0–1.4	0	50.0–52.8	18
1.5–4.2	1	52.9–55.7	19
4.3–7.1	2	55.8–58.5	20
7.2–9.9	3	58.6–61.4	21
10.0–12.8	4	61.5–64.2	22
12.9–15.7	5	64.3–67.1	23
15.8–18.5	6	67.2–69.9	24
18.6–21.4	7	70.0–72.8	25
21.5–24.2	8	72.9–75.7	26
24.3–27.1	9	75.8–78.5	27
27.2–29.9	10	78.6–81.4	28
30.0–32.8	11	81.5–84.2	29
32.9–35.7	12	84.3–87.1	30
35.8–38.5	13	87.2–89.9	31
38.6–41.4	14	90.0–92.8	32
41.5–44.2	15	92.9–95.7	33
44.3–47.1	16	95.8–98.5	34
47.2–49.9	17	98.6–100.0	35

This method of evaluating hearing impairment should be applied only to adults who have acquired language skills. Evidence suggests that language acquisition by children who do not have language skills may be delayed when the average hearing level is in the range of 15 to 25 dB. The *AMA Guides'* methods for calculating impairment are not intended to examine an individual's work disability but are meant to account for daily activities that are common in most people. Some workers in occupations with specific and difficult hearing-critical tasks (e.g., musician, piano tuner) may have significant work disability despite 0% binaural hearing impairment. Similarly, the *AMA Guides'* impairment estimates will not necessarily correlate with difficulties performing unusual nonoccupational hobbies, such as bird watching.

11.2e Evaluation of Monaural Hearing Impairment

If the average of the hearing levels at 500, 1000, 2000, and 3000 Hz is 25 dB or less, according to the 1996 American National Standards Institute (ANSI) audiometric standards,[4] no impairment rating is assigned since there is no change in the ability to hear every day sounds under everyday listening conditions (Table 11-1). The 25-dB "fence" represents this finding; it is not a compensatory adjustment for presbycusis, the hearing loss that occurs with age.

At the other extreme, if the average of the hearing levels at 500, 1000, 2000, and 3000 Hz is more than 91.7 dB, the binaural hearing impairment rating is 100% since the individual has lost the ability to perform an ADL—the ability to hear everyday speech.[1]

According to the above standards for monaural hearing impairment, for every decibel for which the average hearing level or loss of speech exceeds 25 dB, 1.5% of monaural impairment is assigned. Thus, with an average hearing level loss of 67 dB above 25 dB, monaural impairment is 100% (Table 11-1).

11.2f Evaluation of Binaural Hearing Impairment

Hearing impairment of both ears, referred to as binaural impairment, indicates a loss of hearing of greater than 25 dB in both ears at frequencies of 500, 1000, 2000, and/or 3000 Hz.

Binaural impairment is determined by the following formula:

$$Binaural\ Hearing\ Impairment\ (\%) = \frac{5 \times (\%\ hearing\ impairment\ better\ ear) + (\%\ hearing\ impairment\ poorer\ ear)}{6}$$

To calculate binaural impairment when only 1 ear exhibits hearing impairment, use this formula, allowing 0% impairment for the unimpaired ear (the ear with the better hearing).

Alternatively, use Table 11-2, which is derived from the formula given here, to calculate the value for binaural hearing impairment. Then apply the value for binaural hearing impairment to Table 11-3, which converts binaural hearing impairment to impairment of the whole person.

5% Impairment of the Whole Person

Example 11-1: Hearing Loss

Subject: 70-year-old woman.

History: Retired secretary. Chronic recurrent ear infections since teens. Occasional drainage from right ear. Right ear now dry but feels "like stuffed with cotton." Has occasional tinnitus in right ear; not bothersome. No dizziness.

Current Symptoms: Difficulty hearing, especially in right ear, with no impact on ADLs.

Physical Exam: Scarred, retracted right tympanic membrane. Left tympanic membrane is thickened and retracted. Pneumatic otoscopy shows motion of left tympanic membrane, but no motion on right.

Clinical Studies: Tympanograms: B pattern for right ear and A pattern for left ear. Speech discrimination score: 80% for right ear; 95% for left ear. Acoustic immittance reveals normal external auditory canal volumes for both ears. Pure tone audiometry reveals the threshold levels in decibels (dB) given in Comment.

Diagnosis: Mixed (sensorineural + conductive) hearing impairment, right ear. Mild sensorineural hearing impairment, left ear.

Impairment Rating: 5% impairment of the whole person.

Comment: The decimal sum of hearing threshold levels (DSHL) for the right ear is 225 (40 + 55 + 60 + 70), and the DSHL for the left ear is 125 –((25 + 30 + 30 + 40). Combine 225 (worse ear) and 125 (better ear) using Table 11-2 for a binaural hearing impairment rating (BI) of 15.6%. Use Table 11-3 to obtain the 5% whole person impairment rating.

8% Impairment of the Whole Person

Example 11-2: Mixed Hearing Impairment, Bilaterally

Subject: 65-year-old woman.

History: Repeated ear infections for many years. Hearing loss in both ears and roaring, pulsing, rushing-water tinnitus in both ears. No history of dizziness. Tympanoplasty, left ear, 4 months ago.

Current Symptoms: Difficulty hearing in both ears, but hearing much improved in left ear since tympanoplasty. Still has tinnitus in both ears, which despite use of hearing aids and cognitive-behavioral therapy, continues to cause severe sleep difficulties, frequent daytime fatigue, difficulty concentrating on quiet tasks, and moderately severe depression.

Physical Exam: Retracted right tympanic membrane.

Clinical Studies: Left tympanic membrane shows well-healed graft. Tympanograms: B pattern for right ear. Tympanometry was not performed for left ear due to recent otologic

surgery. Speech discrimination scores: 80% for right ear; 85% for left ear. Pure tone audiometry reveals the threshold levels in decibels (dB) given in Comment.

Diagnosis: Mixed (sensorineural + conductive) hearing impairment, bilaterally.

Impairment Rating: 8% impairment of the whole person.

Comment: The DSHL for the right ear is 210 (50 + 50 + 55 + 55), and the DSHL for the left ear is 135 (25 + 30 + 40 + 40). Combine 210 (worse ear) and 135 (better ear) using Table 11-2 for a BI of 17.8%. Add 5% for the presence of tinnitus that severely affects ADLs giving a BI of 22.8%. Use Table 11-3 to obtain the 8% whole person impairment.

Example 11-3: Sensorineural Hearing Impairment, Bilateral

Subject: 64-year-old man.

History: Retired machinist. Progressive hearing loss for 13 years. Worked in several noisy environments; used hearing protection fairly regularly. Exposure to gunfire during 4 years of service in the Marines. General health good. No history of tinnitus or vertigo.

Current Symptoms: Difficulty with communication at home, in restaurants, driving a car, and in noisy environments.

Physical Exam: No abnormalities.

Clinical Studies: Audiologic tests: speech reception threshold of 20 dB. Pure tone audiometry reveals the threshold levels in decibels (dB) given in Comment.

Diagnosis: Sensorineural hearing impairment, bilateral.

Impairment Rating: 8% impairment of the whole person. Apportionment for the military-related hearing loss can be accomplished only by subtracting impairment verified from audiograms reflecting the individual's post-Marine hearing status.

Comment: The impairment calculated from this audiogram is based on the DSHL. The DSHL for the right ear is 175 (20 + 15 + 60 + 80), and the DSHL for the left ear is 160 (25 + 15 + 60 + 60). Combine 175 (worse ear) and 160 (better ear) using Table 11-2 for a binaural hearing impairment of 23.4%. Use Table 11-3 to obtain the 8% whole person impairment.

11.2g –Equilibrium, Spatial Orientation and Balance

Equilibrium, or orientation in space, is maintained by the brain mechanisms that process and integrate visual, kinesthetic (proprioceptive), auditory, and vestibular ~~mechanisms. When~~inputs. Relevant impairments ~~of equilibrium are predominantly due to other organ~~

~~systems, the impairment should be evaluated in the relevant organ system, for example,~~may involve disorders of the nervous system ~~(Chapter 13),~~(Chapter 13), cardiovascular system ~~(Chapter 4), or~~ (Chapter 4), visual system ~~(Chapter 12). Permanent impairment may result from any disorder causing vertigo or disorientation in space. Three regulatory systems—~~(Chapter 12), or vestibular~~, ocular (visual), and kinesthetic (proprioceptive)—are related to the vestibulo-ocular reflex.~~ system. The evaluation ~~of~~for such impairments ~~of equilibrium~~ may include consideration of ~~1~~one or more of these mechanisms.~~[9,10]~~[9, 10] Impairments of any of these systems can affect the vestibulo-ocular reflex and may lead to vertigo, a false sensation of motion or dizziness, or a sense of spatial disorientation. Mental health and specifically anxiety and depression are frequent comorbidities of chronic vestibular disorders. [11,12] These should be taken into account separately when addressing the impairment rating of the whole person. This chapter addresses only disturbances ~~in equilibrium~~ due to vestibular disorders.

Dizziness, like ~~deafness and~~ tinnitus, is a subjective experience and is a symptom, not a disease. ~~Its~~ The lifetime prevalence of dizziness is 15-30%, and the cause must be sought carefully in each case.[13,14,15] Approximately a quarter of these patients with dizziness have a vestibular disorder and present with vertigo.[13]

Benign paroxysmal positional vertigo (BPPV), vestibular migraine (VM), and Ménière's disease (MD) are the three most common causes of episodic recurrent vertigo. These diagnoses represent half of the diagnosed vestibular conditions and are associated with significant quality of life disruption. Of the subjects presented to a dizziness clinic, BPPV is diagnosed in 20-30% of cases, Ménière's disease is present in 13%, and VM accounts for up to 23.4%. The total direct medical costs in the US are in the billions of dollars.[16]

Furthermore, up to 12% of individuals with dizziness end up on disability and over half complain of substantial impact on their daily professional life, with absenteeism and reduced productivity contributing to increased indirect cost.[17,18]

Patients use the term *dizziness* to describe a variety of sensations, many of which are not related to the vestibular system. It is convenient to think of the balance system as a complex conglomerate of ~~senses~~sensorimotor functions that ~~send~~receive sensory information ~~to the brain~~ about one~~'~~'s position in space~~.~~ and adjust the body position accordingly. Components of ~~the balance~~this system include the vestibular~~ labyrinth, the eyes, neck muscles,~~ , visual, somatosensory and proprioceptive ~~nerve endings, and~~ systems, the cerebellum~~.~~, and higher order neural networks within the cerebral hemispheres that process and integrate this sensory information further. These components are essential for detecting, delivering, processing, and integrating sensory information into a coherent sense of spatial orientation. This sensory integration process is

based on the availability and validity of each sensory modality. If all components of the balance system are providing accurate information, ~~one has~~ no equilibrium problem is present. However, ~~if we use the~~for example ~~that~~, if most of the components indicate to the brain that the body is standing still, but ~~1~~one component indicates that the body is turning to one side, the brain becomes confused, and a person will experience dizziness. It is the physician~~'~~'s responsibility to analyze systematically each component of the balance system to determine which component or components are providing incorrect information, or whether correct information is being provided and analyzed in an aberrant fashion by the brain.

~~Disturbances~~The international classification of ~~equilibrium may be classified~~vestibular disorders classifies vestibular symptoms as follows: (1) vertigo, a false sensation of ~~r~~motation of oneself in relation to the ~~subject~~environment that can be spontaneous or ~~of objects about the subject in any plane;~~triggered; (2) spontaneous or triggered dizziness or giddiness ~~or~~ characterized by a disturbed sense of spatial orientation (sometimes referred to as lightheadedness~~,~~ by patients, but not in the sense of presyncope), distinguished from vertigo by the absence of feelings of movement; ~~and (3~~(3) visual-vestibular symptoms including external vertigo (illusion of movement of objects in the surrounding environment), visual lag, visual tilt, movement-induced blur, oscillopsia with or without movements and (4) abnormalities of postural stability and/or standing balance with or without ~~vertigo. Vertigo~~dizziness or vertigo, including drop attacks that can occur in the context of Ménière's disease (Tumarkin crisis) or non-otologic drop attacks, as seen in vestibular migraine.[19]

Vestibular symptoms may be produced by ~~disorders of the vestibular mechanism and its~~peripheral lesions involving the labyrinth or eighth cranial nerve, as well as central nervous system components, including the cerebral cortex, ~~cerebellum,~~ brain stem, and ~~by eye movements. Vertigo~~cerebellum. Vestibular symptoms may be associated with neurovegetative symptoms such as nausea~~,~~ and vomiting, as well as vasovagal symptoms. Other accompanying symptoms include headache, fear of movement, ataxia, hearing loss, and ~~nystagmus. Movement or environmental object movement may worsen these symptoms.~~tinnitus. Some pathologies present with mixed peripheral and central manifestations.

Typically, labyrinthine dysfunction is associated with ~~a sense of motion~~vertigo. It may be true spinning, a sensation of being on a ship or of falling, or simply a vague sense of imbalance when moving. ~~In many cases, it is episodic. Fainting, body weakness, spots before the eyes~~Central vestibular disorders such as trauma or ischemia in the brain stem (acute vestibular deafferentation) can cause true rotary vertigo. Rocking sensations are seen most

often in central vestibular disorders such as vestibular migraine or Mal de Debarquement and rarely are caused by peripheral lesions. Many vestibular disorders can present in an episodic fashion with patients being asymptomatic between episodes. This includes peripheral disorders such as benign paroxysmal positional vertigo (BPPV) or Ménière's disease, but also central disorders with potential peripheral involvement such as vestibular migraine. Fainting, change in mentation, body weakness, visual spots, general lightheadedness, tightness in the head, and loss of consciousness are generally not of vestibular origin. ~~However, such descriptions are of only limited diagnostic help. Even some severe peripheral (vestibular or eighth nerve) lesions may produce only mild unsteadiness or no dizziness at all, as observed in many patients having acoustic neuromas. Similarly, lesions outside the vestibular system may produce true rotary vertigo, as seen with trauma or microvascular occlusion in the brain stem.~~ However, as stated above, they can accompany vestibular disorders or be part of the autonomic manifestations associated with those disorders.

Additional considerations include oculomotor disorders and ocular alignment issues, which may increase the burden of symptoms even if the cause is not a vestibular problem. These should be identified, treated and appropriately taken into consideration in the evaluation of the patient's impairment.

Furthermore, a substantial number of patients with vestibular disorders has diagnosable mental health problems that interfere with their symptoms, quality of life, and in some cases test findings (such as anxiety and increased sway on posturography, and more nonspecific findings on reflexive testing). Some vestibular disorders, such as Persistent Postural Perceptual Dizziness, may be indissociable from the associated mental health problem, with a vicious cycle of hypervigilance and panic leading to, enhancing, or filtering to vestibular symptoms.[20,21,22,23]

Finally, studies have shown the impact of vestibular dysfunction on cognition, specifically in visuospatial functioning, visual/spatial memory, navigation, attention, working memory, short term memory, and executive functioning. Various questionnaires have been used to screen patients to determine whether a referral to a neuropsychologist for further assessment is warranted (e.g. Cognitive Failure Questionnaire, Neuropsychological Vertigo Inventory). The objective assessment makes use of many tests, including the Repeatable Battery for the Assessment of Neuropsychological Status, Montreal Cognitive Assessment, and visuospatial tests including the Card Rotation Test, the Benton Visual Retention Test, and the Virtual Morris Maze Task.[24,25,26,27,28,29] If such issues are identified, they are beyond the scope of this chapter but may be considered separately if appropriate.

Causes of dizziness and/or specific vestibular symptoms are almost as numerous as causes of hearing loss, and some are medically serious, such as multiple sclerosis, acoustic neuroma, diabetes, migraine, anemia, and cardiac arrhythmia. Consequently, any patient with ~~an~~a vestibular or another equilibrium complaint needs a thorough examination. For example, although dizziness may be caused by head trauma, the fact that it is reported for the first time after an injury is not sufficient to establish causation without investigating other possible causes. One of the most common causes of vertigo following head trauma is BPPV. This does not mean that the trauma has caused permanent labyrinthine dysfunction.

The definitions and diagnostic criteria have changed ~~over the past decade~~ since around 2015 and are mostly reclassified by the Barany Society. For instance, the term Ménière's syndrome is no longer considered clinically useful. Instead, we have a better understanding of various endophenotypes of Ménière's disease, including idiopathic, posttraumatic, familial, as well as phenotypes associated with migraine, infection (e.g. syphilis), or autoimmune disorders. Delayed endolymphatic hydrops is also a term that refers to the posttraumatic endophenotype for the most part, but the term is considered obsolete by the recent Barany Society Consensus documents.[30,31] New causes of dizziness have also been recently described and categorized by the Barany Society.[25]

It is important to carry out a systematic inquiry in all cases ~~of disequilibrium~~with vestibular symptoms, not only because the condition ~~is~~can be caused by serious problems ~~in some cases~~ but also because many patients with ~~balance~~these disorders can be helped. Many people believe incorrectly that sensorineural hearing loss, tinnitus, and dizziness are incurable, but many conditions that cause any or all of these symptoms may be treated successfully. In other cases, stabilizing the disorder may prevent further damage of the auditory and vestibular functions as well as secondary repercussions, including depression and anxiety, which are common comorbidities of chronic vestibular disorders and can be implicated in the impairment. It is especially important to separate "peripheral," or noncentral, causes, which are ~~almost always~~more treatable, from more central causes, such as brain-stem contusion, in which the prognosis is often worse.~~11~~[32]

It is also important to assess and identify non-vestibular causes of dizziness and imbalance. For instance, diabetic sensory neuropathy can cause imbalance, and diabetic autonomic neuropathy can cause orthostatic dizziness, which can be attributed incorrectly to a vestibular cause. In contrast, vestibular conditions can lead to a hyperactive vagal response and autonomic dysfunction that is part of the primary vestibular presentation. Thorough assessment of the various causes of dizziness is important to help the patient and to assign the appropriate diagnosis and determine impairment accurately.

Clinical evaluations include history and physical examination, ~~along with possible use of~~ supplemented by electronystagmography (ENG~~) or~~), videonystagmography (VNG)/Videooculography (VOG), video head impulse testing (vHIT), caloric irrigation, ~~positional and~~ rotary ~~tests, dynamic posturography,~~chair testing, vestibular evoked myogenic potentials (VEMP), and video ocular counter-roll (cOCR). Additional postural assessments like the Romberg ~~and~~ test (including tandem Romberg ~~tests, and brain~~) and tandem stance can be supplemented by computerized dynamic posturography. Brain imaging studies~~.~~ can be valuable to identify or rule out structural lesions. The results of these tests should be correlated with validated clinical measures of balance and ambulation to determine the true state of ~~equilibratory~~balance and vestibular dysfunction.

Vestibular and Balance Testing

~~The balance system is extremely complicated, and ideal tests have not been developed. Research is currently under way to develop better tests that will assess accurately the entire composite functioning of the balance system and test each component in isolation. At present, the most commonly performed tests are ENG and computerized dynamic posturography (CDP). Vestibular evoked potential testing is under investigation.~~

~~Electronystagmography~~The balance system is complicated and involves intricate integration of the visual, vestibular, auditory, and somatosensory systems. Currently, reflexive paraclinical testing is standard for investigating function, even though the consensus among experts is that in most cases, a thorough clinical examination is sufficient.[33,34] For instance, the HINTS test (head impulse, gaze evoked nystagmus, test of skew) is more sensitive than diffusion weighed imaging (DWI) MRI in detecting a stroke versus peripheral vestibular lesion in the first 24 hours of an acute vestibular event.[35]

However, advancements in bedside and laboratory assessments have significantly improved diagnostic outcomes and satisfaction. By incorporating video head impulse testing (vHIT), rotary chair testing, and caloric irrigation for evaluating semicircular canal function, along with vestibular evoked myogenic potentials (VEMP) and video ocular counter-roll (vOCR) for assessing otolith function, it is now possible to test each vestibular end organ comprehensively.

Of note, screening for anxiety and depression using validated questionnaires is important in those cases to detect a mental health comorbidity that should be evaluated separately for its impact on the symptoms.

Video-oculography/Videonystagmography

~~Electronystagmography~~ ~~is a technique for recording eye movements to detect spontaneous and induced nystagmus, as well as central and peripheral oculomotor abnormalities. It allows measurement of eye movements with eyes open and closed, and permits quantification of the fast and slow phases, time of onset and duration, and other parameters. Although some centers use only horizontal electrodes, the use of both horizontal and vertical electrodes is preferable. Eye movements also can be tracked using videonystagmography (VNG), a newer and more sensitive technique that uses an infrared camera mounted on goggles instead of electrodes. ENG/VNG must be done under controlled conditions with proper preparation, which includes avoidance of drugs (especially those that affect the central nervous system).~~ Video-oculography (VOG) is a technique for recording eye movements to detect spontaneous and induced nystagmus, as well as central and peripheral oculomotor abnormalities. This method, often referred to as videonystagmography (VNG), uses infrared video cameras to record eye movements, whereas in electronystagmography (ENG), electrodes are placed around the eyes to measure electrical potentials generated by eye movements with eyes open or closed. Both methods measure direction of eye movements in different planes and permit quantification of the fast and slow phases of nystagmus, and characteristics such as latency to onset, duration, and intensity. Most vestibular testing centers use both. ENG/VNG must be done under controlled conditions with proper preparation, which includes avoidance of drugs that affect the central nervous system or peripheral labyrinthine function (e.g. Meclizine, and Benzodiazepines) and ingestion of alcohol within 48 hours prior to testing. Even a small drug effect may cause alterations in the ENG/VNG tracing. The test is performed in several phases. These include ~~calibrationand~~calibration and tests for gaze nystagmus, sinusoidal tracking, optokinetic nystagmus, spontaneous nystagmus, Dix-Hallpike and other ~~positioning testing,~~ positional testing, and caloric irrigations. These tests can give useful information about peripheral and central abnormalities in the vestibular system. Accurate interpretation is complex and requires a comprehensive understanding of the vestibular and ocular motor systems.~~[11,12]~~[32-35] The performance of ENG/VNG is especially helpful when a unilateral reduced vestibular response is identified in conjunction with other signs of dysfunction in the same ear. In such cases, it provides strong support for a peripheral (eighth nerve or end-organ) ~~cause of balance dysfunction~~vestibular dysfunction as the underlying cause of imbalance. The test also can suggest alternative vestibular pathologies. For instance, slow decay on step velocity rotary chair testing is seen often in motion-sensitive individuals and vestibular migraine. A normal video head impulse testing along with an abnormal caloric response/asymmetry has been reported in Ménière's disease and can be a soft marker of the pathology.

Caloric Testing

Caloric testing is a diagnostic procedure used to evaluate the function of the vestibular system, particularly the horizontal semicircular canals and their associated neural pathways. The test involves introducing warm or cool water (or air) into the ear canal to create a temperature gradient, which induces convection currents in the inner ear fluid (endolymph). This thermal stimulation mimics head movement and triggers the vestibulo-ocular reflex (VOR), resulting in nystagmus. The direction and intensity of these eye movements are recorded using VNG or ENG to assess vestibular function. Caloric testing is used commonly to diagnose vestibular disorders such as labyrinthitis or vestibular neuritis. It also is used to assess the vestibular function in chronic and episodic disorders such as Ménière's disease, as well as to differentiate between central and peripheral causes of dizziness or imbalance. A symmetrical response between ears is considered normal, while reduced, absent, or asymmetric responses (typically more than 20-25%) suggest vestibular dysfunction. When there is no response recorded with standard water calorics, ice calorics are performed to see if there is any residual response. Although caloric testing remains a standard diagnostic tool, newer techniques like the video head impulse testing (vHIT) are being used increasingly in clinical practice for a more comprehensive vestibular evaluation.

~~**Computerized Dynamic Posturography**~~

~~For more than 25 years, platforms have been used to try to assess more complex integrated functioning of the balance system. Until recently, most were static posture platforms with pressure sensors used to measure body sway while patients tried to maintain various challenging positions, such as the Romberg and Tandem Romberg maneuvers. Movement was measured with eyes closed and open. The tests had many drawbacks, including an inability to separate proprioceptive function and to eliminate visual distortion. In 1971, Nasher[13] introduced a widely used CDP system, and other systems also have become available.~~

~~Dynamic posturography uses a computer-controlled moveable platform with a sway-referenced surrounding visual environment. In other words, both the platform and visual surround move, tracking the anterior-posterior sway of the patient. The visual surround and platform may operate together or independently. The system is capable of creating visual distortions or totally eliminating visual cues. The platform can perform a variety of complex motions, and the patient's body sway is detected through pressure-sensitive strain sensors under the platform.[13]~~

~~The typical test protocol evaluates sensory organization through 6 test procedures and movement coordination through a variety of sudden platform movements. Balancing strategies and responses are assessed using both the sensory organization and movement~~

~~coordination test batteries. Dynamic posturography provides a great deal of information about total balance function that cannot be obtained from tests such as ENG alone. Dynamic posturography is also valuable in distinguishing organic from nonorganic disequilibrium, an asset that is particularly valuable in some cases of alleged impairment.~~

Rotary Chair Testing

~~Rotary chair testing helps identify whether dizziness is due to dysfunction in the inner ear or brain. Caloric testing (ENG/VNG) generally is considered the gold standard for identifying unilateral vestibular dysfunction. Rotary chair testing is the gold standard for detection of bilateral vestibular dysfunction. ENG/VNG is performed to evaluate most people with suspected labyrinthine dysfunction. Rotary chair testing is obtained when bilateral disease is suspected, or when more sophisticated information is required. It evaluates the vestibulo-ocular reflex (VOR) and the central vestibular system. Rotational chair testing has no contraindications such as neck trauma, which is not the case with ENG/VNG. In rotary chair testing, rotation is computer controlled and extremely accurate. It is well-tolerated and even can be performed in children. Rotary chair testing has been used for decades, and there is extensive literature on the technique. In addition to bilateral semicircular canal paresis, common indications include equivocal or inconclusive ENG/VNG results, evaluation of vestibular compensation, ototoxicity management, and testing of special populations including children and handicapped individuals. Rotary chair testing assesses the integrity of VORR, and suppression of that reflex. The tests are complementary. Unlike caloric testing, rotary chair testing stimulates both ears simultaneously and assesses simultaneously semicircular canals in both ears. Caloric testing assesses the vestibular system at a frequency of only .003 Hz; but rotary chair testing assesses from 0.01 Hz through 0.64 Hz.~~ While caloric testing (ENG/VNG) generally is considered the gold standard for identifying unilateral vestibular dysfunction, rotary chair testing is the gold standard for detecting bilateral vestibular dysfunction. Rotary chair testing involves measuring eye movements while a seated subject is rotated around a vertical axis at speeds that correspond to normal functional head rotation. It helps identify whether dizziness is due to dysfunction in the inner ear or brain and can be especially useful in confirming complete bilateral vestibular loss. Unlike ENG/VNG, rotary chair testing has no contraindications such as neck trauma, is well-tolerated, and can be performed on children. In addition to bilateral semicircular canal paresis, common indications include equivocal or inconclusive ENG/VNG results, evaluation of vestibular compensation and ototoxicity management. Intact brainstem and cerebellar function can be inferred by confirmation of the integrity of the vestibulo-ocular reflex and suppression of that reflex.

Common rotary chair ~~tests~~test algorithms include sinusoidal harmonic acceleration (SHA), VOR suppression, and the velocity step test. Other subtests may include slow harmonic (sinusoidal) testing in darkness, high-velocity or high-frequency sinusoidal rotation, rotation with fixation on head-fixed targets to evaluate suppression, optokinetic after-nystagmus, tilted-axis rotation (OVAR), rotation with fixation on earth-fixed targets, optokinetic testing, and others. Rotary chair testing allows monitoring of the VOR over time, which is important because the phase abnormality and symmetry in VOR recover in some patients following vestibular injury.

~~**Evoked Vestibular Response**~~

~~Evoked vestibular response testing is analogous to brain-stem auditory evoked testing. However, vestibular evoked potentials are not in wide clinical use.~~

Video ocular-counter-roll (vOCR)

Video ocular counter-roll (vOCR) testing is a diagnostic procedure used to evaluate the otolith organs of the vestibular system, specifically the utricle, which detects linear acceleration and gravitational forces. This test examines the eye's compensatory torsional movements, known as ocular counter-roll, in response to head tilts. During the procedure, patients are asked to tilt their head to one side while wearing specialized goggles or video equipment that tracks subtle torsional movements of the eyes. The degree and direction of these movements provide critical information about the functional integrity of the otolith organs and their neural pathways. Video ocular-counter-roll (vOCR) testing is particularly valuable for diagnosing otolith dysfunction, which can contribute to balance disorders, dizziness, and spatial disorientation. Unlike tests focusing on the semicircular canals, vOCR specifically assesses how the vestibular system processes linear acceleration and head tilt relative to gravity. The results help differentiate between peripheral and central vestibular disorders and complement other assessments. like vestibular evoked myogenic potentials (VEMP).

Vestibular Evoked Myogenic Potentials (VEMP)

Vestibular evoked myogenic response, both cervical and ocular, have become much more available and widespread in clinical use in recent years. VEMP assessments allow a specific evaluation of the otolith organs (utricle and saccule) and vestibular nerve through stimulation of two different reflex pathways: the vestibulo-collic reflex (cVEMP) and the vestibulo-ocular reflex (oVEMP). These tests utilize electrode measurements of a myogenic (muscle) potential in response to an acoustic or vibratory stimulus.

Cervical Vestibular Evoked Myogenic Response (cVEMP)

The cVEMP has come into wider clinical use in recent years as an evaluation of the vestibulo-collic reflex (VCR), also known as the "head righting" reflex. The cVEMP specifically assesses the function of the saccule and inferior branch of the vestibular portion of the eighth cranial nerve. In a cVEMP assessment, sound (traditionally a low frequency tone burst stimulus) is delivered through air or bone conduction (most often earphones inserted into the ear). This high-intensity acoustic stimulus elicits an ipsilateral reflex response of the sternocleidomastoid (SCM) muscle in the neck, which can be recorded through strategically placed electrodes.

Response amplitudes of the myogenic potential can be measured bilaterally and compared. This is known as the interaural amplitude asymmetry ratio. Significant asymmetries (>40-50%) can indicate a peripheral pathology, such as inferior vestibular neuritis, vestibular schwannoma, or Ménière's disease. As cVEMP is usually only elicited at higher intensity levels (>75 dBnHL), a lower threshold and/or high amplitude cVEMP can be indicative of a third window syndrome, such as superior semicircular canal dehiscence. Significantly prolonged latencies can be indicative of multiple sclerosis, vestibular schwannoma, or sometimes vestibular migraine. Absent responses also have been reported in Ménière's disease and vestibular migraine.

The cVEMP evaluation can be performed in patients of all ages, including very young children, and can be conducted even in the presence of severe-to-profound sensorineural hearing loss. Bone conducted VEMP responses can be used in patients with conductive hearing loss, which can eliminate the usual air conduction stimulus delivery.

Ocular Vestibular Evoked Myogenic Response (oVEMP)

The oVEMP also has come into wider clinical use in recent years as an additional evaluation of the vestibulo-ocular reflex (VOR). The oVEMP specifically assesses the function of the utricle and superior branch of the vestibular portion of the eighth cranial nerve. In an oVEMP assessment, a stimulus is delivered through air, bone (preferred), or galvanic stimulation. This high intensity acoustic stimulus or vibratory tactile stimulus elicits a contralateral reflex response (contraction) of the inferior oblique muscle (under the eye), which can be recorded through strategically placed electrodes.

Similar to cVEMP, analysis of oVEMP examines the presence or absence of waveforms, the interaural amplitude asymmetry ratio, and latencies of the waveforms of interest. Absent responses, asymmetries ratios >35-50%, low thresholds, or prolonged latencies can indicate possible peripheral vestibular system pathology. Increased amplitudes are suggestive of irritative lesions like Ménière's disease, while increased amplitudes combined with reduced thresholds can suggest third window syndromes such as superior

semicircular canal dehiscence. Completely absent or significantly reduced amplitudes are often indicative of superior vestibular neuritis or other utricular or vestibular nerve pathology, but they also have been reported in Ménière's disease and Vestibular migraine.[36]

Video Head Impulse Test (vHIT)

The vHIT is a test of VOR function utilizing high velocity head thrusts, or impulses, that are delivered by the examiner. The vHIT assesses natural high frequency head rotations. During the vHIT evaluation, the patient wears lightweight recording goggles. The goggles have an accelerometer or rate sensor that measures head velocity, and a camera that records and measures eye movement velocity. This eye-to-head movement velocity ratio is known as gain and is one of the main analysis parameters of the vHIT test. For the test, the patient is asked to stare at a fixation target typically placed approximately one meter away. The examiner then delivers unpredictable, small, brief head impulses in varying planes of movement. The gain of eye to head movement is then calculated. Impulses are delivered in one of three planes: lateral (assessing the left and right horizontal/lateral semicircular canals), RALP (assessing the Right Anterior-Left Posterior vertical semicircular canals), and LARP (assessing the Left Anterior-Right Posterior canals), thus giving gain measurements for each of the six semicircular canals.

In a perfectly functioning system, the VOR should cause the eyes to move in an equal magnitude, opposite direction movement compared to the head, giving an overall gain ratio of 1. However, in pathological systems the VOR may not be working ideally, resulting in an eye lag. If asked to focus on a target the eyes often will move with the head rather than opposite the head. This often causes the eyes to make quick "catch up" movements, called saccades, to refocus the retina on the target, and leads to a low gain calculation on the side of the lesion. Sometimes these saccades occur during the head movement (called covert saccades), and sometimes they occur after the head movement has stopped and are easier to see with the naked eye (called overt saccades). The presence of catch-up saccades is another analysis parameter utilized to identify a vestibular hypofunction in vHIT testing.

A newer paradigm of the vHIT called Suppression Head Impulse Test (SHIMP) was described in 2016 to overcome the challenge of VOR gain calculations in the presence of covert saccades. In this version of the test, the target is head-fixed rather than earth-fixed and moves with the head thrust. Overt saccades in this test paradigm indicate adequate/residual vestibular function, in contrast with the traditional head impulse test in which the presence of saccades indicates vestibular dysfunction.

Overall low gains and/or catch-up saccades in one or more canals indicate dysfunction of that specific semicircular canal. Assessment with vHIT, along with cVEMP and oVEMP can give a picture of the entire peripheral vestibular system - all six semicircular canals, both otolith organs (utricle and saccule), and both superior and inferior branches of the vestibular nerve. However, these assessments do not give significant information on central system function or overall functional balance, and therefore should be utilized as part of a test battery.

Computerized Dynamic Posturography

Posturography platforms are used to assess more complex integrated functioning of the balance system. Over the past 3 decades various types of computerized dynamic posturography systems have been developed. These systems use computer-controlled moveable platforms and a sway-referenced surrounding visual environment[37] or a virtual environment. The platform can perform a variety of complex motions, and the visual surroundings can move while the patient's anterior-posterior sway is tracked using pressure-sensitive strain sensors under the platform.[38] The visual surrounding and platform may operate together or independently, and the system can create visual distortions or totally eliminate visual cues.

The typical test protocol evaluates sensory organization through 6 test procedures and movement coordination through a variety of sudden platform movements. Balancing strategies and responses are assessed using both the sensory organization and movement coordination test batteries. Dynamic posturography provides a great deal of information about total balance function that cannot be obtained from vestibular ocular tests alone. Dynamic posturography is also valuable in distinguishing organic from nonorganic disequilibrium, an asset that is particularly valuable in some cases of alleged impairment.[39] In those cases, certain aphysiologic patterns can be identified. However, not every patient with an aphysiologic pattern on the posturography is a malingerer, with some studies finding significant rates of VNG abnormalities in subjects with nonorganic sway patterns.[40,41]

Dynamic posturography can help determine sensory patterns of dysfunction (e.g. vestibular, visual, or somatosensory), but is generally considered a more functional, rather than diagnostic, site of lesion assessment.

Vestibular System Impairment Rating

Permanent impairment can result from defects of the peripheral vestibular ~~(labyrinthine) mechanism~~system (sensory apparatus and nerve) and its central connections. The defects are evidenced by loss of equilibrium produced by disturbance of, or loss of vestibular function.

Complete loss of vestibular function may be unilateral or bilateral. When the loss is unilateral, adequate central nervous system compensation may or may not occur. With total bilateral loss of vestibular function, equilibrium is totally dependent on the kinesthetic and visual systems, which usually are unable to compensate fully for movement or ambulation. Depending on the ability to perform ADLs, the percentage of permanent impairment of the whole person may range from 0% to 58%.

Assessment of the impairment relies on serial medical evaluations combined with the appropriate vestibular tests that should be interpreted by a qualified vestibular clinician.

Disturbances of vestibular function can present with a variety of symptoms, with external vertigo/vestibulovisual symptoms being the most frequent indicator of peripheral vestibular dysfunction.

Disturbances of vestibular function can present with a variety of symptoms, with external vertigo/vestibulovisual symptoms being the most frequent indicator of peripheral vestibular dysfunction.

~~Disturbances of vestibular function are evidenced by vertigo (vestibular disequilibrium) as defined earlier in this chapter. Lightheadedness and abnormalities of gait not associated with vertigo are not defined here as being disturbances of vestibular function, although such symptoms may occur in some patients with slowly developing or long-standing vestibular dysfunction.~~

~~Vertigo may be accompanied by~~Other symptoms include varying degrees of nausea, vomiting, headache, immobility, ataxia, ~~and nystagmus. Movement may increase the vertigo and the accompanying signs and symptoms.~~visual lag ,and movement induced blur. Peripheral vestibular (labyrinthine) disorders ~~are often~~can be associated with hearing loss and tinnitus. Vestibular disorders may result in temporary or permanent impairments. Evaluation of vestibular impairment should be performed when the condition is stable and maximum adjustment has been achieved, which generally is considered to occur months after resolution of the disease or injury.~~[9,11]~~ [9, 32] Episodic vestibular disorders such as vestibular migraine, Ménière's disease, or BPPV can cause significant impairment depending on the frequency of their occurrence, despite normal function between episodes.

In addition, vestibular disorders may be associated with psychiatric comorbiditiy when chronic or inappropriately treated. Depression and anxiety are diagnosed frequently, with some reports of increased risk of suicide. It is important to consider those issues during the assessment of impairment by separately gauging the impact of the anxiety and/or depression on the subject's functioning (please refer to Chapter 14, Mental and Behavioral Disorders, within the *AMA Guides*.

The classification in Table 11-4 has been developed for evaluation of those individuals with ~~permanent~~ disturbances of the vestibular mechanism. The impairment ratings reflect the severity of the permanent impairment and the ability of the individual to perform ADLs. Although symptoms may be intermittent, the examiner needs to gauge functioning during episodes with exacerbations. In many cases, it is possible to document an impairment in the ~~balance system. In others, the subjective complaint of dizziness may be the only abnormality. When present, vertigo and other conditions of disequilibrium~~vestibular system. In others, the subjective complaint of dizziness may be the only abnormality. Many patients who experience vestibular or other balance disturbances are left with persistent symptoms described using various terms, including space motion disorder, phobic postural vertigo, psychophysiological vertigo, chronic subjective dizziness, and most recently, Persistent Postural-Perceptual Dizziness (PPPD). These symptoms often represent a maladaptive response to an initial event – such as a vestibular insult – in which labyrinthine signals were unreliable and subsequently ignored or misinterpreted. As a result, patients may develop maladaptive patterns of integrating sensory inputs from the visual, vestibular, and somatosensory systems. This dysfunction often manifests as heightened postural responses, increased postural awareness, and excessive motion sensitivity, which can be severe. Associated symptoms frequently include anxiety, panic, phobic behaviors, disorientation, difficulties with concentration and memory, psychological withdrawal, and depression. Thus, when present, vestibular symptoms may cause an additional disability that is not necessarily reflected in the impairment rating because the impairment rating reflects only the effect on ADLs. Quality of life measures such as the self-reported questionnaire, the Dizziness Handicap Inventory (DHI), have been used clinically to try to identify and quantify the effects of dizziness.~~14~~[37] Many studies have shown the robustness of the DHI as a patient-reported outcome measure that captures the degree of handicap, including a recent meta-analysis and a recent reanalysis of its validity using item-response theory in episodic vestibular disorders.[42,43]

Another patient-reported outcome measure is the Vestibular Migraine Patient Assessment Tool and Handicap Inventory (**VM-PATHI**)[44], which is a valid and reliable measure of disease severity in vestibular migraine. Patient-reported outcome measures correlate with treatment response, comorbidities and cognitive dysfunction in some of the chronic and

episodic vestibular disorders. While they cannot be used to calculate impairment, the clinician can use those metrics to gauge clinical severity and to look more carefully for objective signs of vestibular dysfunction.

Table 11-4

Criteria for Rating Impairments due to Vestibular Disorders[a]

CLASS	CLASS 0	CLASS 1	CLASS 2	CLASS 3	CLASS 4
WHOLE PERSON IMPAIRMENT RATING (%)	**0**	**1%–9%**	**11%–27%**	**30%–42%**	**45%–58%**
SEVERITY GRADE (%)		**1 3 5 7 9**	**11 15 19 23 27**	**30 33 36 39 42**	**45 48 51 54 58**
HISTORY[b]	Symptoms or signs of vestibular ~~disequilibrium~~dysfunction present without objective findings	Symptoms or signs of vestibular ~~disequilibrium~~dysfunction consistent with objective findings Activities of daily living: requires assistance for complex activities, eg, riding a bike or certain types of demanding activities related to the individual's work, eg, working on girders or scaffolds	Symptoms or signs of vestibular ~~disequilibrium~~dysfunction consistent with objective findings Activities of daily living cannot be performed without assistance except for simple activities, eg, self-care, some household duties, walking, and riding in a motor vehicle operated by another person	Symptoms or signs of vestibular ~~disequilibrium~~dysfunction consistent with objective findings Activities of daily living cannot be performed without assistance except for self-care	Symptoms or signs of vestibular ~~disequilibrium~~dysfunction consistent with objective findings Activities of daily living cannot be performed without assistance except for self-care not requiring ambulation ***and*** home confinement is necessary
PHYSICAL EXAM	No confirmable findings	No confirmable findings or mildly abnormal gait, Romberg, or other findings	Unsteady gait; abnormal Romberg	Difficulty walking without assistance	Difficulty standing or walking without assistance
DIAGNOSTIC OR OTHER OBJECTIVE FINDINGS	No confirmable diagnostic findings	Abnormal findings on ~~ENG or VNG, such as positional nystagmus or abnormal caloric response,~~vestibular testing may be present	Abnormal findings on ~~ENG or VNG, such as positional nystagmus, abnormal caloric response,~~vestibular testing or abnormal central nervous system signs, or abnormal sway or	Moderately abnormal findings on ~~ENG or VNG, such as positional nystagmus, abnormal caloric response,~~vestibular testing or abnormal	Severely abnormal findings on ~~ENG or VNG, such as positional nystagmus, abnormal caloric response,~~vestibular testing or abnormal

		Electrocochleography may be abnormal	abnormal sensory tests may be present on dynamic posturography	central nervous system signs ***and*** abnormal sway or abnormal sensory tests may be present on dynamic posturography Brain MRI abnormalities may be present - ***and*** abnormal sway or abnormal sensory tests may be present on dynamic posturography Brain MRI abnormalities may be present	central nervous system signs ***and*** abnormal sway and severe abnormalities on all 6 conditions tested with the sensory organization portion of dynamic posturography Brain MRI abnormalities may be present - ***and*** Brain MRI abnormalities may be present

[a]ENG indicates electronystagmography; VNG, videonystagmography; and MRI, magnetic resonance imaging.

[b]Key factor.

The outcome of the symptoms evaluation, the physical signs, and the ~~laboratory~~vestibular test results should be correlated, with emphasis on the validated symptoms—those confirmed by the history, physical findings, and test results ~~of laboratory tests~~—as best representing the subject's true state of impairment. The evaluator should consider that while the *AMA Guides* does not consider occupation in the impairment rating, ~~vertigo~~vestibular symptoms may cause additional disability for people working in hazardous job situations. Persons who lose their balance even momentarily may severely injure themselves or others when working around sharp surfaces, rotating equipment, driving a forklift, working on ladders or scaffolding, or functioning in other similar circumstances. The evaluator can report any appropriate work restrictions that are medically necessary.

Not all symptoms and findings have equal impact. Consideration should be given to symptoms and the physical examination in the same way that the VNG findings are listed as moderately or severely abnormal. For example, oscillopsia and drop attacks are severe manifestations that might warrant a higher class of impairment than some cases of vertigo, non-specific dizziness, or spatial disorientation. The same would apply for bilateral abnormalities seen on head impulse testing versus unilateral abnormalities, spontaneous downbeat nystagmus, or significant skew deviation. These physical examination findings suggest more severe impairment than positional nystagmus, which is suggestive of BPPV.

Many vestibular disorders are treatable, and the rating should be established only after comprehensive treatment by a multidisciplinary team has resulted in maximal improvement.

To use Table 11-4, follow these steps:

1. Place the individual in the appropriate Class based on the key factor, which is History. Begin by selecting the middle Severity Grade number in that Class. The Classes have discrete numbers. They are not a range.
2. Assess the correct Class for the Physical Examination findings on this patient. If the Class selected is 1 Class higher than the Class selected from step 1 (History), move the Severity Grade up 1 number higher **within** the originally selected Class. If the Physical Examination is 2 levels higher than that assigned for History, move the Severity Grade up 2 levels within the History-based Class. If the Class from the Physical Examination is in a lower Class than that selected from the History, move the Severity Grade lower **within the** History-based Class.
3. Assess the Class level of the patient using the Diagnostic or Other Objective Findings. Using the number you established in step 2, move again up or down if the

Class is lower than or higher than the Class originally selected by the History. You may not move out of the Class you have originally selected from your History.

4. If you begin in Class 4, you may move up based on the number of non-key factors also found in Class 4.

For example, if the patient were found to be in Class 2 originally by History, you would begin at 19% as the middle number in Class 2. If the Physical Exam were in Class 4, you would move up to 27%, staying within the Class 2 but moving up 2 Severity Grades. If the Diagnostic or Other Findings were in Class 3, it is 1 Class higher than the original Class but it would not change your rating since you cannot exceed 27% in Class 2. However, if the Diagnostic or Other Findings were in a Class 1, you would move down 1 Severity Grade and the final Impairment Rating would be 23%.

Class 0

0% Impairment of the Whole Person

Example 11-4: Benign Paroxysmal Positional Vertigo

Subject: 70-year-old man.

History: Retired physician. Three-week history of vertigo that occurs whenever he rolls over to his right side when lying in bed. He describes the feeling as a spinning sensation that lasts a few seconds. He also experiences vertigo when he turns to the right when shopping at the grocery store. No nausea or vomiting. His physician did a Dix-Hallpike test, which showed rotary/upbeat nystagmus with the right ear down; he was treated with the Epley maneuver to reposition otoconia. Currently he is asymptomatic with no disruption of ADLs.

Current Symptoms: Asymptomatic; the dizziness has not recurred; no disruption of ADLs.

Physical Exam: Normal.

Clinical Studies: Dix-Hallpike test: no rotary/upbeat nystagmus at the present time.

Diagnosis: Benign paroxysmal positional vertigo.

Impairment Rating: 0% impairment of the whole person.

Comment: Treatment to be repeated as necessary.

Class 1

1% to 9% Impairment of the Whole Person

Example 11-5: Vestibular Neuronitis

Subject: 50-year-old-woman.

History: Sudden onset of severe vertigo, nausea, and vomiting. No history of upper respiratory tract infection, fever, cough, or chills. Confined to bed for 3 days. Hearing normal; no tinnitus. Treated with vestibular suppressors. Gradual, slow recovery of ability to ambulate. Able to return to work as a secretary in 2 weeks but unable to walk in the dark since onset of her spell of vertigo 6 months ago.

Current Symptoms: Can perform ADLs without assistance. Slightly unsteady when fatigued. Does not tolerate rocking motion (sailboat) without visual fixation of horizon. Unable to ride bicycle, but can drive automobile at night.

Physical Exam: Abnormal Romberg but gait and tandem gait are normal. No residual nystagmus.

Clinical Studies: vHIT and VNG with caloric studies: no vestibular function of right ear. Other neuro-otologic findings: within normal limits. Audiogram: normal hearing bilaterally.

Diagnosis: Vestibular neuronitis, probably viral, with total loss of vestibular function, right ear.

Impairment Rating: 5% impairment of the whole person.

Comment: Class 1 impairment, with mild loss of function. History places this individual in class 1. The rating begins at 5%. The mildly abnormal physical exam findings leave the rating number unchanged. The objective findings on VNG testing are in class 1. (If the physical exam findings were in class 3, these findings would have moved the rating up the scale to 9%—the highest rating in class 1—but would not have moved the patient to class 2 or 3, because once you have chosen the class level using the history as your guide, you cannot move out of that class into another.)

Class 2

11% to 27% Impairment of the Whole Person

Example 11-6: Vestibular Disorders

Subject: 40-year-old woman.

History: Nurse; 3-month history of progressive hearing loss in left ear, increased difficulty with gait, some loss of balance with falling to the left, and slurred speech when fatigued.

History of hypertension, controlled with β-blockers. Audiogram showed normal hearing in right ear, 80-dB sensorineural hearing loss in left ear. Tympanograms were type A bilaterally. Auditory brain-stem response showed absence of wave V in left ear. The ~~E~~VNG showed absent ~~caloric~~vestibular response in the left ear. A magnetic resonance image (MRI) with gadolinium showed large left cerebellopontine angle (CPA) mass involving the left internal auditory canal. A 4-cm acoustic neuroma with secondary brain-stem compression was removed via the translabyrinthine approach. Postoperative ophthalmologic exam revealed exposure keratopathy of the left eye. Left lateral canthoplasty with insertion of gold weights in left upper eyelid was performed, plus a cross-face sural nerve graft to the left side of the face.

Current Symptoms: Preoperatively active. Now walks with some difficulty with a broad-based gait. Has fallen twice since surgery. Can perform self-care and limited household activities; unable to drive a car.

Physical Exam: Left-sided facial paralysis. Total hearing loss in left ear. Cerebellar tremor worse in the left upper extremity than in the left lower extremity. Somewhat unsteady gait with moderately abnormal Romberg.

Clinical Studies: Total loss of hearing and of vestibular function, left ear. No residual tumor, but changes in brain-stem area noted on MRI. Electroencephalogram: no evidence of epileptiform activity. Gait and balance scores abnormal for age.

Diagnosis: Large left acoustic neuroma with postoperative total left auditory and vestibular impairments, left-sided facial nerve paralysis and mild to moderate unsteadiness.

Impairment Rating: 23% impairment due to vestibular disorders; combine with appropriate ratings for other impairments to determine whole person impairment (see Combined Values Chart in the Appendix).

Comment: The history places this individual in class 2. The rating begins at 19%. The class 2 physical exam findings leave the rating number unchanged. The objective findings, which are in class 3, move the rating toward the top of category 2 or 23%. Additional ratings would be combined for facial nerve, loss of hearing, and tremor in the upper extremities. Care must be taken not to rate the same impairment of ADLs twice when combining multiple impairments.

Class 3

30% to 42% of Whole Person Impairment

Example 11-7: Unilateral Vestibular Injury

Subject: 48-year-old man.

History: Victim of a mine explosion 3 years ago, with complete loss of hearing and vestibular function in left ear. Chronic dizziness with nausea and weight loss developed from severe vestibular injury. Very unsteady gait. Needs a cane or walker to ambulate. Uses high-dose vestibular suppressants to counteract nausea and maintain his body weight. Vestibular rehabilitation exercises did not provide any benefit. Underwent labyrinthectomy and vestibular nerve section on left side without relief of symptoms.

Current Symptoms: Unable to walk without physical assistance or assistive device. Minimal nausea as long as he uses vestibular suppressants. Riding in a car causes severe dizziness with nausea.

Physical Exam: Very unsteady, broad-based gait. Thin legs with poor muscle tone.

Clinical Studies: The VNG shows absent caloric response in left ear. and the vHIT shows low gains in the left lateral and posterior semicircular canal. vOCR responses are reduced and VEMP responses are absent on the left side. Profound sensorineural hearing loss is also present in the left ear. There are severe abnormalities in all 6 positions tested with sensory organization portion of dynamic posturography. MRI is normal.

Diagnosis: Severe, uncompensated unilateral vestibular injury.

Impairment Rating: 39% impairment of the whole person.

Comment: History places this individual in class 3. The rating begins at 36%. The class 3 physical exam findings leave the rating number unchanged. The objective findings regarding dynamic posturography, which are in class 4, move the rating up 1 number to 39%.

Example 11-8 Bilateral vestibular hypofunction/Vestibulotoxicity

Subject: 55-year-old female.

History: History of ovarian cancer that was treated with platinum-based chemotherapy drugs (Cisplatin), which are known to be vestibulotoxic. Since completing chemotherapy she has been very unsteady when walking, especially in dimly lit environments or on uneven surfaces. No report of spinning vertigo. She underwent a course of exercise-based vestibular rehabilitation. She was better but described an inability to see clearly when she walked and described oscillopsia. She had a couple of falls that happened following rapid head turns. She also had a decline in her hearing, and she reported bilateral tinnitus since she finished chemotherapy.

Current Symptoms: Unable to walk without physical assistance and use of a walker. No reported nausea.

Physical Exam: Very unsteady, broad-based gait. Thin legs with poor muscle tone. No spontaneous nystagmus. Bedside head impulse testing positive bilaterally for overt corrective saccades. Romberg positive. Tandem Romberg positive.

Clinical Studies; VNG showed bilaterally absent caloric responses (even with ice). High frequency (normal to moderately severe sloping) sensorineural hearing loss, bilaterally. Sinusoidal Harmonic Acceleration (SHA) testing showed decreased gains at all frequences. vHIT testing revealed abnormally low VOR gains in both the horizontal and vertical canals, bilaterally below 0.6 with overt saccades in all canals. Sensory Organization Testing (SOT) of the Computerized Dynamic Posturography revealed falls in conditions 2,3, 5 & 6, indicating a visual vestibular dysfunction pattern.

Audiogram showed bilateral moderately severe sensorineural hearing loss with right ear thresholds of 60dB at 500 Hz, 1000 Hz, 2000Hz and 3000Hz, and thresholds in the left ear at 40dB at 500Hz and 1000Hz and 60dB on 2000Hz and 3000Hz.

Diagnosis: Severe, uncompensated, bilateral vestibular hypofunction secondary to vestibulotoxicity.

Impairment Rating: 39% of the whole person

Comment: History places this individual in Class 3. The rating begins at 36%. The Class 3 physical examination findings leave the rating number unchanged. The objective findings regarding dynamic posturography and the severely abnormal findings on vestibular testing are in Class 4, moving the rating up one number to 39%

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