Entitlement Eligibility Guideline

Vertiginous Disorders

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ICD-11 **code:** AB31.7

VAC medical code: 38620 Vertiginous disorders

Definition

Vertigo is an illusion or sensation of rotary movement associated with difficulty in balance, gait, and navigation. The affected individual feels that they are moving in relation to the environment, or that the environment is moving in relation to them.

For Veterans Affairs Canada (VAC) purposes, vertigo, HL (hearing loss), and/or tinnitus may present as part of the symptom complex of a diagnosed medical condition, or they may present as a primary stand-alone diagnosed medical condition. In those presenting with symptoms of vertigo, HL, and/or tinnitus, but with a known diagnosed cause (e.g. Meniere's disease), these symptoms are included in entitlement and assessment of the medical condition. Prior to adjudicating the entitlement and assessment of vertigo, HL, and/or tinnitus, or a diagnosed medical condition that may cause these symptoms, a close review of previously entitled medical conditions with potentially overlapping symptoms is required.

Diagnostic standard

A diagnosis of a vertiginous disorder by a qualified physician (ear, nose, and throat specialist [ENT]/otolaryngologist, neurologist, family physician) or nurse practitioner is required.

Any diagnostic investigations confirming the diagnosis should be provided.

For some diagnoses, the episodes of vertigo may be short-lived and intermittent; these episodes may or may not recur after initial therapy. For VAC entitlement purposes, a permanent disability of chronic vertigo is established when the condition has lasted six months or longer; a single incident or complaint of vertigo is not sufficient for entitlement purposes. The condition must be permanent or recur on a chronic basis. Signs and symptoms are expected to persist despite medical attention, although the symptoms may increase or decrease in severity or frequency.

Note: Dizziness is not equivalent to vertigo. The term dizziness may refer to symptoms of vertigo, lightheadedness, faintness/presyncope, any gait unsteadiness and psychophysiologic symptoms such as floating or spinning inside.

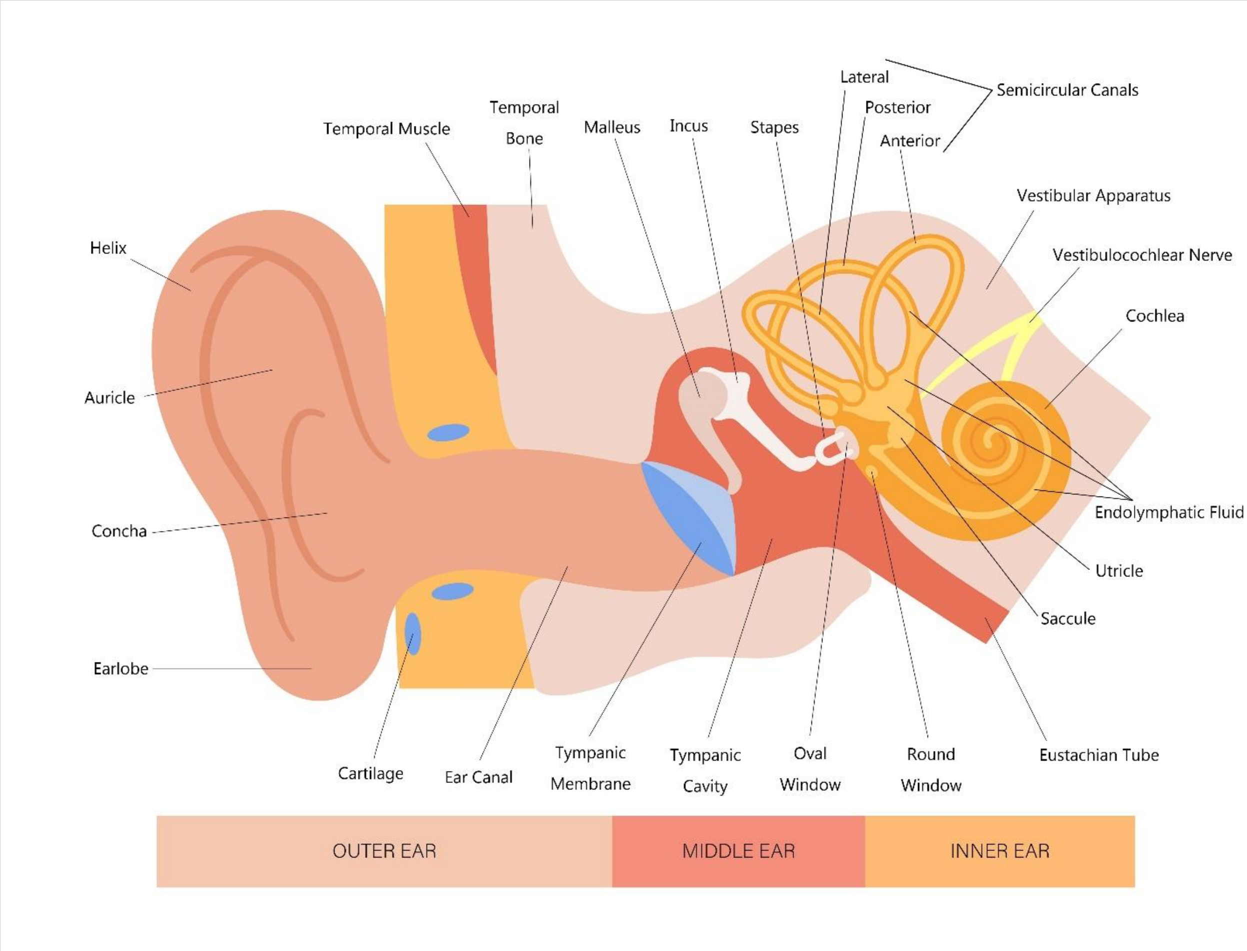
Vertigo arises from disorders of either the inner ear, the nerves serving the inner ear, or the parts of the brain interpreting the signals from the inner ear. Only conditions resulting in true vertigo are addressed in this entitlement eligibility guideline (EEG).

Anatomy and physiology

The end organs responsible for movement and balance perception are in the labyrinthine system within the inner ear (Figure 1: Ear anatomy). They are filled with endolymphatic fluid and include:

- Three semicircular canals, at right angles to one another. The fluid in the canals moves with head movement, and the fluid stimulates the sensory hair cells. The stimulus is interpreted by the brain as angular movement. Disorders here, or in the central nervous system areas that process signals arising here, can produce a spinning sensation.
- Two otolith organs, the utricle and the saccule which contain crystals (otoliths, octonia, otoconia) that rest by gravity on the sensory areas. The stimulus is interpreted by the brain as the linear motion. Disorders here, or in the central nervous system areas that process signals arising here, can produce a tilting or floating sensation.
- A vestibular system in each ear with the brain comparing information from each side. Differences in the signals in one side as compared to the other is perceived as motion. If a disease process causes abnormal signals, this can be perceived as motion, even when not moving.

Figure 1: Ear anatomy



An illustration of the human ear, clearly labeling its three main sections and associated structures. **Outer ear:** Includes visible parts such as the helix, auricle, concha, earlobe, and external ear canal. **Middle ear:** Contains the tympanic membrane (eardrum), three small bones called ossicles (malleus, incus, stapes), and the eustachian tube. **Inner ear:** Features the semicircular canals, cochlea, utricle and saccule – all filled with endolymphatic fluid. Associated nerves are the cochlear and vestibular nerves. Between the middle and inner ear is the oval window and the round window. Source: Veterans Affairs Canada (2024).

Sensory information from the vestibular labyrinth travels through vestibulocochlear nerve/cranial nerve VIII to the vestibular nuclear complex in the brainstem. Fibers from different receptor organs terminate in different vestibular nuclei at the pontomedullary junction of the brainstem and in the cerebellum. From the vestibular nuclei, second-order neurons make important connections to the vestibular nuclei of the other side, to the cerebellum, to motor neurons of the spinal cord, to autonomic nuclei in the brain stem, and to the nuclei of the oculomotor system. These fibres may also reach the cerebral cortex bilaterally. These connections enable the brain to respond with appropriate corrective actions to the nervous and muscular systems to ensure that balance and spatial awareness are maintained.

The cerebellum is located at the back of the brain and is responsible for motor coordination, posture maintenance and balance control. Conditions affecting the cerebellum can result in symptoms of imbalance, gait disturbances, uncoordinated movements (ataxia), dizziness, vertigo, speech problems (dysarthria), tremors or shaking, and visual problems (diplopia, nystagmus).

Clinical features

Vertigo can be caused by a dysfunction of either the peripheral or central vestibular apparatus. The peripheral vestibular apparatus includes the vestibular system/semicircular canals and the vestibulocochlear nerve/cranial nerve VIII. Peripheral vertigo is more severe and more likely to be associated with hearing loss and tinnitus. It often leads to nausea and vomiting. Nausea associated with vertigo is included in the entitlement and assessment of any vertiginous disorder.

The central vestibular apparatus includes the brain (most often the cerebellum or temporal lobes) and the brainstem. Central vertigo is less severe than peripheral vertigo and is often associated with other signs of central nervous system disease.

The presence of persistent nystagmus in someone suffering from the symptom of dizziness indicates vertigo as the cause of dizziness. Nystagmus is involuntary movements of the eye with slow movement in one direction and a quick phase that brings the eye back to the original position. Nystagmus can have other origins.

With respect to differences in presentation of vertiginous disorders, there is an increased incidence of:

- benign paroxysmal positional vertigo (BPPV) (2-3:1) in females as compared to males
- persistent and disabling symptoms of ‘mal de debarquement’ lasting for weeks, months, or years in females as compared to males
- migraine (2-3:1) in females as compared to males
- vestibular migraine has a further increased incidence (5:1) in females as compared to males
- motion sickness (2:1) in females as compared to males. Females are more likely to experience increased severity and frequency of symptoms compared to males.

When comparing males and females, there are no differences in rates of occurrence, presentation or symptoms of acute unilateral vestibulopathy.

Entitlement considerations

Section A: Causes and/or aggravation

For VAC entitlement purposes, the following factors are accepted to cause or aggravate the conditions included in the Definition section of this EEG, and may be considered along with the evidence to assist in establishing a relationship to service. The factors have been determined based on a review of up-to-date scientific and medical literature, as well as evidence-based medical best practices. Factors other than those listed may be considered, however consultation with a disability consultant or medical advisor is recommended.

The timelines cited below are for guidance purposes. Each case should be adjudicated on the evidence provided and its own merits.

Factors

1. Having chronic vertigo of an **idiopathic** (unknown) origin despite investigations.

2. Being treated with a **medication** from the specified list of medications below during or just prior to clinical onset or aggravation of chronic vertigo. The medications include, but are not limited to, the following:

    - Parenteral aminoglycoside antibiotic:

        - gentamicin
        - streptomycin
        - kanamycin
        - amikacin
        - netilmicin
        - tobramycin.

    - Intravenous administration of:

        - ethacrynic acid
        - bumetanide
        - vancomycin
        - erythromycin.

    - Chemotherapeutic agents:

        - nitrogen mustard

- bleomycin
- cisplatin
- a-difluoromethylornithine
- vincristine
- vinblastine
- misonidazole
- 6-amino nicotinamide
- carboplatin.

Note:

- Individual medications may belong to a class of medications. The effects of a specific medication may vary from the class. The effects of the specific medication should be considered.
- If it is claimed a medication resulted in the clinical onset or aggravation of chronic vertigo the following must be established:

  - the medication was prescribed to treat an entitled condition
  - the individual was receiving the medication at the time of the clinical onset or aggravation of chronic vertigo
  - the current medical literature supports the medication can result in the clinical onset or aggravation of chronic vertigo.

3. Having **physiologic vertigo** at the time of clinical onset or aggravation of chronic vertigo. This includes common disorders such as motion sickness, including seasickness. It is attributed to a mismatch in the sensory input from the vestibular and visual systems. This happens when the vestibular system senses movement but the visual system does not. The symptoms often resolve.

4. Inability to obtain **appropriate clinical management** of chronic vertigo.

For the following factors: Vertigo may be a symptom of a primary stand-alone medical disease or disorder. If the medical evidence indicates that vertigo is caused by one of the following entitled primary medical conditions, vertigo is included in the entitlement and assessment of the primary condition.

5. Having **direct trauma to the inner ear with structural damage of the inner ear** prior to clinical onset or aggravation of chronic vertigo. Causes would include, but are not limited to:

   - penetrating injury
   - blast injury.

6. Having **inner ear barotrauma (IEBt)** at the time of clinical onset or aggravation of chronic vertigo. IEBt can result from:

- diving underwater
- pressure changes associated with heavy lifting.

7. Having **inner ear surgery** prior to clinical onset or aggravation of chronic vertigo. Vertigo can occur up to several years after the development of the primary condition.

8. Having a **perilymphatic fistula** at the time of clinical onset or aggravation of chronic vertigo. Causes of a perilymphatic fistula include, but are not limited to:

    - head injury
    - blast injury
    - barotrauma
    - penetrating injury of the ear
    - middle ear surgery
    - pressure changes associated with heavy lifting.

    A perilymphatic fistula is an abnormal communication between the perilymph filled inner ear and any adjoining structure; most commonly these occur at the oval or round windows. The symptoms can include unilateral sudden hearing loss, tinnitus, and vertigo occurring at the time of injury. The condition is rare and diagnosis is based on either observance of fluid leakage or computed tomography (CT) and/or magnetic resonance imaging (MRI) scanning.

    Note: For VAC entitlement purposes, the diagnosis of perilymphatic fistula should be provided by a treating ear, nose and throat (ENT) specialist/otolaryngologist.

9. Having **decompression sickness** at the time of clinical onset or aggravation of chronic vertigo. Decompression sickness can occur with a rapid ascent during underwater diving or ascent to high altitude in a poorly pressurized aircraft cabin. Vertigo due to decompression sickness would start at the time of the injury. In the majority of cases, complete resolution of symptoms occurs.

10. Having sustained a **labyrinthine concussion** at the time of clinical onset or aggravation of chronic vertigo. Direct trauma to the labyrinth can result from a blow to the head or abrupt changes of head motion. Damage to the labyrinth can result in symptoms of vertigo, nausea, and imbalance. The symptoms are at their peak at the time of the injury and may improve over several months depending on the extent of the injury.

11. Having a **head injury with temporal bone fracture** within a few weeks prior to clinical onset or aggravation of chronic vertigo. Transverse fractures of the temporal bone may cause injury to the cochleovestibular nerve resulting in

vertigo. Fractures of the temporal bone may also result in vertigo as a result of ossicular chain injuries.

12. Having **benign paroxysmal positional vertigo (BPPV)** at the time of clinical onset or aggravation of chronic vertigo. This is by far the most common cause of pathologic vertigo and results from crystals from the otoliths floating in the posterior semicircular canal.

    Classically the symptoms last only seconds and are brought on by head movement (for example head tilt, rolling over in bed). There may be some nausea. There are no ear symptoms.

    The diagnosis is based on the history and confirmed by the Dix-Hallpike maneuver and the supine roll test. When positive, these tests cause nystagmus in a direction corresponding to the involved canal. Most cases do not have a defined cause. BPPV usually subsides within weeks to months but may recur after months or years.

    BPPV can occur after a head injury. This is not due to a traumatic brain injury but rather to disturbance of otolithic crystals. This type of BPPV occurs at the time of the head injury and is more likely to recur than BPPV of other etiologies.

13. Having **acute unilateral vestibulopathy (AUVP)**/acute labyrinthitis /acute vestibular neuronopathy, vestibular neuronitis, vestibular neurites, viral neurolabyrinthitis at the time of clinical onset or aggravation of chronic vertigo.

    This results from viral or post viral inflammation of the vestibular portion of the vestibulocochlear nerve/cranial nerve VIII. It presents with an acute onset of severe vertigo, nausea, vomiting and gait disturbance lasting several days to weeks. There may be associated hearing loss or tinnitus which indicates the more specific diagnosis of acute labyrinthitis.

    There may be a history of flu like illness for 7-10 days prior to onset. There is usually complete recovery.

14. Having **Meniere's disease** at the time of clinical onset or aggravation of chronic vertigo. Meniere's disease presents with a combination of vertigo, hearing loss, tinnitus and/or aural fullness. The episodes are spontaneous. The vertigo may last for several days. The hearing loss, while worse with episodes, can remain diminished even between episodes.

15. Having **migraine** at the time of clinical onset or aggravation of chronic vertigo. Migraine can cause recurrent chronic vertigo. The diagnosis is based on history. Some, but not necessarily all episodes of vertigo are accompanied by migraine pattern type headache and/or aura and photophobia.

16. Having a **moderate or severe** **traumatic brain injury (TBI)** at the time of onset or aggravation of chronic vertigo. It is important that timelines are established. Mild TBI is not considered to be a causal factor for chronic vertigo.

    Note: If considering entitlement for vertigo due to TBI, consultation with a disability consultant or a medical advisor is recommended.

17. Having **otosclerosis** at time of clinical onset or aggravation of chronic vertigo. Otosclerosis is a primary disorder of the labyrinthine capsule characterized by new bone formation and often involving the footplate of the stapes. Vertigo is reported in 10% of cases.

18. Having **Cogan's syndrome** at time of clinical onset or aggravation of chronic vertigo. Cogan's syndrome is a rare autoimmune disorder with episodes of hearing loss, tinnitus and/or vertigo. It is usually accompanied by eye signs of interstitial keratitis, scleritis and conjunctivitis (eye redness, pain, photophobia, and blurred vision).

19. Having **cerebellopontine-angle tumors including vestibular schwannoma/acoustic neuroma** at time of clinical onset or aggravation of chronic vertigo. In vestibular schwannoma, minor disturbances of balance are common, resulting in unsteadiness while walking. These episodes may last several seconds, or minutes to hours, and vary in intensity.

20. Having one of the below **vascular insufficiencies** at the time of clinical onset or aggravation of chronic vertigo:

    - **Atherosclerotic emboli:** Sustaining an occlusion of the arterial supply of the vestibular system at the time of clinical onset or aggravation of chronic vertigo. Vertigo is abrupt in onset and frequently associated with nausea and vomiting. Occlusion of the arterial system to any portion of the vestibular system can produce transient or permanent symptoms.

      The length and type of symptoms depend on where the arterial occlusion occurs. Vertigo would rarely be the only symptom present when the ischemic event is in the brainstem; cerebellar lesions may present with severe vertigo only. Ischemia at the level of the labyrinth can produce vertigo and hearing loss.

    - **Wallenburg syndrome:** Sustaining an occlusion of the arterial supply of the medulla at the time of clinical onset or aggravation of chronic vertigo. This is a specific type of vascular insufficiency causing vertigo. It results from an occlusion of the blood supply to the medulla. It usually is due to atherosclerosis but can also be due to traumatic vertebral artery dissection.

- **Vertebral artery obstruction due to compression,** including rotational vertebral artery syndrome/bow hunter syndrome at the time of clinical onset or aggravation of chronic vertigo. This results in decreased blood supply of the hindbrain which can present with vertigo as well as other neurologic findings. The initial presentation may resemble recurrent transient ischemic attacks (TIA).

  The compression may be related to different diseases in the neck including large osteophytes, muscular hypertrophy, herniated discs and tumors. Compression may also follow physical trauma to the head and neck, including spinal surgery.

Note: If vertigo is claimed as a symptom of any of the above vascular insufficiencies, consultation with a disability consultant or medical advisor is recommended.

21. Having **multiple sclerosis** at the time of clinical onset or aggravation of chronic vertigo. Vertigo occurs in about 20% of established cases of multiple sclerosis.

    Note: If vertigo is the only presenting symptom, consultation with a disability consultant or medical advisor is recommended.

22. Having **Mal de debarquement/disembarkment syndrome** at the time of clinical onset or aggravation of chronic vertigo. In this condition, vertigo begins after cessation of movement, most commonly after getting off a watercraft. Vertigo can be characterized by a recurrent bobbing sensation, sensation of unsteadiness, and/or ground tilting. The symptoms generally resolve within 48 hours. Rarely can symptoms persist for months or years.

23. Having **herpes zoster oticus (Ramsay-Hunt syndrome)** at the time of clinical onset or aggravation of chronic vertigo. In this condition, herpes zoster of the facial nerve spreads to the vestibulocochlear nerve/cranial nerve VIII. Typical symptoms are one sided facial paralysis, ear pain, and vesicles in the ear canal. Hearing loss, tinnitus and vertigo can also occur.

24. Having **epilepsy** at the time of clinical onset or aggravation of chronic vertigo. Isolated vertigo is rarely due to epilepsy. Vertigo as part of seizure activity is most commonly associated with temporal lobe epilepsy; most cases have other seizure associated symptoms as well.

Note:

- At the time of publication, the health-related expert opinion and scientific evidence- indicates that chronic noise exposure does not cause vertigo.
- Persistent postural perceptual dizziness (PPPD) is a cause of chronic dizziness characterized by persistent non-vertiginous dizziness, unsteadiness (or both) that is present on most days for three months or more. As a non-vertiginous

disorder, it is excluded from this EEG as a causative factor for chronic vertigo. It is, however, considered a section “B” condition for all vertiginous disorders.

Section B: Medical conditions which are to be included in entitlement/assessment

Section B provides a list of diagnosed medical conditions which are considered for VAC purposes to be included in the entitlement and assessment of vertiginous disorders.

- Nystagmus associated with vertigo
- Ataxic gait
- Chronic nausea
- Persistent postural perceptual dizziness (PPPD)

Section C: Common medical conditions which may result, in whole or in part, from vertiginous disorders and/or their treatment

No consequential medical conditions were identified at the time of the publication of this EEG. If the merits of the case and medical evidence indicate that a possible consequential relationship may exist, consultation with a disability consultant or medical advisor is recommended.

Links

Related VAC Guidance and Policy:

- Meniere’s Disease – Entitlement Eligibility Guidelines
- Otosclerosis - Entitlement Eligibility Guidelines
- Traumatic Brain Injury – Entitlement Eligibility Guidelines
- Vestibular Schwannoma (Acoustic Neuroma) - Entitlement Eligibility Guidelines
- Pain and Suffering Compensation - Policies
- Royal Canadian Mounted Police Disability Pension Claims - Policies
- Dual Entitlement – Disability Benefits - Policies
- Establishing the Existence of a Disability - Policies
- Disability Benefits in Respect of Peacetime Military Service – The Compensation Principle - Policies
- Disability Benefits in Respect of Wartime and Special Duty Service – The Insurance Principle - Policies
- Disability Resulting from a Non-Service Related Injury or Disease - Policies

- Consequential Disability - Policies
- Benefit of Doubt – Policies

Appendix A: Dix-Hallpike maneuver for BPPV

Technique:[1]

- Begin with patient seated upright and examiner standing on patient's side.
- Examiner rotates head 45 degrees toward first side to be examined.
- Patient keeps eyes open and, while supporting head, examiner quickly moves patient to supine position so that the patient's head is extended past the examination table and is hanging about 20 degrees below horizontal plane with patient's chin slightly pointed upwards.
- Examiner checks patient's eyes for nystagmus observing:
    - latency period before onset
    - duration
    - direction.

Appendix B: Supine roll test

To be performed on patients with history consistent with BPPV but with negative Dix-Hallpike maneuver:[2]

- Patient lying on back with head in face-up position.
- Quickly rotate head to one side while examining for nystagmus.
- After any vertigo and nystagmus stops, slowly return head to neutral position.
- Then, quickly rotate 90 degrees to other side and again check for nystagmus.

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