ENTITLEMENT ELIGIBILITY GUIDELINE

VERTIGINOUS DISORDERS

MPC 00643
ICD-9 780.4 (for Benign Paroxysmal see 386.1)

DEFINITION

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

Vertigo as a **symptom** of a primary disease or disorder should be claimed as part of the disease or disorder.

Of the vertiginous disorders, BENIGN PAROXYSMAL POSITIONAL VERTIGO (BPPV) is one of the most common.

DIAGNOSTIC STANDARD

Diagnosis by a qualified medical practitioner, with investigations to support the diagnosis, are required.

While the diagnosis is made primarily on the basis of history and physical examination, current diagnostic evaluation may also include audiologic assessment, electronystagmography with caloric testing, a test to induce positional nystagmus, or an MRI/CT of the head with particular attention to the internal auditory canals so as to exclude either conditions. Investigations done to confirm the diagnosis must be provided.

ANATOMY AND PHYSIOLOGY

In addressing the vestibular system generally, the paired vestibular end organs lie within the temporal bones next to the cochlea. Each organ consists of three semicircular canals that detect angular acceleration and two otolith structures, the utricle and sacule, that detect linear acceleration (including gravitational). These organs possess hair cells that act as force transducers, converting forces associated with head acceleration into afferent nerve impulses. The hair cells of the three semicircular canals are embedded in a gelatinous mass called the cupula. Movement of the head causes the endolymph

to flow either toward or away from the cupula. The hair cells of the utricle and sacule are concentrated in an area called the macule. The hair cells are embedded in a membrane that contains calcium carbonate crystals or otoliths. Linear accelerations of the head combines with the linear acceleration of gravity to distort the otolith membrane, bending the underlying hair cells and modulating the activity of the afferent nerve terminals at the base of the hair cells.

The nerve fibers travel in the vestibular portion of the eight cranial nerve. Fibers from different receptor organs terminate in different vestibular nuclei at the pontomedullary junction. There are also direct connections with portions of the cerebellum. Efferent fibers from the brain stem travel through the vestibular nucleus to reach hair cells of the semicircular canals and macules. 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 fibers may also reach the cerebral cortex bilaterally.

CLINICAL FEATURES

Vertigo can be caused by either the peripheral or central vestibular apparatus. Peripheral vertigo is generally more severe and more likely to be associated with hearing loss and tinnitus. It often leads to nausea and vomiting.

Central vertigo is generally less severe than peripheral vertigo and is often associated with other signs of central nervous system disease.

Vertigo is multifactorial. Common causes of vertigo include the following:

- **Benign Paroxysmal Positional Vertigo (BPPV)**

  This is by far the most common cause of pathologic vertigo. Symptoms may spontaneously remit but commonly recur. Diagnosis rests on a finding of characteristic fatigable paroxysmal positional nystagmus after a rapid change from the sitting to the head-hanging position. It is thought to result from free floating calcium carbonate crystals that inadvertently enter the posterior semicircular canal. The crystals move within the endolymph and artificially displace the cupula. This cause of vertigo will be discussed in greater detail herein.

The symptoms of BPPV include vertigo, light-headedness, nausea, and imbalance. The vertigo may be violent and last less than 60 seconds. Symptoms are often precipitated by a position change of the head or body, e.g. when a person lies on one ear or the other or when the head is tipped backward to look up. Nystagmus also occurs. There is no associated hearing loss or tinnitus.

BPPV usually subsides within weeks to months but may recur after months or years.

- **Physiologic vertigo**
  This includes common disorders such as motion sickness, space sickness, and height vertigo. Rarely, the symptoms can last from months to years. Cause is unknown.

- **Acute peripheral vestibulopathy (acute labyrinthitis)**
  This presents with an acute onset of vertigo, nausea and vomiting lasting several days, with no associated auditory or neurologic symptoms. It may recur over months or years.

- **Meniere syndrome**
  This presents with episodic severe attacks accompanied by tinnitus and fluctuating hearing levels (see Entitlement Eligibility Guideline on Meniere's Disease).

- **Migraine**
  Vertigo is a common symptom with migraine, sometimes accompanied by damage to the inner ear.

- **Post Traumatic Vertigo**
  A "labyrinthine concussion" may result from a blow to the head. Symptoms include vertigo, hearing loss and tinnitus. The labyrinthine membranes are susceptible to blunt trauma. As well, fistulae or abnormal passages of the oval and round windows of the eardrum can result from impact
  noise, deep water diving, blunt head injury without skull fracture, or "severe physical exertion." "Severe physical exertion" would be exertion sufficient to rupture the labyrinthine membrane. The mechanism of rupture is a sudden negative or positive pressure change in the middle ear, or a sudden increase in

cerebrospinal fluid pressure transmitted to the inner ear by the cochlear aqueduct and internal auditory canal.

The rupture leads to sudden onset of vertigo or hearing loss, or both. The vertigo signs and symptoms would occur at the time of or within a few days of the trauma.

- **Post Concussion Syndrome**
  In some cases less pronounced symptoms are associated with mild head injury judged to be trivial at the time. Symptoms usually begin within a few days to a month following the injury.

- **Other Peripheral Causes**
  1. Chronic Bacterial Otomastoiditis
  2. Otosclerosis
  3. Drugs that damage the auditory system
  4. Cerebellopontine-Angle Tumors, including Acoustic Neuroma (see Entitlement Eligibility Guideline on Acoustic Neuroma).

- **Vascular Insufficiency**
  This is abrupt in onset and frequently associated with nausea and vomiting. It is usually caused by athrosclerosis and may be precipitated by, but not exclusively, mechanical compression from cervical spondylosis.

- **Other Central Causes**
  1. Multiple Sclerosis
  2. Parainfectious Encephalomyelitis
  3. Parainfectious Cranial Polyneuritis
  4. Ramsay-Hunt Syndrome
  5. Granulomatous Meningitis
  6. Cerebral or Systemic Vasculitis
  7. Temporal Lobe Epilepsy

PENSION CONSIDERATIONS

A. CAUSES AND/OR AGGRAVATION

THE TIMELINES CITED BELOW ARE NOT BINDING. EACH CASE SHOULD BE ADJUDICATED ON THE EVIDENCE PROVIDED AND ITS OWN MERITS.

1. Idiopathic

2. Head Trauma including, but not limited to, concussion prior to clinical onset or aggravation

   Vertigo usually begins within a few days to a month following the injury. Vertigo is a common sequela of head trauma. See also discussion of Post Traumatic Vertigo in *Clinical Features*.

3. Inner ear disease and/or inner ear surgery prior to clinical onset or aggravation

   Vertigo can occur up to several years after the development of the primary condition.

   Vertigo can be associated with a number of peripheral and central diseases where there is inner ear involvement (see *Clinical Features*).

   In general, a consequential ruling linking the vertigo to the primary condition should be sought.

4. Vascular insufficiency or occlusion prior to clinical onset or aggravation

   Vertigo can occur years after the primary conditions begin to develop.

   Vascular insufficiency or occlusion includes occlusion of the anterior vestibular artery, and cerebrovascular insufficiency, which includes strokes.

   In general, a consequential ruling linking the vertigo to the primary condition should be sought.

5. Inability to obtain appropriate clinical management

B. MEDICAL CONDITIONS WHICH ARE TO BE INCLUDED IN ENTITLEMENT/ASSESSMENT

- nystagmus associated with vertigo
- labyrinthitis
- ataxic gait

C. COMMON MEDICAL CONDITIONS WHICH MAY RESULT IN WHOLE OR IN PART FROM VERTIGINOUS DISORDERS AND/OR ITS TREATMENT

REFERENCES FOR VERTIGINOUS DISORDERS

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5. Kelly, W., M.D., eds. *Textbook of Internal Medicine*. Philadelphia: J.B. Lippincott, 1989. p. 2371

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