Vertigo and Dizziness — A Clinical Approach

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Abstract

Dizziness is a term which is used to describe a variety of sensations. It is possible to group these complaints into four types: a rotational sensation (Type I dizziness), impending faint (Type II dizziness), dysequilibrium (Type III dizziness) and vague lightheadedness (Type IV dizziness). Type I dizziness or vertigo is due to disease of the vestibular system - peripheral or central, and is characterized by a feeling of movement relative to one's surrounding. The majority of dizzy patients, however, belong to Types II, III and IV, collectively called the non-vestibular system disorders. The distinction is usually possible by a detailed history and clinical examination, but some special bedside tests - the dizziness simulation battery - are often required for properly distinguishing the various types of dizziness. Important causes of vertigo and the non-vestibular system disorders have been discussed with focus on benign positional vertigo, acute peripheral vestibulopathy, Meniere's disease, toxic damage to labyrinths, perilymph fistula, cerebrovascular disease, multiple sclerosis, cerebellopontine angle tumors, basilar migraine, vestibular epilepsy, cervical vertigo and phobic postural vertigo.

INTRODUCTION

The perception of our head and body positions and motions in space depend on fundamental types of information provided by five sensory modalities: vision, vestibular sensation, joint position sense, touch-pressure sensation, and hearing. These inputs are integrated in the central nervous system. Disturbances of any of these information or of the integration there of result in a number of uncommon sensations that are not part of daily experience. Patients affected with such problems frequently complain of "dizziness". Although they may suffer from a variety of symptoms, it is the thread of unfamiliar spatial disorientation that binds these complaints together. Patients can use a number of words to convey this sense: giddy, fainting, vertiginous, floating, light-headed, unsteady, clumsy, off-balance, swaying, spinning are few of the commoner ones. However, it is possible to group these complaints into four types: a rotational sensation, impending faint, dysequilibrium, and vague lightheadedness.

Types of Dizziness

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Type I dizziness is a definite rotational sensation (vertigo) in which the patient feels that either he/she or the environment is spinning. Vertigo is often accompanied by nausea, vomiting, and a staggering gait. Oscillopsia, a visual hallucination of rotational movements of the environment, may occur. The onset of vertigo is often instantaneous, and patients sometimes describe a sensation of being hurled to the ground. Whenever the patient's dizziness is exclusively rotational, it is due to a disorder of the vestibular system: either the peripheral labyrinth or its central connections. Because of this close relationship, it is important to separate definite rotational vertigo accurately from other types of dizziness. It is extremely important to elicit this 'dynamic' component in the patient's symptomatology.

Type II dizziness is a sensation of impending faint or loss or consciousness. Pallor, dimness of vision, roaring in the ears, and diaphoresis, with recovery upon assuming the recumbent position, are common, Type II dizziness of cardiovascular origin is of abrupt onset and short duration. When faintness is gradual in onset or persists despite lying down, hypoglycemia or other disorders of cerebral metabolism should be sought.

The complaint of impending faint usually implies an inadequate supply of blood or nutrients to the entire brain, such as occurs in postural hypotension. It is not a feature of focal cerebral ischaemia.

Sudden alteration in the state of consciousness (“dreamy states”) may be due to temporal lobe seizures, occurring in the absence of other, more obvious seizure phenomena.

Type III dizziness or dysequilibrium is loss of balance...
without an abnormal sensation in the head. This experience occurs only when the patient is walking and disappears upon sitting down. It is due to a disorder of motor system control.

**Type IV dizziness** is vague lightheadedness other than vertigo, faintness or dysequilibrium. This designation includes dizziness that cannot be identified with certainty as any of the other types. When patients complain of lightheadedness, fractional or poorly described symptoms of vertigo, faintness or dysequilibrium must first be looked for, e.g., a rocking sensation instead of spinning. This is often aided by testing with the dizziness simulation battery described later. Evidence of hyperventilation symptoms should next be sought, as well as symptoms pointing to a psychiatric disorder, particularly depression, anxiety, panic states or agoraphobia. Finally, the evidence for multiple sensory impairment should be examined, especially peripheral neuropathy, cervical spondylosis, or visual impairment in an elderly or diabetic patient.

The term vertigo seems to be much more specific to a disorder of the vestibular balance system comprising of the inner ear, vestibular nerve, brain stem, cerebellum and often including the eyes and the neck proprioceptors. The most important character is its ‘movement’ or its dynamic aspect. So the most important task a neurologist has is to question the dizzy patient to bring out the sole fact of whether his problem has a component of movement in it or not. This alone distinguishes between two major groups of disorders - the vestibular system disorders-VSDs and the non-vestibular system disorders - NVDs.

Once this distinction is achieved the task is much easy thereafter. The NVDs have large number of disorders in the causative list but most of the patients have no detectable pathology and comprises almost 70-80% of dizzy patients coming to a doctor. In this brief review we would like to deal a little about these NVDs and non-vertigo patients, before embarking upon the true vertigo patients with some disorder in their balance apparatus.

**THE NON-VERTIGO DIZZY PATIENTS - THE NVDs**

The majority of dizzy patients presenting to a doctor falls in this category. They describe their symptoms in various words but usually lack the rotatory component of movement either of self or of surroundings and do not have associated features of nausea, vomiting etc. Giddiness is a favourite term used by these patients, who on enquiry may be found to have antecedent history of other medical diseases, head or neck injury, significant life stress or medications.

Hypertension, diabetes, heart and lung diseases, endocrine disturbances and full check list of drugs used are most important things to ask about. The behavior of the symptoms with either exercise or valsalva maneuver often can give a clue to the diagnosis. Intracranial SOL, heart disease and hypotension are likely to worsen with valsalva, while cardiopulmonary disorders will result in exacerbation of the symptoms with exercise. If symptom decreases with exercise or distractions, it is likely to be of psychological origin. Associated alterations in consciousness clearly implies a syncopal element and excludes an organic VSD. Although pure hypoperfusion of the brain-stem due to any cause secondary to hypotension can cause true vertigo, this is usually associated with altered levels of consciousness and therefore fall under the category of NVDs. Relation of the giddiness to meals may reveal hypoglycaemia or the early postprandial dumping syndrome.

The general physical examination is often not helpful but BP measurement for asymmetry, orthostatic change, cardiac irregularity and neck bruits are often detectable. A ‘sharpened Romberg’ test is often very useful to exclude organic neural disease. This test comprises of standing tandem with eyes closed on either leg with arms folded across the chest for 30 seconds. If an individual can perform this test it almost excludes organic neurologic disease.

A proper psychiatric and cognitive function test is essential in such dizzy patients although panic and anxiety often accompanies the organic vestibular disorders as well. A new term ‘perceptual dizziness’ is much talked about these days. It encompasses the idea that the cortex plays a primary role in integrating all sensory inputs and preparing and response to it. In this task it frequently has to suppress some information or increase the gain of another system. Any disorder in the ability of this function of the cortex to select appropriate or reject inappropriate balance information may result in the feeling of dizziness where no physical finding is seen and neuro-otologic tests are normal. This form of dizziness probably occurs in the vast majority of patients who complain of feeling dizzy following minor head trauma. This is certainly not true vertigo. Two other forms of NVDs seem worth mentioning in this context. The first is ‘cervical vertigo’ associated with neck (not body) movements. It is too commonly seen in clinical practice to ascribe any form of loss of balance to cervical spondylosis specially in older patients on the belief that vertebro-basilar insufficiency occurs in these individuals due to compression of vertebral arteries in the neck. The literature had never been very clear about this concept and the authors feel that cervical spondylosis is too often made a ‘scapegoat’. Vertebrobasilar insufficiency should better not be diagnosed unless the vertigo-dizziness is accompanied by other brainstem signs/symptoms. Dizziness (true vertigo unlikely) in such individuals mostly result from abnormal proprioceptive stimuli from diseased neck joints resulting in a mismatch at the final integrative level. Such a situation also arises following neck injuries specially of the whiplash variety.

Brandt and Dieterich described a syndrome of phobic-postural vertigo which is distinguishable from agoraphobia (fear of open space) and acrophobia (fear of heights). It is characterized by the combination of initial vertigo with subjective postural and gait instability and the fear of impending death. Patients complain of vertigo rather than anxiety and feel physically ill. The illusory perception (vertigo) can be explained by the hypothesis that an
impairment of the space-constancy mechanism in these patients leads to partial uncoupling of the efferent copy for active head movements.

A short list of NVDs causing dizziness is given in Table 1, but the list is far from exhaustive.

**THE PATIENT WITH VERTIGO - THE VSDS**

True vertigo has been variously described but the essence of all description is its dynamic nature - an illusion of movement with respect to one’s surrounding space. In most cases it has a rotatory component. A patient with vertigo usually has the perception of movement with respect to a stationary state, i.e. either the person is moving with the surrounding static, a condition called subjective vertigo, or vice-versa when it is called objective vertigo. However these distinctions are of no localizing value. Another clinically distinguishing feature is its incapacitating effect on the patient, who is almost bed-bound at least in the beginning and is accompanied by anxiety, nausea and vomiting. The patients with NVDs who continue complaining of dizziness however may be seen to carry out normal life activities despite having their problem.

A brief history on confronting a patient with vertigo is helpful in a big way in directing our search for the cause. Sudden onset of symptoms, episodic nature, and relatively short duration of symptoms, localizes the lesion to the inner ear, particularly if there are associated features of tinnitus, hearing loss and violent nausea and vomiting. In general, continuous symptoms implies a CNS origin. Increase in vertigo with change of position or with eye closing is characteristic of all VSDs and is of no localizing value, however if symptoms occur only in certain positions of head, it suggests otolith dysfunction. Vertigo exacerbated by loud noise and valsalva maneuver denotes a peripheral inner ear disorder with a perilymphatic fistula (the Otolith - Tullio phenomenon). Another crude but very useful method of activating nystagmus is by vigorous head-shaking. This often increases or results in emergence of nystagmus in patients with VSDs whose signs are well compensated. This is mostly true of central VSDs. Nystagmus is so vital in VSDs that if it is absent (while patient complaining of vertigo) when observed with Frenzel’s glasses, the patient with vertigo can safely be stated to have a psychogenic origin for the symptom.

A neurological examination usually allows the distinction between central and peripheral causes of dizziness, and may reveal certain pattern of involment that point to the diagnosis; e.g. brainstem syndrome, cerebellopontine angle syndrome, multiple sensory impairment. A neuro-otologic evaluation should include tests of hearing and of vestibular function.

Electronystagmography (ENG) may help to identity and distinguish disorders of the peripheral (labyrinth, eighth nerve) and central vestibular systems. Recording of the amplitude, speed, and duration of ocular movements is first made during a series of eye and head position maneuvers. In the caloric test the patient is positioned so that the horizontal semicircular canals are exactly vertical, and each ear is irrigated with cool and warm water (or air) to produce a convection flow of endolymph, which mimics rotational stimulation of each canal. Observations on the speed, duration, and morphology of the nystagmus generated by this test provide information on the function of the vestibular apparatus and its central connections.

Audiometric studies are used to evaluate lesions of the middle ear, labyrinth, and cochlear nerve, particularly in Meniere’s disorder and cerebellopontine angle tumours. Routine pure tone audiometry indicates the presence or absence of a hearing loss and may also distinguish banal causes (acoustic trauma, aging, otosclerosis) from specific cochlear and nerve disorders. More elaborate “site of lesion” studies, such as the speech discrimination, alternate binaural loudness balance (ABLB), and acoustic reflex tests, improve

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**Table 1: Non-Vertigo Dizziness - Causes**

<table>
<thead>
<tr>
<th>Cardiac / Vascular</th>
<th>Non-Cardiac</th>
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<tbody>
<tr>
<td>Valvular heart disease</td>
<td>Anaemia</td>
</tr>
<tr>
<td>Arrhythmias</td>
<td>Tachy</td>
</tr>
<tr>
<td>Heart failure</td>
<td>Brady</td>
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<tr>
<td>Carotid sinus hypersensitivity</td>
<td>Hypo- and hyperglycaemia</td>
</tr>
<tr>
<td>Hypotension from any cause</td>
<td>Hyperventilation</td>
</tr>
<tr>
<td>Miscellaneous</td>
<td>Medications and toxins</td>
</tr>
<tr>
<td>Anxiety, phobias, depression</td>
<td>e.g. post-head injury</td>
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</tbody>
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**Evaluating The Dizzy Patient**

The history should explore four major questions that distinguish the disorders producing dizziness:

1. The type of dizziness (I to IV)
2. The abruptness of attacks or continuity of symptoms.
3. The relation or independence of dizziness to position or motion (standing/sitting/lying; sudden change in position; walking)
4. The age of the patient.

A good neurological examination is the cornerstone for establishment of the site of lesion and guides our investigations appropriately. The VSDs in contrast to the NVDs at least provide one physical sign to identify itself during physical examination - Nystagmus. In some patients secondary to compensation, this sign may be masked. There the use of ‘Frenzel glasses’ which impair the patients ability to fixate, but still allows the examiner to observe the patient’s eye motion, brings out the nystagmus. Another crude but very useful method of activating nystagmus is by vigorous head-shaking. This often increases or results in emergence of nystagmus in patients with VSDs whose signs are well compensated. This is mostly true of central VSDs. Nystagmus is so vital in VSDs that if it is absent (while patient complaining of vertigo) when observed with Frenzel’s glasses, the patient with vertigo can safely be stated to have a psychogenic origin for the symptom.

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the accuracy of diagnosis; brainstem auditory evoked response (BAER) testing is useful in distinguishing between brainstem and peripheral disorders.

Dizziness Simulation Battery

This is a series of eight bedside maneuvers that have proven valuable in distinguishing the various types of dizziness. Some produce dizziness in all patients, whereas others induce it only in patients with underlying disorders. After each maneuver the patient is questioned as to the similarity of test-evoked sensation to his own dizziness. Identification of a provoked sensation as identical to the patients dizziness is often more reliable than a verbal description, particularly if a single maneuver exclusively reproduced the symptoms.

1. *Orthostatic hypotension*: Blood pressure is measured supine, immediately on standing, and after three minutes.

2. *Potentiated Valsalva maneuver*: The patient squats for 30 seconds, then stands and strains against a closed glottis, or blows into a mercury sphygmomanometer, raising the column to 40 mmHg for 15 seconds.

3. *Carotid sinus stimulation*: The carotid sinus is unilaterally massaged for 15 seconds without continuous compression of the artery.

4. *Neck twist*: The patient rotates the head in each direction for 15 seconds. Dizziness may result from vestibular disorder or cervicogenic dizziness.

5. *Walking and turning*: The patient walks in one direction and then quickly turns, reversing direction. This test reproduces dizziness occurring with multisensory deficits, gait apraxia, and disorders of balance.


7. *Nylen-Barany maneuver*: The examiner carries the patients head backward from a seated position, so that it is hanging 45 degrees below the horizontal and turned 45 degrees to one side. Vertigo accompanied by nystagmus indicates positional vertigo. The characteristics distinguishing a “benign” from a “malignant” form of this condition are listed in Table 2.

8. *Barany rotation*: The patient is seated in a rotating chair, head tilted 30 degrees forwards from the vertical. The examiner spins the patient in one direction ten times within 20 seconds, then abruptly stop this rotation.

A flow chart showing major pathways of approaching a dizzy patient is provided in Table 3.

A brief resume of important conditions producing true vestibular vertigo follows. It is most important to distinguish between peripheral (labyrinthine) abnormalities and those involving central vestibular connection. The key to this distinction is the neurologic evidence for or against involvement of neighbouring brainstem structures.

**Peripheral Causes Of Vertigo**

*Benign Positional Vertigo*: Benign positional vertigo (BPPV) is probably the most frequent cause of vertigo, accounting for about 25 per cent of patients with this complaint. The patient experiences a sensation of spinning when rolling over in bed or making other sudden head movements. Symptoms are often greatest when the patient lies on the side with the affected ear underneath. The vertigo, sometimes accompanied by nausea and vomiting, lasts for less than five minutes, and between episodes the patient is free of symptoms. This condition, brought about only on change of position, differs from other vestibular disorders in which vertigo is increased by head motion but is present at other times as well. Benign positional vertigo occurs at any time during adult years; the cause is usually obscure, although it occasionally follows head trauma. The diagnosis is based on the typical history and on finding the “benign” type of vertigo and nystagmus on performing the Nylen-Barany maneuver (Table 2). This condition must be distinguished from so-called “central” or “malignant” positional vertigo and nystagmus (Table 2), which may occur with lesions (tumours, infarcts) involving posterior fossa structures; benign positional vertigo may be uncommonly associated with such conditions.

Typical posterior canal BPPV is caused by canalolithiasis, a free floating clot within the endolymph on the posterior semicircular canal. Some workers have hypothesized that immune complexes in the inner ear have an influence on the macular organ and cause the otoconia to dislodge more easily.¹

**Acute Peripheral Vestibulopathy (Acute Labyrinthitis; Vestibular Neuronitis)**: This condition is defined as a single bout of spontaneous vertigo, lasting for hours or days. Attacks occasionally follow a trivial respiratory or other infection, but the relation is not clear. Symptoms of vertigo, nausea, and vomiting usually improve within 48 hours, but may persist for seven to fourteen days. On examination the patient appears acutely ill, often pale and diaphoretic, resisting motion of the head. Nystagmus invariably accompanies the vertigo. As recovery takes place the patient may feel “off balance” for weeks or months owing to unilateral impairment of vestibular function, present in about 50 per cent of patients. Hearing is not impaired in this condition.

Several lines of evidence favour a viral hypothesis e.g. temporal bone pathology in patients with vestibular neuronitis or the wide spread detection of herpes simplex virus type 1.

### Table 2: Positional vertigo and nystagmus

<table>
<thead>
<tr>
<th></th>
<th>Peripheral or benign</th>
<th>Central or malignant</th>
</tr>
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<tbody>
<tr>
<td>1.</td>
<td>Latency</td>
<td>Usual</td>
</tr>
<tr>
<td>2.</td>
<td>Appearance</td>
<td>Torsional, upbeat or horizontal geotropic</td>
</tr>
<tr>
<td>3.</td>
<td>Fatigue</td>
<td>Usual</td>
</tr>
<tr>
<td>4.</td>
<td>Vertigo and systemic symptoms</td>
<td>Severe</td>
</tr>
<tr>
<td>5.</td>
<td>Localization</td>
<td>Posterior or horizontal semicircular canal</td>
</tr>
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</table>
DNA in human vestibular ganglia and nuclei. Some cases of acute rotational vertigo without hearing impairment are caused by unilateral central vestibular lesions in the pontomedullary brainstem.

**Acute and Recurrent Peripheral Vestibulopathy**: Acute and recurrent peripheral vestibulopathy is clinically similar to the entity described above, but consists of repeated bouts of vertigo occurring over a period of months or years. This condition occurs in an older age group than acute peripheral vestibulopathy, and is associated with less severe vestibular impairment on caloric testing. Approximately half the patients presenting with a single attack of peripheral vestibulopathy experience a recurrence linking this with the previous condition. The absence of auditory impairment distinguishes this condition from Ménière’s disorder.

**Ménière’s Disorder**: Ménière’s disorder is widely considered to be one of the most frequent causes of dizziness, but actually accounts for only about 5 percent of all dizziness and 10 to 15 per cent of vertigo. It usually occurs in adults and consists of recurring bouts of vertigo associated with hearing loss and tinnitus which may precede or follow the first bout of vertigo. Patients often complain of “fullness” in the ears, and are sometimes aware of recruitment as a sensation of auditory discomfort produced by loud noises. Bouts of vertigo last from hours to days, recurring as often as every week or as infrequently as every ten years. Hearing loss is unilateral in 80 to 90 percent of patients, with a severe deficit in half the patients. Most patients develop chronic impairment of vestibular function, resulting in the syndrome of vestibular imbalance. In many patients the recurrent episodes of vertigo may “burn out” over the years.

Diagnosis depends on the characteristic history and the audiometric findings (low frequency pure tone impairment, poor speech discrimination comparable to the pure tone hearing loss, and recruitment). Caloric testing demonstrates abnormal vestibular function in 80 percent of patients.

Pathologic studies have identified distention of the membranous labyrinth with endolymph as the immediate cause of this condition, but the explanation for the excess of endolymph remains obscure.

In recent years, numerous papers have discussed the possibility of an immunological, infectious, vascular or genetic pathogenesis.

**Toxic Damage to the Labyrinths**: Drugs of the aminoglycoside group (e.g., streptomycin, gentamicin, amikacin) may produce toxic damage to the peripheral vestibular apparatus. Although toxicity is generally dose-related, some patients develop labyrinthine damage after brief treatment with ordinary doses of these drugs, particularly if renal function is impaired. Tinnitus, hearing loss, or vertigo may be the presenting symptom, along with severe impairment of balance, nausea, and vomiting. Vertigo continues for days or weeks. If the ototoxic drug is immediately discontinued, damage to the labyrinth is usually arrested. A characteristic loss of balance follows the acute stage of vertigo and may include blurring of vision on motion owing to loss of the vestibulo-ocular reflexes. With the loss of vestibular sensation, these patients are dependent on visual cues to maintain balance, and are unable to walk in the dark. After several months, adaptation to the loss of vestibular sensation develops, and many can lead fairly normal lives.
Perilymph Fistula (PF) and Superior Canal Dehiscence Syndrome: PF may lead to episodic vertigo and sensorineural hearing loss as a result of pathologic elasticity of the otic capsule or leakage of the perilymph, usually at the oval or round window. The fistula and the partial collapse of the membranous labyrinth permit the abnormal transfer of ambient pressure changes to the maculae or capolae receptors. PF may be caused by barotraumas or e.g. cholesteotoma, in which case the horizontal canal is most often involved. The clue to diagnosis is precipitation of vertigo by acts of straining and can be simulated by asking the patient to perform the valsalva maneuver. A new type of PF is the superior canal dehiscence syndrome caused by dehiscence of bone overlying the superior (anterior) canal. As a result on this dehiscence a third mobile window is formed and changes in pressure are pathologically transduced to the anterior canal. The condition is diagnosed by high resolution temporal bone computed tomography which demonstrates the defect of the bone overlying the superior semicircular canal.

Central Causes Of Vertigo

Cerebrovascular Disease: Cerebrovascular disease produces vertigo when basilar-vertebral artery ischemia damages the vestibular nuclei or their connections. In virtually all cases injury to adjacent brainstem structures occurs, and vertigo is unlikely to be due to a stroke when other neurologic symptoms or signs are absent.

Transient ischemic attacks producing vertigo may be particularly difficult to diagnose, because at the time of examination the patient may have recovered completely. They rely on a history of neighborhood brainstem symptoms, such as diplopia, dysarthria, weakness or clumsiness of the limbs.

Multiple Sclerosis: Multiple sclerosis may produce vertigo in young patients, although this condition accounts for no more than 5 percent of acute vertigo in those below age 40.

Further, although multiple sclerosis may begin with vertigo, this is far less common than onset with optic neuritis or paresthesias.

Vertigo associated with ocular motor disorders that cannot be caused by purely peripheral vestibular disease (e.g. persistent diplopia, median longitudinal fasciculus syndrome, ophthalmoplegia) is strongly suggestive of this condition.

Cerebellopontine Angle Tumors: Cerebellopontine angle tumors are a rare cause of vertigo, but must not be overlooked early in their growth when they are readily removable. The large majority are benign acoustic neuromas arising in the internal auditory meatus. These tumors develop in middle-aged patients who experience vague unsteadiness that progresses over a period of years. Vertigo when present may be of the “malignant” positional type (Table 1), and only rarely does acute spontaneous vertigo occur. Hearing loss, tinnitus, facial numbness or weakness and cerebellar ataxia complete the picture. Unilateral or bilateral acoustic neuromas are especially common in von Recklinghausen’s disease (neurofibromatosis).

Basilar Migraine: In vertiginous migraine, vertigo may occur preceding the headache, during the headache phase, or as a “migraine equivalent” in place of the headache. Since both migraine and vertigo are common conditions, the occurrence of vertigo without a constant time relation to headache may represent only a coincidence in some unfortunate patients. The diagnosis should be considered only when vertigo is accompanied by other brainstem signs/symptoms (e.g. diplopia) and associated with headache.

Vestibular Epilepsy: It is a rare cortical vertigo syndrome secondary to focal epileptic discharges in either the temporal lobes or the parietal association cortex. Scanty information is available on this condition in recent literature and great care need be taken to diagnose this condition. A new clinical sign of vestibular epilepsy is skew deviation of eyes with nystagmus during attacks.

Treatment Of Vertigo

Treatment of vertigo consists of symptomatic relief and treatment of the underlying disease. Central causes of vertigo like cerebrovascular disease, multiple sclerosis or cerebellopontine angle tumours require urgent institution of specific treatment. In vertigo due to peripheral causes, symptomatic treatment tends to get more importance. This is usually done only with drugs and less attention is given to other treatment modalities. In a patient with acute or recurrent vertigo due to peripheral cause it is important to see that appropriate symptomatic treatment is advised and potentially treatable causes are not missed.

Any patient with vertigo should be advised to take bed rest and avoid sudden head movement in the initial stage. Vestibular suppressant medicines including antiemetics, antihistamines and other drugs are given. They should however be prescribed over a limited period of time following an acute vestibular lesion. Chronic use is not advocated as it probably retards the process of central vestibular compensation.

A process of rehabilitation should be started after the acute phase and this includes vestibular rehabilitative exercise. Vestibular suppressants and other central depressant medicine should be discontinued as both can interfere with the adaptive plasticity considered the basis for successful rehabilitation. Patients must be motivated to experience discomfort during the early stage of rehabilitation, with the expectation that they will benefit from markedly reduced (or absent) symptoms later. The very movements that precipitate dizziness should be encouraged, rather than avoided.

Benign paroxysmal pontonal vertigo should be treated with canalith repositioning procedure (Epley maneuver). A properly performed maneuver cures the disorder in 50%-80% cases of posterior canal BPPV.

Conservative medical management for Meniere’s disease consists of low salt diet and treatment with a diuretic.

For refractory cases in which the offending ear has been unequivocally determined, ablative therapy can be considered.
Destruction of the inner ear via a labyrinthectary result in hearing loss. Selective sectioning of the vestibular portion of the VIII th nerve (vestibular neurectomy) usually spares hearing. Chemical labyrinthotomy in which gentamicin injections are made into the middle ear, has recently been shown to be effective, with hearing preserved in most cases.

Rare, but surgically treatable causes of peripheral vertigo like perilymphatic fistula and newly recognized superior semicircular canal dehiscence syndrome, should be kept in mind and looked for by appropriate tests.

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Announcement
4th Congress of the Asian Pacific Society of Atherosclerosis and Vascular Disease, Bali, Indonesia, to be held from May 6-9, 2004.

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