Dizziness and Vertigo in Vertebrobasilar Disease

Part 1: Peripheral and Systemic Causes of Dizziness

B. Todd Troost, M.D.

DIZZINESS or dysequilibration is a not uncommon symptom in patients with cerebrovascular disease, particularly those with posterior circulation dysfunction. Vertigo, strictly defined, refers to the illusory sensation of unidirectional movement. While occasional patients with transient ischemic attacks (TIA) involving the vertebrobasilar system experience a definite illusion of environmental spin or of self-rotation, most do not present solely with "true" vertigo as defined. However, many patients complain of "dizziness," a term which describes a host of symptoms such as those listed in table I, and it is necessary to elicit the set of symptoms actually experienced by careful history taking. Since the ongoing or episodic sensations encompassed by the terms "vertigo," "unsteadiness," "presyncope," etc., are produced by a wide variety of causes, despite careful evaluation a significant number of patients cannot easily be diagnosed as having 1) peripheral vestibular, 2) systemic, or 3) central neurological causes to account for their complaints.

Some ambiguity exists in the term "central." It is used by otolaryngologists to include causes proximal to the vestibular end organ, including the vestibular portion of the eighth nerve. Most neurologists would consider conditions affecting the eighth nerve, such as neurofibromas, as peripheral. Since these lesions enlarge to involve other structures in the cerebello-pontine angle, including the brainstem, conditions affecting the eighth nerve are grouped here in the central category.

Generally, peripheral and systemic causes of vertigo and dysequilibration are more common than central etiologies, including cerebrovascular disease. Therefore, the features of peripheral and systemic dizziness and/or vertigo will be characterized in Part I of this paper, and central types will be considered in Part II.

Peripheral Causes

"Peripheral" causes result from dysfunction of the vestibular end organ — semicircular canals, utricle, and saccule (table 2). Peripheral vestibulopathy is a category proposed by Drachman and Hart to include such nonspecific descriptions as "vestibular neuronitis," which imply unproven inflammatory mechanisms. The condition is characterized by single or recurrent sudden episodes of vertigo lasting from hours to a few days and not associated with hearing loss or other neurological dysfunction. The vertigo may be evoked by head movement but not necessarily by a particular head position. A small number of patients with peripheral vestibulopathy are eventually found to have more specific disorders.

Whether isolated viral involvement of the vestibular nerve is a cause of acute or episodic vertigo is controversial. Many patients present with sudden, severe, vertigo, nausea, and vomiting without any hearing disturbance or facial weakness. The acute symptoms usually resolve in a few days to a week but sometimes are recurrent. Epidemic and seasonal outbreaks have suggested an infectious origin, but this remains an unproven assumption in the majority of patients with this symptom complex. Viral labyrinthitis may be one manifestation of a systemic viral infection such as mumps, measles, infectious mononucleosis, or upper respiratory infection.

Benign positional vertigo refers to a symptom complex usually indicating benign peripheral (end organ) disease. The differences between peripheral and central positional vertigo are outlined in table 3. The signs and symptoms of benign positional vertigo are transient (rarely longer than 60 seconds) and occur when a certain posture of the head is assumed, such as in lying down or turning in bed. In vertigo of other causes, changes in position may also intensify the symptom. Depending on whether the symptom (vertigo) or sign (nystagmus) is being emphasized, this condition is also called paroxysmal positional nystagmus or benign paroxysmal positional nystagmus. However, most normal people will demonstrate some positional nystagmus transiently in one position or another, especially when eye movements are recorded in the dark or behind closed eyelids with standard electroneystagmography. Many patients with benign
TABLE 1  Descriptions of Episodic Vertigo or "Dizziness"*

<table>
<thead>
<tr>
<th>Vertigo</th>
<th>Unsteadiness</th>
<th>Imbalance</th>
<th>Spinning</th>
<th>Floating</th>
<th>Fainting</th>
<th>Lightheadedness</th>
<th>Swaying</th>
<th>Twisting</th>
<th>Blurring vision</th>
<th>Disorientation</th>
<th>Poor equilibrium</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bouncing</td>
<td>Falling</td>
<td>Swimming</td>
<td>Staggering</td>
<td>Weaving</td>
<td>Moving</td>
<td>Passing out</td>
<td>Tilting</td>
<td>Listing</td>
<td>Rocking</td>
<td>Oscillating</td>
<td>Rolling</td>
</tr>
</tbody>
</table>

*Tables 1 to 4 are adapted from publications cited in references 1, 2, and 3.

TABLE 2  Peripheral Etiologies of Vertigo

1. Peripheral vestibulopathy—labyrinthitis, vestibular neuritis, acute and recurrent peripheral vestibulopathy
2. "Benign" positional vertigo—benign positional nystagmus, benign paroxysmal vertigo
3. Post-traumatic vertigo
4. Vestibulotoxic drug-induced vertigo
5. Ménière's syndrome
6. Other focal peripheral disease—local bacterial infection, degeneration, genetic anomalies, cupulolithiasis, tumor, otosclerosis, fistula, and rarely focal ischemia and others

TABLE 3  Characteristics of Peripheral Versus Central Positional Vertigo

<table>
<thead>
<tr>
<th>Symptom or sign</th>
<th>Peripheral</th>
<th>Central</th>
</tr>
</thead>
<tbody>
<tr>
<td>Latency—time to onset of vertigo or nystagmus</td>
<td>~3 to 45 seconds</td>
<td>No latency, begins immediately</td>
</tr>
<tr>
<td>Fatigability—signs and symptoms decrease after onset</td>
<td>Yes</td>
<td>No</td>
</tr>
<tr>
<td>Adaptation (habituation)—lessening signs and symptoms with repetition of provocative maneuver</td>
<td>Yes</td>
<td></td>
</tr>
<tr>
<td>Nystagmus direction</td>
<td>Direction fixed</td>
<td>Direction changing</td>
</tr>
<tr>
<td>Intensity of signs and symptoms</td>
<td>Severe vertigo, marked nystagmus, systemic symptoms such as nausea</td>
<td>Usually mild vertigo, less intense nystagmus, rare nausea</td>
</tr>
<tr>
<td>Reproducibility</td>
<td>Inconstant</td>
<td>More consistent</td>
</tr>
</tbody>
</table>

The time from injury to onset of symptoms may be days or weeks. When the symptoms are delayed, the mechanism may be hemorrhage into the labyrinth with the later development of serous labyrinthitis. Post-traumatic positional vertigo may also be produced if the calcareous deposits (otoconia) that lie on the sensory vestibular receptors are displaced to new and sensitive regions of the ampulla of the posterior canal, causing it to be more susceptible to stimulation in certain head positions. In post-traumatic vertigo the symptoms are usually those of peripheral vestibulopathy or benign positional vertigo. Generally, the prognosis is good, with symptoms gradually resolving in weeks to months.

Vestibulotoxic drug-induced vertigo is caused by agents which are presumed or have been proven to injure the peripheral end organ. Among such agents are the aminoglycosides, streptomycin, and gentamycin which are selectively concentrated in the endolymphatic fluid and produce a dose-related irreversible injury to vestibular hair cells. Symptoms of bilateral end organ injury persist despite immediate cessation of the drug. In contrast, many drugs have widespread reversible central and peripheral nervous system effects including transient vertigo which subsides on stopping the medication (table 4).
Ménière's syndrome is characterized by attacks of severe vertigo and vomiting, tinnitus, fluctuating hearing loss, distortions of sound perception, ill-described aural sensations of fullness and pressure, and spontaneous recovery in hours to days. Occasional sufferers experience such severe disturbances of vestibular function that they fall as though thrown to the ground. They do not lose consciousness in such episodes, although their awareness of surroundings may be altered by the intensity of the vertiginous sensations and nausea. The most consistent pathological finding in Ménière's syndrome is an increase in the volume of the endolymphatic fluid and distention of canals — hence, the term "endolymphatic hydrops." While specific causes such as bacterial, viral, and syphilitic infection may lead to the same pathological changes and symptoms, the majority of cases are idiopathic. A variety of surgical procedures to "shunt" endolymphatic fluid have met with variable success, and final assessment of therapeutic effectiveness is not available.

Other focal diseases listed in the last group of peripheral vestibular disorders in table 2 include acute otitis media, chronic ear infection, hereditary degenerative disorders of the end organ, and local tumors. Focal ischemia or "stroke" of the end organ is often cited as a specific cause of vertigo, but such isolated and localized vascular occlusive disease is difficult to document. This condition is discussed further under central causes.

Systemic Causes

"Systemic" causes are considered as a separate category so as to include disorders that secondarily affect peripheral and/or central vestibular structures to produce vertigo (table 4).

A frequent cause of dizziness in the broad context of that term is drug ingestion. Vestibulotoxic drugs can produce true vertigo. The dizziness produced by many other drugs is more a sense of weakness, dys-equilibration, or "fuzzy-headedness." The agents listed are among the most common offending drugs. It is important that every attempt be made to determine the type and quantity of medication being taken by the dizzy patient. Frequently, the elimination or reduction of medication, such as mild "tranquilizers," will produce clear-cut improvement.

The multiple causes of presyncope or postural hypotension are often responsible for complaints of vertigo or dizziness. Again, careful historical review and documentation of physical findings such as postural hypotension or cardiac arrhythmia direct further investigation and therapy. Presyncopal symptoms may be described as "lightheadedness" among other phrases. Presyncope is a common mechanism for the complaint of "dizziness" and may even produce vertiginous sensations. Postural hypotension is a common side-effect of antihypertensive agents, diuretics, and dopaminergic agents. When the symptom is intermittent, a history of lightheadedness following change from recumbent or sitting posture to an erect position, but not the reverse, is more helpful than blood pressure measurements. In adolescents a hyposensitive carotid sinus reflex during periods of rapid growth is not rare, and transient symptoms of postural dizziness might be explained by this mechanism.

Among the endocrinopathies which cause disorders of equilibration are notably hypothyroidism and, less commonly, diabetes. While not common as a specific cause, hypothyroidism is frequently associated with vestibulopathy and disorders of hearing; it should always be considered when the symptoms of vertigo remain undiagnosed. Indeed, dizziness is often a presenting complaint in patients with thyroid deficiency. The mechanism in diabetes is probably the orthostatic hypotension which may accompany the disease. Since both of these endocrine states predispose to rapid development of atherosclerotic occlusive disease, it is apparent that interpretation of the symptom must be made with care. The remaining systemic conditions rarely present with isolated vertigo but are included as additional primary or secondary causes.

Multiple or isolated cranial neuropathies occur in focal or systemic disease, including vasculitis, granulomatous disease, and meningeal carcinomatosis, but the etiology is often elusive. Evidence of systemic involvement is elicited by history, physical examination, and laboratory evaluation.

References
Dizziness and vertigo in vertebrobasilar disease. Part 1: peripheral and systemic causes of dizziness.
B T Troost

Stroke. 1980;11:301-303
doi: 10.1161/01.STR.11.3.301

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://stroke.ahajournals.org/content/11/3/301.citation

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in Stroke can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to Stroke is online at:
http://stroke.ahajournals.org//subscriptions/