Dizziness and Vertigo in Vertebrobasilar Disease

Part II. Central Causes and Vertebrobasilar Disease

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IN PART I, the general topic of dizziness and vertigo was introduced, with a discussion of the major peripheral and systemic causes. Part II concentrates on the central causes of dizziness and vertigo, including vertebrobasilar disease.

Central Causes

Dysfunction of the vestibular portion of the eighth nerve and the vestibular nuclei within the brainstem and their central connections is a central cause of dizziness and vertigo. Neural connections with the central vestibular nuclei include interaction with the vestibular portions of the cerebellum (primarily cerebellar flocculus), the visual sensory system, and afferent connections from muscle, joint, and tactile receptors. Central causes of vertigo are less common than peripheral or "systemic" etiologies, the vertiginous symptomatology is usually less prominent, and additional neurological signs are usually present on examination. Central disorders include demyelinating disease, tumors, seizures, and vertebrobasilar artery disease (table).

Demyelinating disease should only be diagnosed after documentation of disseminated central nervous system lesions, such as optic neuritis, transverse myelitis, internuclear ophthalmoplegia, or other focal signs.

Cerebellopontine angle tumors do not usually present symptoms of episodic vertigo alone. The most common tumor in this location results from a proliferation of the Schwann cells (which produce the myelin of nerves), hence the name Schwannoma. Most of these tumors arise in the vestibular portion of the eighth nerve within the internal auditory canal. They progressively enlarge, deforming the internal auditory meatus and compressing adjacent neural structures: the acoustic portion of the eighth nerve, facial nerve, root of the trigeminal nerve, brainstem, and cerebellum. Other tumors occurring in the cerebellopontine angle include meningiomas, epidermoids, and metastases.

The most common symptoms associated with eighth nerve tumors are progressive hearing loss and tinnitus, symptoms unlikely to occur with vertebrobasilar vascular disease. Vertigo occurs in approximately 20%, but a symptom of "imbalance" or dysequilibration may affect as many as 50%. Nonetheless, a rare person with subtle hearing loss, tinnitus, and episodic vertigo is encountered with a vestibular nerve tumor. All those with progressive unilateral hearing loss and particularly those with any vestibular symptomatology should be carefully examined for additional neurological signs such as a depressed corneal reflex. This reflex depends on normal sensation of the cornea supplied by the fifth nerve. The earliest sign of a tumor in the cerebellopontine angle may be decreased corneal sensation produced by pressure on the trigeminal nerve.

Seizure disorders, especially temporal lobe or psychomotor epilepsy, are rare causes of dizziness or vertigo. The history almost always reveals additional symptomatology such as loss of awareness, automatic behavior, or generalized seizure activity following an aura of vertigo. However, some epileptics with psychomotor seizures have isolated auras of dizziness or other symptoms similar to those listed in table 1 of Part I. Despite the rarity of isolated vertigo as a TABLE

<table>
<thead>
<tr>
<th>Central Neurological Causes of Vertigo*</th>
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<tr>
<td>1. Brainstem ischemia and infarction.</td>
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<tr>
<td>3. Cerebellopontine angle tumor—acoustic neuroma, meningioma, cholesteatoma, metastatic tumor, etc.</td>
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<td>4. Cranial neuropathy—focal involvement of eighth nerve or in association with systemic disorders.</td>
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<td>5. Intrinsic brainstem lesions—tumor, arteriovenous malformation, trauma, etc.</td>
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<td>6. Other posterior fossa lesions—primarily other intrinsic or extra-axial masses of the posterior fossa such as hematoma, metastatic tumor, and cerebellar infarction.</td>
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<td>7. Seizure disorders.</td>
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<td>8. Heredofamilial disorders—spinocerebellar degenerations: Friedreich's ataxia, olivopontocerebellar atrophy, etc.</td>
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*This table is adapted from publications cited in references 1 and 3, Part I.
seizure manifestation, the duration of one to two minutes, and vaguely described, associated symptoms of disorientation, blurred vision, or nausea may cause considerable difficulty in distinguishing the aura from vertebrobasilar disease symptomatology.

Vertebrobasilar Arterial Disease

With this background of the types of dizziness and vertigo produced by other conditions, it is appropriate to consider their association with vertebrobasilar arterial disease. The posterior circulation supplies blood to the medulla, pons, cerebellum, mesencephalon, thalamus, occipital lobes, and even portions of the temporo-occipital and parieto-occipital junctions. The most common defects with disease in this system are disorders of motor functions such as weakness, clumsiness, or paralysis. A crossed defect (motor or sensory on one side of the face and the opposite side of the body) is good evidence of a defect (motor or sensory on one side of the face and the opposite side of the body) is good evidence of brainstem dysfunction. If the occipital lobes are the site of ischemia, visual loss in the form of complete or partial homonymous hemianopsia will occur. Ataxia, imbalance, unsteadiness or dyssequilibrium not necessarily associated with spinning vertigo may occur because of labyrinthine or cerebellar ischemia.

Before deciding the symptoms and signs of vertigo or dizziness are due to vertebrobasilar arterial disease, one should seek additional signs of brainstem involvement. Brainstem ischemia (transient ischemic attack or TIA) should be accompanied by other symptoms in addition to vertigo or dizziness before such a diagnosis is seriously entertained. Among these symptoms are transient clumsiness, weakness, loss of vision, diplopia, perioral numbness, ataxia, drop attack, and dysarthria. The belief that dizziness is a more or less required symptom before posterior circulation disease can be diagnosed is incorrect because isolated symptoms may occur without dizziness. On the other hand, such symptoms may not always accompany dizziness due to posterior circulation dysfunction. A moderate number of elderly patients without laboratory evidence of peripheral vestibulopathy or systemic disease will have dyssequilibration or dizziness due to vertebrobasilar artery disease.

Sudden hearing loss with moderate dizziness may be due to infarction in the distribution of the internal auditory artery. In isolation this symptom complex is uncommon in the elderly patient with atherosclerotic vertebrobasilar arterial disease and is more suggestive of disease affecting small and intermediate arteries such as lues, systemic lupus erythematosus, or periarteritis nodosa. In the atherosclerotic patient the symptoms are usually accompanied by other signs of brainstem or cerebellar dysfunction which allow a more certain diagnosis. If actual brainstem infarction occurs, neurological signs are often present on examination. These signs, which may not be obvious and should be carefully sought, include nystagmus of the central type, hyperreflexia, internuclear ophthal-moplegia, homonymous visual field defects, dysarthria, vertebral bruits, and disorders of coordination or cerebellar function.

Symptoms of dizziness are also quite common in proximal extracranial occlusion of the vertebral arteries and in the subclavian steal syndrome. Up to this point emphasis has been placed on the accompanying signs and symptoms which almost always occur with vertebrobasilar artery disease. However, acute severe vertigo, mimicking labyrinthine disease, has been an early symptom of acute cerebellar infarction in the distal territory of the posterior inferior cerebellar artery. To differentiate this condition from labyrinthine disease, particular attention is directed to the type of nystagmus which is present. Acute peripheral vestibulopathy usually causes unidirectional nystagmus with the fast phase away from the side of the lesion and with increase during gaze to the opposite side. Swaying or falling occurs toward the side of the lesion (opposite the nystagmus fast phase). With inipient cerebellar infarction, the sway or fall is to the side of the lesion; the nystagmus is direction changing but is also most prominent to the side of the lesion. To describe it another way, with central lesions the fast phase of the nystagmus is in the direction of gaze but becomes more prominent when gaze is directed toward the side of the lesion (see table 3, Part I).

Acquired disease of the brainstem and cerebellum produces a variety of types of nystagmus which sometimes present as a complaint of "oscillopsia," an illusion of environmental movement characterized by "bouncing" or "jiggling." While oscillopsia is a common complaint when there is significant bilateral labyrinthine abnormality, the presence of vertical oscillopsia should alert the physician to look for primary position upbeat or downbeat nystagmus. These types of nystagmus are reliable indicators of central nervous system abnormality due to intrinsic structural disease or drugs.

Discussion

The vestibular system functions to provide man with (1) spatial orientation at rest or during acceleration; (2) visual fixation during head and/or body movement (vestibulo-ocular reflex); and (3) feedback control of muscle tone to maintain posture. These functions and their control mechanisms are interconnected in a highly complex manner. It is now realized that the central vestibular nuclei respond actively to visual input as well as to afferent information from the peripheral labyrinth. An intact person easily confuses afferent sensory information. For example, a sensation of spin or true vertigo is experienced when viewing a visual environment filled with moving stripes (full field optokinetic stimulation). Almost every individual, while quietly seated, will have a compelling illusion of rotation if he is placed in a circular room with a moving environment of stripes (the circular-vection illusion). A more common illusion of motion is that of linear movement occurring when one is seated in a car and the vehicle alongside begins slowly moving forward. Most people have a sensation of rolling backwards and attempt to brake. It is,
therefore, not surprising to find that patients with subtle abnormalities of peripheral or central vestibular mechanisms suffer definite, momentary periods of disorientation while viewing a moving, patterned environment. Some have episodic vertigo during vehicular travel while observing repetitive visual patterns. The history of disorientation or dizziness when viewing stripes or the production of such symptoms during optokinetic testing may well be a sign of central vestibular dysfunction due to vascular disease.

It should now be apparent that the symptoms of dizziness and vertigo reflect disturbances in more than one system. The combination of “multiple sensory deficits” can produce disorientation or dysequilibrium interpreted as “dizziness” or “vertigo.” This often occurs in the elderly when vision (cataracts) and hearing (presbycusis) are both impaired. While most younger patients readily compensate for unilateral peripheral vestibular damage, older patients frequently cannot, indicating either bilateral peripheral vestibular dysfunction or a separate central abnormality which decreases their ability to compensate. Vertigo and dizziness sometimes cannot be simply classified as peripheral, central, or systemic. The symptom complex may well represent a combination of abnormalities, including incomplete adaptation. This is particularly true when vertebrobasilar disease is a contributing factor.

References
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