K. J. Lee: Essential Otolaryngology and Head and Neck Surgery (IIIrd Ed)

Chapter 5: The Vestibular System and its Disorders: Part II

The term dizziness has often been used imprecisely. It is imperative for an otolaryngologist to be able to differentiate the following terms:

Dizziness: Encompasses any discomfort, other than pain, related to the head. The etiology could be visual, cerebral, vestibular, or gastrointestinal.

Vertigo: Describes a discomfort in a patient experiencing an actual sensation of motion in which either the patient or his environment is moving. The direction is often rotatory.

Unsteadiness: Is a loss of equilibrium in relationship to one's environment. It is often described by the patient as "bumping into things" or "almost falling". The etiology could be cerebellar, cerebral, pyramidal tract, posterior column, or vestibular. A pure labyrinthine etiology seldom gives rise to unsteadiness without vertigo.

Lightheadedness: Is described by the patient as a feeling of "going to faint". It is also used to describe mild vertigo.

The next step when evaluating a "dizzy" patient is to determine the duration of each attack, the frequency of each episode, and whether it is constant, episodic, or related to position. A past medical history of dizziness, no matter how remote, should be taken into consideration when arriving at the diagnosis. It is also imperative to obtain a general medical history to rule in or out diabetes mellitus, hypertension, and other cardiovascular or neurologic diseases.

A complete ENT and neurologic examination which includes observation for spontaneous nystagmus in three directions of gaze, either through Frenzel glasses (+20 lenses) or other methods, is a prerequisite to other studies. Audiometric tests, mastoid and internal acoustic meatus view, caloric tests, or ENG can be obtained if needed. Positional testing is performed if the symptoms are questionably induced or provoked when the patient assumes a particular position. One should note that a sudden change of position may aggravate the symptoms in any type of dizziness without necessarily implying a disease of labyrinthine origin or positional vertigo. A feeling of lightheadedness upon rapidly assuming an upright position does not indicate a labyrinthine or vestibular disorder.

Nystagmus

The slow phase of the nystagmus is the direction of the flow of the endolymph and it is vestibular in origin, whereas the quick phase is most likely initiated by the reticular formation as a compensatory mechanism.

Spontaneous: Nystagmus present without positional or other labyrinthine stimulation.
Induced: Nystagmus elicited by stimulation, i.e. caloric, rotation, parallel swings, etc.
Positional: Nystagmus elicited by assuming a specific position as in positional testing.

Spontaneous Nystagmus

Spontaneous nystagmus can be pendular without a fast or slow phase. Pendular nystagmus usually points to a congenital disorder, ocular disease (Miner's nystagmus) or multiple sclerosis. Labyrinthine nystagmus usually has a fast and a slow phase. By convention, the direction of the nystagmus is determined by the fast component.

Spontaneous dissociated nystagmus is also indicative of central nervous system disease.

Spontaneous rotatory nystagmus is rare although not infrequently observed with a horizontal component during positional testing of patients with positional vertigo of the benign paroxysmal type.

Spontaneous vertical or diagonal nystagmus is very rarely observed. It usually signifies a central nervous system disorder. Vertical or diagonal nystagmus induced by stimulation on positional testing also suggests central disorders.

First-degree spontaneous nystagmus: Nystagmus present only when gazing in the direction of the fast component.

Second-degree spontaneous nystagmus: Nystagmus present when gazing in the direciton of the fast component and on straight gaze.

Third-degree spontaneous nystagmus: Nystagmus present in all three directions of gaze.

Clinical correlation:

1st degree = peripheral lesion

2nd degree = central lesion

3rd degree = central lesion.

When testing for spontaneous nystagmus one should not bring the patient to a complete lateral gaze as this will induce fatigue or "end point" nystagmus. The patient should be tested with eyes in straight gazew, 30-45° to the left and 30-45° to the right.

Spontaneous nystagmus due to peripheral disease can be inhibited by fixation - its manifestation is made possible by eliminating fixation through eye closure or darkness. Spontaneous nystagmus of the central type is present during eye fixation and may be eliminated during eye closure or darkness. When the lesion is below the level of the vestibular nuclei, spontaneous nystagmus is exaggerated by eye closure or darkness. When above this level, spontaneous nystagmus is subdued by eye closure or darkness.

Positional Nystagmus

Positional nystagmus is nystagmus elicited during positional testing.

Technique of Positional Testing

1. Sit the patient on a bench in an upright position with arms folded.

2. Reassure the patient that he is not going to fall regardless of his sense of direction. Insist that it is of utmost importance to keep his eyes open during the test.

3. Bring the patient backward swiftly with head hanging. Watch for nystagmus induced by assuming this position. Notice (a) the latency between assuming the position and the onset of nystagmus, (b) the character and direction of nystagmus, (c) the duration of the nystagmus. If the nystagmus has a rotatory component it is classified as clockwise or counterclockwise as illustrated in Fig. 5-1.)

4. Bring the patient back to the upright position either after the nystagmus has stopped or after 3 minutes and determine: (a) the direction of the nystagmus, (b) the duration of the nystagmus.

5. Repeat steps 3 and 4 except that now the head is positioned with the left ear down in step 3.

6. Repeat steps 3 and 4 again except that now the patient's head is positioned with the right ear down in step 3.

The nystagmus elicited can be classified according to Nylen's classification or as modified by Aschan.

Nylen's Classification

1. Type I: The direction of nystagmus varies with the positions of the head in the positional testing.

2. Type II: The direction of nystagmus remains fixed regardless of the position of the head during positional testing. When present in different head positions, the nystagmus is stronger in a particular position.

3. Type III: The nystagmus is irregular, characterized by variations in its behavior. It is thus sometimes direction-changing, sometimes direction-fixed, and sometimes changes its direction with the same head position. Type III is used to label all forms of positional nystagmus that cannot be classified under type I or type II.

Clinical Correlation

1. Type I: Implies a cental lesion, i.e. multiple sclerosis or a cerebellar tumor.

2. Type II: Implies peripheral lesion or acoustic neurinoma.

3. Type III: Unknown significance.

Aschan's Classification

1. Type I: The nystagmus is nonfatigable and persistent. Its direction changes with head position.

2. Type II: The nystagmus is nonfatigable and persistent. Its direction remains fixed with change of head position.

3. Type III: ALl varieties of transitory positional nystagmus with latency and fatigue are included.

Clinical Correlation

1. Type I: The majority of these patients have CNS disorder.

2. Type II: Possible end organ lesion, but mainly CNS.

3. Type III: Peripheral disease. Usually indicating positional vertigo of the benign paroxysmal type.

The positional testing has many implications. However, the only practical clinical application to date is to separate positional vertigo of the benigh paroxysmal type from positional vertigo secondary to CNS disease (Table 5-1). Characteristics of positional vertigo of the benign paroxysmal type are:

Table 5-1. A Comparison of the Features of Peripheral and Central Nystagmus

Feature	Peripheral	Central
Latency	**5-15 seconds**	**No latency**
Persistence	**Disappears in 50 sec**	**Lasts more than 1 min**
Fatigability	**Disappears on repetition**	**Repeatable**
Position	**Present in one head position**	**Present in multiple head pos**
Vertigo	**Always present**	**Occasionally absent**
Direction	**One direction**	**Changing in different head pos**
Incidence	**85% of all positional vert**	**10-15% of all positional vert.**

1. The nystagmus elicited is rotatory.

2. If the left ear is the pathologic, the patient will manifest a clockwise rotatory nystagmus when assuming the left ear down position.

3. There will be a latency of 5-15 seconds between assuming that position and the onset of nystagmus.

4. The nystagmus will "fatigue out" (stop after a while).

5. Upon reassuming the upright position, the patient may manifest a nystagmus in the opposite direction.

6. On repeat testing without rest in between, the positional nystagmus can no more be elicited.

Stimulation Tests

Simple Caloric Test

There are many modifications of this test. The important point is that it is a qualitative test measuring the difference in response between the right and left ears. It does not matter which modification of this test is used provided the physician is familiar with the test chosen as well as with its clinical implications and limitations. It is important that the irrigating fluid reaches the tympanic membrane and is not just reflected by the anterior osseous canal or cerumen impaction. This is particularly crucial if small amounts of water are used.

To bring the horizontal canal to a vertical plane for the caloric test, it is necessary to tilt the head back 60° when the patient is in an upright position or elevate the head 30° when the patient is supine.

The test as devised by Kobrak (Kobrak test) used 0.2-5 mL of ice water instilled against the tympanic membrane of the patient in a sitting position with head tilted 60° back. The latency and duration of nystagmus are measured. Other tests include Veit's minimal caloric test, Barany's mass caloric test and Dundas-Grant cold air test for patients with perforated tympanic membrane.

Directional Preponderance

This is a standardized test to measure canal paresis and directional preponderance. To determine directional preponderance Fitzgerald and Hallpike used the following system:

The patient is placed supine with head elevated 30°. Each ear is douched in turn with water at exactly 30°C (86°F) and with water at 44°C (112°F), each douche consisting of no less than 250 mL. At least 5 minutes should elapse between each douche. Directional preponderance is calculated as follows.

1. Right ear irrigated with cold H_2O: duration of nystagmus to (L) = a.

2. Right ear irrigated with warm H_2O: duration of nystagmus to (R) = b.

3. Left ear irrigated with cold H_2O: duration of nystagmus to (R) = c.

2. Left ear irrigated with warm H_2O: duration of nystagmus to (R) = d.

If a + b is less than c + d, the right ear is hypoactive. If a + d is less than b + c, there is directional preponderance to the right.

Directional preponderance is believed to be toward the side of a central lesion and away from the side of a peripheral lesion.

ENG

This test is based on the difference in potential between the cornea (+) and the retina (-). During nystagmus, movements of the eyes cause this corneal-retinal potential to be displaced laterally giving rise to changes in potential that can be recorded by electronic equipment. This electronic recording of the nystagmus is called electronystagmography or ENG.

1. Electrodes are placed as shown: A, B, C, D, E (Fig. 5-2).

2. By convention and calibration, an upward swing of the pen indicates nystagmus to the right while a downward swing indicates nystagmus to the left.

3. The patient lies supine with the head elevated 30°. He is 8.5 ft from the wall and gazes at points A, B, and C for calibration (Fig. 5-3).

4. Recordings are then taken with eyes open and eyes closed to check for spontaneous nystagmus.

5. ENG recordings can be obtained from the rotation test, positional test, etc.

6. The patient is then irrigated with 250 mL of water at 30°C and 250 mL of water at 44°C in turn as outlined in the Fitzgeral and Hallpike test.

7. The parameters measured by electronystagmography include: intensity (frequency of beats, amplitude of pen displacement, and velocity of slow component) and duration.

Rotation Test

This test stimulates the labyrinth by the force of rotation. It has little clinical application because it stimulates both ears simultaneously.

Technique

1. Sit patient up with head brought forward 30°.

2. Rotate the chair at the speed of approximately 10 turns per 20 seconds then stop abruptly.

3. If the subject was rotated to his right, the normal response would be nystagmus to the left with past-pointing to the right. A patient rotated to his right and brought to an abrupt stop undergoes the same effect as beginning a turn to his left, i.e. the quick phase is to the left, slow phase to the right. The direction of the slow phase is that of the flow of the endolymph.

Past-pointing and falling also would be to the right, in the direction of the slow phase. Past-pointing is a compensatory body musculature reflex, its nerve pathways being entirely separate from those of the ocular reflex.

Parallel Swing

Parallel swing tests utricular function and is still a research tool.

Fistula Test

In the presence of a fistula, stimulation of the ear with positive pressure causes nystagmus to the same side while negative pressure brings about nystagmus to the opposite side. The presence of nystagmus may be accompanied by vertigo. However, it is the presence of the nystagmus that is significant in this test. If the patient experiences vertigo without nystagmus of the type mentioned, he could be undergoing a cool caloric stimulation without a positive fistula test. Example: When a fistula is present in the right ear, stimulation of this ear with:

(+) pressure gives nystagmus to the (R)
(-) pressure gives nystagmus to the (L).

Optokinetic Nystagmus

Optokinetic nystagmus can be elicited by various methods. One practical way is to have the patient watch a drum 30 cm high by 25 cm in diameter. The surface has 1.5 cm wide white vertical stripes. The drum is rotated about its vertical axis taking 1-2 seconds for a complete revolution. The optokinetic nystagmus is measured while the drum is rotated in one direction. The direction is then reversed and the measurements taken again. When the optokinetic nystagmus is asymmetrical for the two directions of drum rotation, a central lesion is implied. Labyrinthine spontaneous nystagmus can be altered to optokinetic nystagmus by fixation on a rotating drum, whereas spontaneous nystagmus of central origin remains unchanged.

Practical benefits of ENG:

a. To record spontaneous nystagmus and positional nystagmus with eyes open and eyes closed.

b. To record caloric responses.

c. To study optokinetic nystagmus (eye-tracking is still nonclinical).

Differential Diagnosis

When evaluating a patient with vertigo, one should try to differentiate between vertigo of peripheral origin and that of central origin (Table 5-2).

Table 5-2. Comparison of Vertigo of Peripheral Origin with That of Central Origin

Peripheral

--> Central

A definite sensation of movement is present.

--> Vertigo is mild and more like a sensation of unsteadiness.

Verigo is severe and paroxysmal.

--> Vertigo is vague with no specific onset or termination.

Attacks last from minutes to days; are accompanied by spontaneous nystagmus and associated with autonomic nervous system disorders; patient almost never loses consiousness.

--> Attacks of vertigo last for weeks, often with no apparent nystagmus.

The following list of differential diagnoses constitutes the more common etiologies of the "dizzy" patient:

1. Ménière's disease.

2. Acoustic neurinoma.

3. Vestibular neuronitis.

4. Bacterial labyrinthitis.

5. Nonbacterial "labyrinthitis".

6. Positional vertigo of the benign paroxysmal type.

7. Congenital syphilis.

8. Cogan's syndrome.

9. Vertigo due to whiplash injury.

10. Temporal bone fracture and labyrinthine concussion.

11. Multiple sclerosis.

12. Vascular insufficiency.

13. Cervical vertigo.

14. Vertiginous epilepsy.

Ménière's Disease

The medical history of Ménière's disease is usually typical. The patient suffers episodic vertigo lasting from 30 minutes to 2 hours. The attack is associated with nausea, vomiting, and prostration. The patient may experience fluctuating hearing loss, tinnitus, and a sensation of fullness in the affected ear or ears during an attack of vertigo. After a severe attack, patients may feel lightheadedness for half a day or so but they are completely well by that evening or the next day. In the early stage of the disease, the episodic vertigo may occur once every yar or so but sometimes as far apart as 5-10 years. In most patients the disease affects only one ear (85%). Should the second ear be involved, it usually happens within 36 months.

Diagnostic labyrinthotomy through the oval window may reveal the characteristics of endolymph rather than perilymph. However, the fluid obtained from the round window is perilymph.

Audiometric testing will document fluctuating hearing loss usually in the low frequencies with high SISI score, type II Bekesy, and little or no tone decay. Caloric testing or ENG will demonstrate hypofunction of the vestibular labyrinth in the affected ear.

Crisis of Tumarkin

This is a variant of Ménière's disease in which the patient loses his extensor powers and falls to the ground during a sudden, severe, and short episode of vertigo. He is completely conscious throughout this episode and recovers promptly afterwards.

Lermoyez's Syndrome

This is generally agreed to be a rare variant of Ménière's disease in which there is a dramatic restoration of hearing after an episodic attack of vertigo. Recurrence of this phenomenon can be expected.

Glycerol Test

It has been speculated that administration of glycerol in the dose of 1.2 mL/kg of body weight with addition of an equal amount of physiologic saline, to a patient with Ménière's disease with sensorineural hearing loss, tinnitus, and sensation of fullness in the ear, has improved the symptoms within an hour with maximum effects in 2-3 hours. After 3 hours, the symptoms slowly return.

Acoustic Neurinoma

Acoustic neurinoma accounts for 80% of angle tumors. Most patients with acoustic neurinomas complain of unsteadiness rather than episodic vertigo. However, it has been reported that about 10% of acoustic neurinoma patients presented with episodic vertigo of Ménière's type.

Classically, the caloric reaction is markedly depressed. Audiometric studies reveal a high frequency hearing loss in many of the cases. However, other audiometric patterns are not uncommon. The patients usually have a disproportionately low discrimination score, low SISI score, high tone decay score, and type III or type IV Bekesy score. Definitive diagnosis of this disease is made from x-rays of the internal auditory canal.

Vestibular Neuronitis

Vestibular neuronitis, of which 50% is unilateral and 50% bilateral, usually follows an upper respiratory tract infection. A patient experiences a sudden onset of vertigo with nausea, vomiting, the sensation of blacking out accompanied by severe unsteadiness. This severe attack can last from days to weeks. Cochlear symptoms are surprisingly absent and without associated neurologic deficits. When seen initially, the patient has spontaneous nystagmus to the contralateral side. A caloric test would show marked hypofunction of the labyrinth. Audiometric tests and x-rays of the internal auditory canal are within normal limits.

After the acute episode has subsided, which may take weeks, the patient continues to experience a slight sensation of light-headedness for some time, particularly in connection with sudden movements. In some patients, there may be an exacerbation of the acute attack within 3-6 months. The caloric reaction may remain mildly hypoactive for the duration of the patient's life. The acute episode may also be followed by a period of positional vertigo of the benign paroxysmal type.

Bacterial Labyrinthitis

This is usually a complication of an ear infection which makes the diagnosis obvious.

Nonbacterial "Labyrinthitis"

Some patients have presented with a sudden attack of vertigo associated with nausea, vomiting, and sensorineural hearing loss without a previous history of vertigo. There is no associated neurologic deficit. Is this the first attack of Ménière's disease, or does the patient have an acoustic neurinoma? Perhaps it is viral labyrinthitis or thrombosis of one of the

labyrinthine vessels? Should one consider the possibility of a round or oval window rupture? A careful evaluation and long follow-up may reveal the mystery.

Cupulolithiasis

Cupulolithiasis is a term used by Schuknecht to designate positional vertigo of the benign paroxysmal type. The symptoms include sudden attacks of vertigo precipitated by certain head positions. These attacks have been reported to be prompted by sudden movement of the head to the right or left or by extension of the neck when looking upward. The sensation of vertigo is always of short duration even when the provocative position is maintained. Diagnosis can be confirmed by positional testing which will indicate positional nystagmus with latency and fatigability.

Etiologies include degenerative changes, otitis media, labyrinthine concussion, previous ear surgery, and occlusion of the anterior vestibular artery. Histopathologically, otoconia has been found deposited in the posterior semicircular canal ampulla. It is probable that some of these deposits have resulted from postmortem degeneration of the utricular otolithic membrane. Treatment of this disease is symptomatic and by reassurance.

Congenital Syphilis

The majority of these patients develop hearing loss during young adulthood. This hearing loss is of a flat sensorineural type. When the onset of hearing loss occurs in adulthood, the loss in both ears is asymmetric and fluctuates with the episodic vertigo and tinnitus. However, when the onset occurs in childhood, the hearing loss is abrupt, bilaterally symmetric, and more severe.

Acquired syphilis seldom leads to hearing loss but neurosyphilis and congenital syphilis (38% of congenital syphilis) frequently give rise to hearing loss, with bilateral hearing loss being more prevalent than unilateral hearing loss. Vertigo in congenital syphilis is episodic, similar to Ménière's disease.

These patients usually have a positive Hennebert's sign, i.e. postive fistula test without any demonstrable fistula along with a normal external auditory canal and tympanic membrane. The positive fistula test indicates an abnormally mobile footplate. The patient also may demonstrate Tullio's phenomenon (see Chaps. 8 and 23.)

Histopathologically, mononuclear leukocytic infiltration is evident with obliterative endarteritis. Inflammatory fibrosis and endolymphatic hydrops are present. Osteolytic lesions are often seen in the otic capsule. Interstitial keratitis is another common manifestation of congenital syphilis.

Cogan's Syndrome

(See Chap. 23: Syndromes and Eponyms).

Vertigo Due to Whiplash Injury

Patients often complain of dizziness following a whiplash injury. In some cases, there is no physiologic evidence for this complaint. In others, ENG has documented objective findings such as spontaneous nystagmus. The onset of dizziness often occurs 7-10 days following the accident, particularly upon head movements toward the side of the neck most involved in the whiplash. The symptoms may last for months or years after the accident.

Otologic examination is usually normal. Audiometric studies are normal unless there is associated labyrinthine concussion. Vestibular examination can reveal spontaneous nystagmus or positional nystagmus with the head turned in the direction of the whiplash. The use of ENG is essential in evaluating these patients.

Temporal Bone Fracture and Labyrinthine Concussion

Transverse Fracture

Since a transverse fracture destroys the auditory and vestibular function, the patients have no hearing or vestibular response in that ear. When seen initially, they present with spontaneous nystagmus to the contralateral side and are severely vertiginous, very much like a recently postoperative labyrinthectomized patient. The severe vertigo subsides after a week or so and the patients remain mildly unsteady for 3-6 months depending on their age and athletic inclination. The patients also may have labyrinthine concussion in the opposite ear. During the acute phase, they usually fall toward the involved side.

Longitudinal Fracture

Longitudinal fracture constitute 80% of the temporal bone fractures. In this type of fracture, there is usually bleeding into the middle ear, with perforation of the tympanic membrane and disruption of the tympanic ring. Hence, the patient has a conductive hearing loss as well as a sensorineural high-frequency hearing loss fro the concomitant labyrinthine concussion. Dizziness is mild and there may be no vertigo exceopt during positional testing.

Labyrinthine Concussion

Labyrinthine concussion is secondary to head injury. The patient complains of mild unsteadiness or light-headedness particularly with change of head position. Audiometric testing reveals a high-frequency hearing loss. The ENG may show spontaneous or position nystagmus; occasionally, the caloric response is hypoactive.

Multiple Sclerosis

Multiple sclerosis is one of the more common neurologic diseases encountered in a clinical practice. Vertigo is the presenting symptom of multiple sclerosis in 7-10% of the patients or eventually appears during the course of the disease in up to one-third of the cases. The patient usually complains of unsteadiness along with vertigo. Diagnosis of the disease depends on other signs of demyelination. Vertical nystagmus, bilateral internuclear

ophthalmoplegia, and ataxic eye movemnts ar other clues to this disease. Charcot's triad (nystagmus, scanning speech, intention tremor) may be present.

Vascular Insufficiency

Vascular insufficiency can be a common cause of vertigo among the over 50-year-olds as well as in patients with diabetes, hypertension, or hyperlipidemias. The following syndromes have been recognized among patients with vascular insufficiency:

Labyrinthine Apoplexy

Labyrinthine apoplexy is due to thrombosis of the internal auditory artery of one of its branches. The symptoms include acute vertigo with nausea and vomiting. Hearing loss and tinnitus may or may not occur.

Wallenberg Syndrome (Also see Chap. 23)

Wallenberg's syndrome also is known as the lateral medullary syndrome secondary to infarction of the lateral portion of the medulla which is supplied by the posterior inferior cerebellar artery. This syndrome is believed to be the most common brain stem vascular disorder. The symptome include:

1. Vertigo, nausea, vomiting, nystagmus.

2. Ataxia, falling toward the side of the lesion.

3. Loss of the sense of pain and temperature sensations on the ipsilateral face and contralateral body.

4. Dysphagia with ipsilateral palate and vocal cord paralysis.

5. Ipsilateral Horner's syndrome.

Subclavian Steal Syndrome (Also see Chap. 23)

The subclavian steal syndrome is characterized by intermittent vertigo, occipital headache, blurred vision, diplopia, dysarthria, pain in the upper extremity, loud bruit or palpable thrill over the supraclavicular fossa, a difference of at least 20 mm Hg in systolic blood pressure between the two arms, and a delayed or weakened radial pulse. The blockage can be surgically corrected.

Anterior Vestibular Artery Occlusion

The symptoms include:

1. Sudden onset of vertigo, without deafness.

2. Slow recovery followed by months of positional vertigo of the benign paroxysmal type.

3. Histologically, utricular macula, the cristae of the lateral and superior semicircular canals and the superior vestibular nerve show signs of degeneration.

4. This symptom complex was first described by Lindsay and Hemenway in 1956.

Basilar-Vertebral Insufficiency

The symptoms include vertigo, hemiparesis, visual disturbances, dysarthria, headache, and vomiting. These symptoms are a result of a drop in blood flow to the vestibular nuclei and surrounding structures. The postero- and anteroinferior cerebellar arteries are involved. Tinnitus and deafness are unusual symptoms.

Drop attacks without losing consciousness are characteristic of basilar-vertebral insufficiency. These drop attacks can be precipitated by neck motion.

Cervical Vertigo

Cervical vertigo can be caused by cervical spondylosis as well as by other etiologies. Cervical spondylosis in turn can be brought about by degeneration of the intervertebral disc. As the disc space narrows, approximation of the vertebral bodies takes place. With mobility, the bulging of the annulus is increased causing increased traction on the periosteum to which the annulus is attached and stimulating proliferation of bone along the margins of the vertebral bodies to produce osteophytes.

Barre believed that the symptoms of cervical spondylosis (including vertigo) are due to irritation of the vertebral sympathetic plexus, which is in close proximity to the vertebral artery. Laskiewicz claims that spondylosis irritates the periarterial neural plexus in the wall of the vertebral and basilar arteries leading to contraction of the vessels. Temporary ischemia then gives rise to vertigo. Others claimed that the loss of proprioception in the neck can give rise to cervical vertigo. Emotional tension, rotation of the head, and extension of the head can cause the neck muscle (including the scalenus anticus) to be drawn tightly over the thyrocervical trunk and subclavian artery, compressing these vessels against the proximal vertebral artery. In elderly individuals a change from the supine to the upright position may give rise to postural hypotension which in turn may cause vertebral-basial insufficiency. The aortic arch syndrome and subclavian steal syndrome also may cause cervical vertigo.

Symptoms

1. Headache, vertigo.

2. Syncope.

3. Tinnitus and loss of hearing (usually low frequencies).

4. Nausea and vomiting (vagal response).

5. Visual symptoms such as flashing lights are not uncommon. This is due to ischemia of the occipital lobe which is supplied by the posterior cerebral artery, a branch of the basilar artery.

6. Physical examination may reveal a supraclavicular bruit in one-third of the patients.

All of the above symptoms usually appear when the head or neck assumes a certain position or change of position.

Treatment

Proper posture, neck exercises, cervical traction, heat massage, anesthetic infiltration, and immobilization of the neck with a collar temporarily are all good therapeutic measures. If traction is required it can be given as a few pounds horizontally for several hours at a time. In cervical spondylosis without acute root symptoms, heavy traction (100 lb) for 1-2 minutes continuously or 5-10 minutes intermittently is considered by some as more effective.

Vertiginous Epilepsy

Cortical vertigo can either be severe and episodic like Ménière's disease, or may manifest as a mild unsteadiness. It is usually associated with hallucinations of music or sound. The patient may exhibit "daydreaming", and purposeful or purposeless repetitive movements. Motor abnormalities such as chewing, lip smacking, and facial grimacing are not uncommon. The patient may experience an unusual sense of familiarity (deja vu) or a sense of strangeness (jamais vu). Should the seizure discharge spread beyond the temporal lobe, grand mal seizures may ensue.

Vertigo in Migraine

Vertebral-basilar migraine is due to impairment of circulation of the brain stem. The symptoms include vertigo, dysarthria, ataxia, paresthesia, diplopia, diffuse scintillating scotomas, or homonymous hemianopsia. The initial vasoconstriction is followed by vasodilatation giving rise to an intense throbbing headache, usually unilateral. A positive family history is obtained in over 50% of these patients. Treatment of migraine includes Fiorinal, ergot derivatives, and methysergide (Sansert). (Sansert has the tendency to cause retroperitoneal fibrosis.)

Miscellaneous

Streptomycin

Streptomycin sulfate is believed to destroy the cristae of the semicircular canals and not the maculae of the utricle and saccule.

Ewald's Law

1. When a semicircular canal is stimulated, it tends to elicit nystagmus in its own plane.

2. The horizontal semicircular canal is maximally stimulated by an ampullopetal flow (i.e. endolymph directed towards the ampulla). The superior semicircular canal and posterior semicircular canal are maximally stimulated by amullofugal flow (i.e. endolymph directed away from the ampulla).

3. When a semicircular canal is maximally stimulated, it elicits a quick phase of nystagmus to its own side.

Coriolis Forces

Any body which is set in motion off the surface of the earth towards a distant earth target is deflected from a straight course because the earth is rotating. When a subject is on a machine which is rotating at a steady velocity, any movement of the body, head, or limbs that are made about an axis different from, and at an angle to, that of the machine, generates extra forces, some of which are also described as Coriolis forces or acceleration.

Coriolis Phenomenon

When the subject's head is tilted about an axis which is perpendicular to the main axis of rotation, spatial disorientation will be experienced.

Internuclear Ophthalmoplegia

Internuclear ophthalmoplegia is a disturbance of the lateral movements of the eyes, characterized by a paralysis of the internal rectus on one side and weakness of the external rectus on the other side. In testing, the examiner has the patient follow his finger, first to one side, and then to the other, as in testing for horizontal nystagmus. Internuclear ophthalmoplegia is recognized when the adductive eye (III nerve) is weak while the abducting eye (VI nerve) moves normally and displays a coarse nystagmus (? vestibular nuclei involvement). The pathology is in the medial longitudinal fasciculus. When the disorder is bilateral it is pathognomonic of multiple sclerosis, when unilateral, one should consider a tumor or vascular process.