Quick Reviews: Dizziness

Dizziness is a common complaint and too often the symptom is attributed to an "inner ear problem." Numerous cochleovestibular, neurologic, cardiovascular, metabolic, ocular, and systemic diseases are capable of eliciting the sensation of dizziness; the ear, however, is responsible for only 50-60 percent of the known causes of dizziness

Anatomy and Physiology of Labyrinth:

The semicircular canals

Anatomy: The semicircular canals (lateral, posterior, and superior) lie at right angles to one another and are encased within the otic capsule of the temporal bone. Each canal contains perilymph which bathes the membranous ducts within the canal. Each canal has an ampulla or a widening of the canal at the point it communicates with the vestibula. The crista ampullaris is located in each respective ampulla. The crista contains specialized neuroepithelium with hair cells imbedded in a gelatinous material to form the cupula. Physiology: The cupula extends into the ampulla to detect rotational movement of the endolymph.

The maculae of the utricle and saccule:

Anatomy: the utricle and saccule are located in the bony vestibula. The endolymph contained within the utricle and saccule is continuous with the cochlear duct and the membranous duct of the semicircular canals. Both the saccule and utricle have maculae which contain hair cells embedded in a gelatinous material covered with calcium carbonate crystals (otoconia.)
Physiology: the maculae are responsive to the effect of gravity and linear movement.

Posture and movement: The labyrinth acts as a bilateral frequency modulator. Acceleration, deceleration, or rotational movement of the head leads to excitation of one system and inhibition of the opposite. Sensory output is relayed to the vestibular nuclei and pathways for central interpretation.

The manifestation of disease: In order to maintain posture and move through the environment in an orderly and safe fashion, appropriate information from three sensory modalities is important: the visual axis, proprioception in the joints and muscles, and the labyrinths. This input is integrated in the brain stem and cerebellum to adjust posture and motor activity and to maintain orientation. A failure in sensory input, poor integration in the central nervous system, or diseased support systems (cardiovascular and metabolic), can provoke the feeling of disorientation or dizziness. The interdependence of so many organ systems accounts for the large differential diagnosis of dizziness.

COMMON CAUSES OF DIZZINESS

Cochleovestibular system

BPPV:

Symptoms: A 15-30 second episode of vertigo induced by position change.
Signs: The positional nystagmus may be observed by purposefully inducing the position change and observing the eyes. Nystagmus tends to be toward the involved ear and exhibits latency and fatiguability.
Laboratory: ENG documents the presence of positional nystagmus.
Treatment: Reassurance and vestibular exercises. Singular nerve section for the recalcitrant and disabled patient.

Viral labyrinthitis:

Symptoms: acute onset of vertigo usually associated with nausea and vomiting, Hearing loss may or may not be present.

Signs: Unilateral hearing loss, spontaneous nystagmus with the slow component toward the involved ear. Pass pointing and falling to the side of the lesion in the acute stages.
Laboratory: Audiograms may show sensorineural hearing loss. ENG reveals nystagmus and often a caloric weakness.
Treatment: The condition is self-limiting and the most effectivetherapy includes rest and sedation. Meclizine or diazepam are often beneficial.

Vestibular neuritis:
Symptoms and signs: Presentation and physical findings can be identical to those of viral labyrinthitis except that hearing loss is not present.
Laboratory: ENG will show a unilateral weakness in the involved ear.
Treatment: Same as for viral labyrinthitis. Vestibular neuronitis can be recurrent. A vestibular nerve section will cure the vertigo and preserve hearing.

Acoustic neurinoma. Patients rarely present with true vertigo secondary to acoustic neurinoma but more frequently complain of unsteadiness, tinnitus, or hearing loss.

Labyrinthine concussion:
Symptoms: Vertigo or dizziness with or without hearing loss following severe head injury.
Signs: spontaneous nystagmus with the slow component toward the involved ear may be present along with a sensorineural hearing loss.
Laboratory: A basic audiogram will document the presenc eof sensorineural hearing loss. The ENG may document the presenceof a caloric weakness or a positional nystagmus. Polytomography of the skull base or CT scanning is indicated when skull fractures are suspected.
Treatment includes the use of antivertiginous medications in the initial stages. Labyrinthectomy or vestibular nerve section are indicated for a patient with symptoms persistent beyond six months.

Orthostatic hypotension:
Symptoms: Patient complains of light-headedness or thesensation of faintness upon sitting upright or standing.
Signs: Include an increased pulse rate or a fall of systolicpressure with the change in position.
Laboratory: none.
Treatment: Reassurance and a change in medication when indicated

Hyperventilation:
Symptoms: Light-headedness, fullness in the throat, perioral paresthesias, chest tightness, anxiety.
Physical examination: Normal.
Laboratory: If the patient is symptomatic, an arterial blood gas may reveal the presence of hypocarbia.
Treatment: reassurance along with methods to increase the pCO2 (paper bag).

Vertebrobasilar insufficiency.
Symptoms: These patients can experience true vertigo but this is usually associated with visual disturbances, dysarthria, disorientation, and ataxia.
Signs: Physical examination may be normal between attacks.
Laboratory: CT scan is usually normal. Findings on arteriography can be variable.
Treatment: ASA, Persantine, anticoagulation in severe cases.