Approach to Dizziness and Vertigo

What is Dizziness?
■Definition Varies
■Description Varies
■Pathophysiology Varies
■Management Strategies Vary

Classification according to *Complaint*
■Vertigo
■Pre-syncope
■Dysequilibrium
■Light headedness

Dizziness is a Non-specific symptom
■Vestibular
■Cardiovascular
■Neurologic
■Metabolic
■Psychiatric

Data to be gathered about patient's dizziness
■Onset
■Duration
■Consistency
■Context of dizziness
■Associated symptoms

Causes of Dizziness (Hain, 1999)

■Otologic	50%
■Central or neurologic	5%
■Medical	5%
■Ophthalmologic	5%
■Psychologic	15%
■Multiple disorders/Vague	20%

Types of Dizziness
History
Describe the sensation
How did it begin?
How long did it last?
How frequently did it occur?
What induced it?
Associated Symptoms

Medications

■Past history of migraine
■Head trauma
■Vascular risk factors- smoking, HTN, DM

Categories
■True vertigo
■Near- syncope
■Disequilibrium
■"other"

■Vertigo – felt in the head
■Dysequilibrium – felt in the legs
■Lightheadedness – floating sensation
■Psychogenic – vague complaints

What is vertigo?
■Vertigo is:
–A type of dizziness
–Specifically, a sensation of movement typically characterised by feelings of rotation or spinning

Vertigo can be objective or subjective
■Objective vertigo:
–the patient perceives that the environment is moving round him/her
■Subjective vertigo:
–the patient feels himself/herself moving in a static environment

True Vertigo
■Illusory sense of motion
■Patient feels dizzy in the head
■Spinning or rotary sensation
■Episodic
■Indicates a lesion w/n the vestibular system anywhere from the inner ear and vestibular nerves to the many central pathways

Near Syncope
■Sense of " black out" or "pass out"
■Hypovolemic states
■Medication side effects
■Systemic infections
■Arrythmias
■Vascular disease
■Pulmonary embolus
■Obstruction to cardiac output

Dizziness…
■Passing out
■Fainting
■Candle being slowly exhausted
■Light-headedness

Near Faint Dizziness(Pre-syncope)
■A light headedness sensation
■The sensation experienced before fainting

Dysequilibrium
■Sense of being "dizzy in the body" rather than in the head
■More common in the elderly- falling down sensation
■Often Multifactorial- problems in hearing, vision, cerebellar function, peripheral neuropathy, Parkinson's disease and polypharmacy
"Other"
■Can't describe dizziness in further detail
■Hyperventilation
■Anxiety
■Depression
■"constant" dizziness

Pathophysiology
A. Pre-syncope
•Cardiac Disease
•Orthostatic Hypotension
•Vasovagal Episodes
•Hyperventilation
■- *Reduce Blood Flow in the Brain*

Pathophysiology
B. Psychophysiologic Dizziness
•Anxiety/Panic Disorder
•Phobic Dizziness
•Mechanism unclear
•Impaired central integration of sensory signals
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Pathophysiology
C. Disequilibrium
•Bilateral vestibulopathy
•Peripheral Neuropathy
•Cerebellar Ataxia
•Parkinson's Disease
•Hydrocephalus
•Multi-infarct Syndrome

■Loss of peripheral sensory inputs
■Central lesions invoving the motor centers the basal ganglia, cerebellum and cortex

Pathophysiology
D.Vertigo
•Benign paroxysmal positional vertigo
•Acute peripheral vestibulopathy
•Meniere's syndrome
•Migraine
•Other peripheral causes of vertigo
•VBI
•C-P angle tumor
•Other central causes of vertigo
Vertigo can be of central or peripheral origin
Vertigo of peripheral origin: causes
Vertigo of central origin: causes

■An imbalance in the vestibular tone
■Loss of Peripheral Input due to damage in the labyrinth or vestibular nerve or unilateral vestibular nuclear or vestibulocerebellar activity
The vestibular system is the dominant sensory input guiding balance
Displacement of sensory cells in the ampullae informs on head movements
Activation of cells in the sacculus and utriculus maculae inform on head position

BPPV
■the most common cause of vertigo.
■ It results from free floating calcium carbonate crystals (normally attached to the utricular macule) that inadvertently enter the long arm of the posterior semicircular canal. With positional change, the crystals move within the endolymph and displace the cupula

BPPV
■Vertigo associated with change in head position
■No auditory symptoms
■48% idiopathic
■Pathophysiology-Otolith displacement
Presyncope Diagnosis

▪The diagnosis of orthostatic hypotension requires a documented drop in mean blood pressure of more than 10 to15 mm of mercury when the patient moves from a lying to standing position.
▪In patients with autonomic insufficiency, the pulse rate will remain unchanged despite the hypotension.

▪Although physical examination and routine electrocardiograms will identify most serious heart diseases, intermittent arrhythmias are not identified.
▪Any patient with episodic presyncope of unknown cause should undergo EKG monitoring to search for sinus pauses, sinus bradycardia, atrial fibrillation, and sustained supraventricular tachycardia.
▪Diagnosis of hyperventilation rests on identifying the characteristic associated symptoms in the setting of anxiety dyspnea.

Psychophysiologic Dizziness Diagnosis
■Lightheadedness, giddiness
■Associated symptoms of acute and chronic anxiety
■Panic Attacks

Disequilibrium Diagnosis
■Assess gait,strength,coordination,reflexes and sensory function
■Broad-based gait- cerebellar disorder
■Bilateral vestibular loss- ±hearing loss
■↓ or absent response to caloric and rotational stimulation

Vertigo Diagnosis
■BPPV- find positional nystagmus
■Head Hanging positon test(Dix- Hallpike)

Vestibular Neuronitis Diagnosis
■Spontaneous prolonged vertigo
■Unilateral peripheral vestibular loss
■Absence of associated neurological symptoms and signs
■Head-thrust test
■If available- Electronystagmography

Meniere's Disease Diagnosis
■Fluctuating hearing loss
■Vertigo
■Cochlear site of hearing loss
■Caloric testing ±
Meniere's Disease

■Spontaneous episodic vertigo
■Sensorineural hearing loss
■At least one of the ffg.:
a. Tinnitus
b. Aural fullness

VBI Diagnosis
■Abrupt vertigo w/o apparent precipitating factor
■Ends abruptly with minimal or no residual symptoms

■Nearly always associated symptoms
■Visual loss, diplopia,dysarthria, weakness or numbness
■MRA rapidly becoming the procedure of choice for assessing V-B circulation

Vertigo: Clinical Differentiation

Spontaneous Nystagmus

Peripheral
Visually suppressed
With auditory symptoms
(+) Vertigo
Direction fixed
Conjugate

Central
Not suppressed
(-) auditory symptoms
(+)/(-) Vertigo
Any direction
Dysconjugate

Vestibular vs. Psychogenic

Features	Vestibular	Psychogenic
Duration	min-hrs	"flash"
Frequency	1 wk,days,mos	several/ day
Head movement	worsen	no effect
Ataxia during spell	+	–
Hyper-ventilation test	unlike spell	mimics spell

Treatment Aspects

Treatment is dependent on vertigo type

Medications for Acute Vertigo

Drug	Dosage	Route
Dimenhydramine	50 - 100 mg qid	PO, IM, IV, PR
Diphenhydramine	25 - 50 mg tid to qid	PO, IM, IV
Meclizine	12.5 - 25 mg bid to qid	PO
Promethazine	25 mg bid to qid	PO, IM, IV, PR
Hydroxyzine	25 - 100 mg tid to qid	PO, IM

(adapted from Lerner, 1995)

Canalith Repositioning Procedure
■BPPV Therapy
■Office based therapy
■Full explanation about the procedure prior to initiation of Tx
■May need to be repeated if unsuccessful on 1st attempt

Treating the cause: a simple particle repositioning manoeuvre effectively treats most BPPV cases

Management:
Symptomatic Relief
■Histamine analogue- Betahistine
■Histamine antagonist- Cinnarizine
■TCAs
–Imipramine
■BZDs
–Diazepam, lorazepam
■Ca^{+2} blockers
– flunarizine,cinnarizine
■Blood oxygenators
–Almitrine-raubasine

Vascular and neuronal Central/Perepheral compensation
■Piracetam-enhanced control of the cerebral cortex on the subordinated vestibular centers
■facilitate vestibular compensatory processes by improving blood supply and glucose utilization, increasing the efficacy of neurotransmission and promoting interhemispheric transfer of information
Balance requires information of similar intensity from both vestibular systems
Central vertigo results from a dysfunction in central processing
Peripheral vertigo results from a dysfunction in vestibular system functioning
Vestibular suppressants suppress vestibular function in both ears

Treatment Vestibular Neuronitis
■Steroids- Methylprednisolone
■Vestibular suppressants
–Antihistamines
–Anticholinergics
–Benzodiazepines
■Antiemetics
–Metoclopramide
–Chlorpromazine

Presyncope Treatment-
Orthostatic Hypotension

■Remove offending drug
■Correct cause of blood volume depletion
■ salt intake, elastic stockings
■Salt retaining steroids –fluorocortisone

■Midodrine-α-1 adrenergic agonist

Vasovagal Presyncope
■Reassurance and education
■ dietary salt intake
■B-blockers, ACEI,Midodrine, SSRIs, not convincing efficacious

Cardiac Disease Presyncope
■Treat underlying heart disease
■Improve cardiac output
■Correct arrhyhmias
■Pacemaker

Hyperventilation
■Educate and reassure patient
■Vigorous exercise program
■Psychotherapy
■SSRI or TCA for panic disorders
■Avoid prolonged benzodiazepines

Psychophysiologic dizziness
■TCAs, SSRI, benzodiazepines
■Behavioral Therapy and supportive psychotherapy
■Exercise program to improve diminished physical fitness
■Make patient responsible for their therapy program

Dysequilibrium
■Improve sensory function and adjustment to sensory loss
■Physical therapy for gait and balance retraining
■Treat peripheral neuropathy
■Remove ototoxic drugs
■Stop alcohol for alcohol cerebellar degeneration
■Treat Parkinsons- levodopa, NPH- V-Pshunting
■Avoid tranquilizers-impair central integration of sensory informations

Rehabilitation exercises can encourage vestibular adaptation and compensation
▪Effective recovery from vertigo often requires neural reorganisation and adaptation
▪Vestibular rehabilitation exercises aim to promote adaptation and compensation of the nervous system
▪They instigate sensory conflicts to promote neural learning
▪Rehabilitation exercises are particularly useful when:
-Medical therapy is ineffective
-Patients have poor central integration or motor function

■Thank you for your attention!