VERTIGO-NEUROPHYSICIAN’S PERSPECTIVES

PROF. A.V. SRINIVASAN, MD, DM, PhD, DSc, FRCP (LON) F.A.A.N, F.I.A.N, EMERITUS PROFESSOR- THE TAMILNADU DR MGR MEDICAL UNIVERSITY
FORMER PROFESSOR OF NEUROLOGY AND HEAD- INSTITUTE OF NEUROLOGY
MADRAS MEDICAL COLLEGE

CHENNAI AUGUST 10, 2014
Vertigo denotes a hallucinatory sensation of movement.

Balance the imbalance

Being ignorant is not so much a shame as being unwilling to learn.
Prevalence of Vertigo and Giddiness

- 5% of Patients visiting the General Practitioner
- 10% of Patients visiting the Otorhinolaryngologist


“My Opinions are founded on knowledge but modified by experience”
What causes vertigo?

- Contradictory information from:
  - The vestibular system (ears)
  - The visual system (eyes)
  - The Proprioceptive system (muscles, joints)

*Daroff RB, ‘Faintness, Syncope, Dizziness and Vertigo’ IN Harrison’s Principles of Internal Medicine, 14th edn, 105*

*Experience can be defined as yesterday’s answer to today’s problems*
Anatomical Structures

Vestibular System (Motion Detectors)

SCC
Angular

VOR
1. Imbalance
2. Bi-lateral Symmetric hypoactive.

OTOLITH
Linear head position (gravity and body)

Being ignorant is not so much a shame as being unwilling to learn
Physiology

Vestibular System (Motion Detectors)

- Detection of Head motion
- Detection of Head position
- Control of the VOR

Being ignorant is not so much a shame as being unwilling to learn
Saccades and Vestibular Ocular motor adaptation

- Saccade adaptation
- Cross-axis adaptation
- On axis adaptation: New findings
- Changes in the dynamic properties of adapted saccades
- A role for forward models in the control of saccades
- Behavioral deficits in saccade adaptation with cerebellar lesions
- Vestibulo-Ocular Reflex (VOR) adaptation
- VOR adaptation induced by position and alternate error signals
- Compensatory saccades as an adaptation to abnormal peripheral VOR function
- Physiological correlates of saccade and VOR adaptation

*Imagination is more Important than Knowledge*
Types of Dizziness

1. Light headedness
2. Multiple sensory deficit
3. Cervical spine disease
4. Imbalance
5. Faintness
6. Acute vertigo with nausea and worse with head motion
7. Vertigo present only with head motion

Being ignorant is not so much a shame as being unwilling to learn
Sites of Vertigo

The secret of walking on water is
Knowing where the stones are
Causes of Vertigo
(Peripheral Vestibular - arises in Vestibule)

- Benign Paroxysmal Positional Vertigo
- Meniere’s Disease
- Labyrinthitis
- Head Injuries & Surgical Trauma
- Pressure Vertigo

Memory, the daughter of attention,
is the teeming mother of knowledge - Martin Tupper
Causes of Vertigo
(Intermediate Vestibular - arises in Vestibular Nerve)

• Vestibular neuronitis
• Acoustic neuroma
• Drugs
Causes of Vertigo
(Central vestibular - arises in Vestibular Nuclei)

- VBI (Vertebrobasilar Insufficiency)
- Arteriosclerosis
- Cervical Spondylosis
- Whiplash injuries of Neck
- Brain Tumors

Success is a prize to be won. Action is the road to it.
Chance is what may lurk in the shadows at the road side.
Non-Vestibular Causes of Vertigo

- Ocular vertigo
- Anemia
- Cardiovascular (orthostatic hypotension)
- Cerebrovascular disorders
- Psychogenic
- Brain tumors

- Head injuries
- Epilepsy
- Multiple sclerosis
- Hypoglycemia
- Migraine

In any field, find the strangest thing and explore it
Another classification of vertigo

• Paroxysmal Vertigo - sudden attack comes on quickly, lasts for a short time
• The single attack - sudden intense attack fading away slowly
• Chronic vertigo - not severe
• Positional vertigo - occurs following sudden movements of head in certain positions
• Dizzy spells - lasting a few seconds occurring irregularly

What is mind no matter; What is matter never mind
**DIAGNOSIS OF VERTIGO**

**Medical History**

- Description of symptoms by patient
- Classification of vertigo attacks (Which type, how debilitating, frequency, duration, vegetative symptoms)
- Influencing circumstances (Injuries, drugs taken, stress, eating pattern, Illnesses)
- Secondary symptoms (Tinnitus, Hearing loss, Headache, nausea/ vomiting)

_Biswas A., ‘Neurotological History Taking’ IN An Introduction to Neurotology, 1998, 8-11_

*Take time to think; it is the source of power*
*Take time to read; it is the foundation of wisdom*
*Take time to work; it is the price of success*
Clinical Examination

• Duration of Vertigo
  - < 5 seconds – Hypoactive Labrynth
  - 5 – 90 seconds – BPPV
  - 90 seconds – 20 minutes – Migraine, TIA
  - 20 minutes – 24 hours – Meniere’s disease
  - Days – CNS Disorder, Vestibular Neuronitis

Character gets you out of bed; Commitment moves you to action; Faith, Hope and Discipline follow through to completion
Clinical Examination

• Neurological Examination

• Expanded vestibular Examination
  ➢ Study of Nystagmus
  ➢ Hallpike – Dix Test
  ➢ Test of vestibular imbalance or hypofunction
    • Head thrust test
    • Dynamic Visual Acuity
    • Head shaking test
Schematic representation of a putative model of the pathomechanism of DBN. We propose that all patients with DBN share a final common pathway (disinhibition of the SVN and neurons of the Y group). The ocular motor circuitries involved are the two smooth pursuit eye movement pathways (I, II) and the vertical gaze-holding pathway (III). The different lesion sites that can lead to DBN are shown in red (1 - 3). See Discussion for details (from [65]).


Nystagmus Videos

Gaze evoked Nystagmus

Rebound Nystagmus

Vertical Nystagmus
Spontaneous nystagmus

Eyes open

No nystagmus

eyes closed

No

Nystagmus

Peripheral disorder

Nystagmus

eyes closed

No

Nystagmus

Peripheral or central disorder

Nystagmus

eyes closed

No

Nystagmus

Central disorder

Truth comes out of error sooner than that of confusion
**Induced nystagmus**

- Positional nystagmus
  Any nystagmus that occurs when the head is in position other than normal upright

- Positioning nystagmus
  occurs when change of head position and used to diagnose BPPV

The Truth is Fear & Immorality are two of the greatest inhibitors of Performance to progress
### Differentiation of Peripheral and Central Vertigo

<table>
<thead>
<tr>
<th>Sign / Symptom</th>
<th>Peripheral</th>
<th>Central</th>
</tr>
</thead>
<tbody>
<tr>
<td>(Brainstem)</td>
<td>(Labyrinth) or Cerebellum)</td>
<td></td>
</tr>
<tr>
<td>Direction of associated nystagmus</td>
<td>Unidirectional; fast phase opposite lesion*</td>
<td>Bidirectional or unidirectional</td>
</tr>
<tr>
<td>Purely horizontal nystagmus without torsional component</td>
<td>Uncommon</td>
<td>Common</td>
</tr>
<tr>
<td>Vertical or purely present torsional nystagmus</td>
<td>Never present</td>
<td>May be</td>
</tr>
<tr>
<td>Visual fixation</td>
<td>Inhibits nystagmus and vertigo</td>
<td>No inhibition</td>
</tr>
</tbody>
</table>

* In Meniere’s disease, the direction of the fast phase is variable.

Daroff R. B., 'Faintness Syncope, Dizziness and vertigo IN Harrisons Principles of Internal Medicine, 14th Edition, 105

“Fools Admire but of men of sense approve”
**Differentiation of Peripheral and Central Vertigo**

<table>
<thead>
<tr>
<th>Sign / Symptom (Brainstem or Central)</th>
<th>Peripheral (Labyrinth)</th>
<th>Central Cerebellum</th>
</tr>
</thead>
<tbody>
<tr>
<td>Severity of vertigo</td>
<td>Marked</td>
<td>Often mild</td>
</tr>
<tr>
<td>Direction of spin</td>
<td>Toward fast phase</td>
<td>Varied</td>
</tr>
<tr>
<td>Direction of fall</td>
<td>Toward slow phase</td>
<td>Variable</td>
</tr>
<tr>
<td>Duration of symptoms</td>
<td>Finite (minutes, days, weeks) but recurrent</td>
<td>May be chronic</td>
</tr>
<tr>
<td>Tinnitus and /or deafness</td>
<td>Often present</td>
<td>Usually absent</td>
</tr>
<tr>
<td>Associated central common abnormalities</td>
<td>None</td>
<td>Extremely</td>
</tr>
<tr>
<td>Common causes</td>
<td>Infection (labyrinthitis),</td>
<td>Vascular,</td>
</tr>
<tr>
<td>demyelinating,</td>
<td>Meniere's, neuronitis,</td>
<td></td>
</tr>
<tr>
<td></td>
<td>ischemia, trauma, toxin</td>
<td>neoplasm</td>
</tr>
</tbody>
</table>

*Daroff R. B., ‘Faintness Syncope, Dizziness and vertigo IN Harrisons Principles of Internal Medicine, 14th Edition, 105*
Clinical Pearls

Peripheral Disease

Acute Peripheral Vestibular Disease
- Distortion of Sounds
- ANS symptoms

Meniere’s Disease
- Episodic Tinnitus
- HOH

Peripheral less predictable in CNS
- Postural Aggravation
Clinical Pearls…contd

Viral Labyrinthitis or Internal. AUD. Artery

CNS
- Acute Vertigo
- Hearing Loss
- Headache
- LOC
- Seizures
- Hemiparesis
- Cranial nerve

Drugs
- Benzodiazepine
- Anti-histaminics
- Scopolamine
"We Sometimes think we have forgotten something when in fact we never really learned it in the first place”

Imp.Your Memory Skills
### Clinical and genetic features of familial episodic ataxia syndromes

<table>
<thead>
<tr>
<th></th>
<th>EA1</th>
<th>EA2</th>
<th>EA3</th>
<th>PATX/EA4</th>
<th>EA5</th>
<th>EA6</th>
<th>EA7</th>
<th>Other episodic ataxias</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Online Mendelian Inheritance in Man</strong></td>
<td>160120</td>
<td>108500</td>
<td>606554</td>
<td>606552</td>
<td>601949</td>
<td>600111</td>
<td>unassigned</td>
<td>unassigned</td>
</tr>
<tr>
<td><strong>Attack duration</strong></td>
<td>seconds/minutes</td>
<td>hours</td>
<td>1 min-6h</td>
<td>brief</td>
<td>hours</td>
<td>hours/days</td>
<td>hours/days</td>
<td>hours/days</td>
</tr>
<tr>
<td><strong>Age of onset (years)</strong></td>
<td>2 – 15</td>
<td>2 – 20</td>
<td>1 – 42</td>
<td>23 – 60</td>
<td>3 – teens</td>
<td>5</td>
<td>teens</td>
<td>after 30</td>
</tr>
<tr>
<td><strong>Myokymia</strong></td>
<td>usual</td>
<td>no</td>
<td>usual</td>
<td>no</td>
<td>no</td>
<td>no</td>
<td>no</td>
<td>no</td>
</tr>
<tr>
<td><strong>Nystagmus</strong></td>
<td>no</td>
<td>usual</td>
<td>occasional</td>
<td>usual</td>
<td>usual</td>
<td>no</td>
<td>no</td>
<td>usual</td>
</tr>
<tr>
<td><strong>Epilepsy</strong></td>
<td>occasional</td>
<td>infrequent</td>
<td>occasional</td>
<td>occasional</td>
<td>usual</td>
<td>yes</td>
<td>no</td>
<td>no</td>
</tr>
<tr>
<td><strong>Migraine</strong></td>
<td>no</td>
<td>usual</td>
<td>usual</td>
<td>no</td>
<td>no</td>
<td>no</td>
<td>no</td>
<td>variable</td>
</tr>
<tr>
<td><strong>Tinnitus</strong></td>
<td>infrequent</td>
<td>no</td>
<td>usual</td>
<td>occasional</td>
<td>no</td>
<td>no</td>
<td>no</td>
<td>no</td>
</tr>
<tr>
<td><strong>Acetazolamide</strong></td>
<td>occasional</td>
<td>usual</td>
<td>usual</td>
<td>no</td>
<td>transient</td>
<td>no</td>
<td>no</td>
<td>occasional</td>
</tr>
<tr>
<td><strong>Vertigo</strong></td>
<td>no</td>
<td>yes</td>
<td>yes</td>
<td>yes</td>
<td>yes</td>
<td>no</td>
<td>yes</td>
<td>usual</td>
</tr>
<tr>
<td><strong>Weakness</strong></td>
<td>no</td>
<td>yes</td>
<td>no</td>
<td>no</td>
<td>no</td>
<td>no</td>
<td>yes</td>
<td>occasional</td>
</tr>
<tr>
<td><strong>Dysarthria</strong></td>
<td>no</td>
<td>yes</td>
<td>yes</td>
<td>yes</td>
<td>no</td>
<td>no</td>
<td>yes</td>
<td>usual</td>
</tr>
<tr>
<td><strong>Inheritance</strong></td>
<td>autosomal</td>
<td>autosomal</td>
<td>autosomal</td>
<td>autosomal</td>
<td>autosomal</td>
<td>autosomal</td>
<td>autosomal</td>
<td>autosomal</td>
</tr>
<tr>
<td><strong>Chromosome locus</strong></td>
<td>12q13</td>
<td>19p13</td>
<td>1q42</td>
<td>unknown</td>
<td>2q22-q23</td>
<td>5p</td>
<td>19q13</td>
<td>unknown</td>
</tr>
<tr>
<td><strong>Mutated gene</strong></td>
<td>KCNA1</td>
<td>CACNA1A</td>
<td>unknown</td>
<td>unknown</td>
<td>CACNB4</td>
<td>SLC1A3</td>
<td>unknown</td>
<td>unknown</td>
</tr>
<tr>
<td><strong>Mutant protein</strong></td>
<td>Kv1.1</td>
<td>Cav2.1</td>
<td>unknown</td>
<td>unknown</td>
<td>Cav2.1</td>
<td>excitatory</td>
<td>unknown</td>
<td>unknown</td>
</tr>
</tbody>
</table>

*amino acid transporter type 1*
### Summary of the clinical features, pathophysiology, etiology, site of lesion, and current treatment options of downbeat and upbeat nystagmus.

<table>
<thead>
<tr>
<th></th>
<th>Direction of the nystagmus (quick phase)</th>
<th>Waveform (slow phase)</th>
<th>Special features</th>
<th>Sites of lesion</th>
<th>Etiology</th>
<th>Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Downbeat nystagmus</td>
<td>Downward, may be diagonal at lateral gaze</td>
<td>Jerk, linear, increasing or decreasing velocity of the slow phase</td>
<td>Increase in intensity during lateral and downward gaze</td>
<td>Cerebellum (bilateral floccular hypofunction); lower brain-stem</td>
<td>Degenerative cerebellar disorders, ischemia, idiopathic; often associated with bilateral vestibulopathy</td>
<td>4-aminopyridine, 3,4-diaminopyridine, baclofen, clonazepam</td>
</tr>
<tr>
<td>Upbeat nystagmus</td>
<td>Upward</td>
<td>Jerk, linear, increasing or decreasing velocity of the slow phase</td>
<td>Increase in intensity during upward gaze</td>
<td>Medulla, pontomesencephalic and cerebellum</td>
<td>Ischemia, bleeding, Wernicke’s encephalopathy</td>
<td>Since often transient, treatment not necessary; baclofen, 4-aminopyridine</td>
</tr>
</tbody>
</table>

*Whatever the Mind can conceive and believe, the mind can achieve* - Napoleon Hill
Balance Tests

- Postural tests
  - Romberg test
  - Unterberger test
  - Babinski-weill test
  - Barany Pointing test

- Eye movement test
  - Nystagmus

Adapted from Biswas A., ’Clinical tests in Neurotology’ IN An Introduction to Neurotology, 1998, 13-25

Character gets you out of bed; Commitment moves you to action; Faith, Hope and Discipline follow through to completion
Every thing should be made as simple as possible; but not simpler
**UNTERBERGER’S TEST**

- Patient closes eyes and stretches arms out in front
- Walks on spot for a minute
- The knees raised as high as possible
- Patients with vertigo will start to turn his axis in particular direction

“Healthy Mind and Healthy expression of Emotion go hand in Hand”
BARANY'S PAST POINTING TEST

- doctor holds an object in front of the patient
- patient closes his/her eyes and points to object several times
- Deviation to one side in pointing occurs in patients with vertigo

Give us the **GRACE** to accept with serenity the things that cannot be changed
the **COURAGE** to change the things that should be changed
and the **WISDOM** to know the difference
Babinsky- Weill Test

Patient closes his eyes and takes 5 steps forward and 5 steps back for 30 seconds.

Patient with vertigo starts to walk in a star shape.

Nature, Time and Patience are the 3 great physicians.
Management of vertigo

- Pharmacotherapy
- Adaptation exercises
- Surgery

“Social Isolation is in itself a pathogenic Factor for disease production”
I WANT ..

• Fewer attacks every month
• Attacks should not be as bad as before
• Attacks should not last long

A Vertigo Patient

A true commitment is a heart felt promise to yourself from which you will not back down
Pharmacotherapy (Antivertigo drugs)

Vertigo suppression drugs
- Anticholinergics
- Sympathomimetics
- Antihistaminics
- Psychotherapeutic drugs
- Antiemetic phenothiazines

Drugs modifying underlying pathology
- Cerebroactive drugs
- Vasodilators
- Diuretics
- Corticosteroids
- Antibacterial drugs

Thinking is the hardest work there is, which is probable reason why so few engage in it.
Site of action of anti-vertigo drugs

- Labryinth – Diuretic and corticosteroids
- Blood flow – Vasodilators
- Reticular formation – Sympathomimetics
- Reticular formation (cholinergic pathway) – Antiemetic
- Antiemetic (Antihistamines and Anticholinergics)
- GABAnergic suppression – Psychotherapeutic drugs of vestibular nuclei

Mind is the great level of all things; human thought is the process by which human ends are ultimately answered
Phenothiazines  
(Prochlorperazine, Thiethylperazine)

• Prochlorperazine is less sedating than some other phenothiazines but drowsiness still occurs
• Also causes hypotension, Parkinsonian side effects

  -- Curley JWA, E N T Journal, 1984, 65, 555-560

• “The drug which most commonly causes parkinsonism in general practice is Prochlorperazine”

  -- Chaplin S, Geriatric Medicine, 1989, Feb, 13-14

Serious, sincere, systematic studies, surely secure supreme success
Anxiolytics (Tranquilizers)
(Benzodiazepines such as diazepam, Lorazepam)

- No effect on the underlying vertigo
- Helps patient endure the symptoms by allaying anxiety
- Many side effects drowsiness and sedation, dependence and addiction abuse potential, psychomotor impairment, memory loss, interactions with alcohol

Harris T, Ear Nose Throat J, 1984, 65, 551-5

“Men of Genius Admired: Men of Wealth envied women of power feared but only women of character are trusted”
Diuretics
(e.g. Furosemide, Hydrochlorothiazide)

• Used in vertigo and Meniere’s disease
• Reduce the volume of endolymph by promoting urine flow and reducing fluid retention.
• Use mainly associated with electrolyte imbalance


“Motivation is the Spark that lights the Fire of Knowledge and fuels the engine of Accomplishment”
Antihistamines

*Cinnarizine, Flunarizine, Cyclizine*

- Drowsiness and blurred vision (Difficult for patients who drive or operate machinery)
- Delay normal vestibular compensation process
- Cinnarizine and Flunarizine act via calcium antagonism, unspecific action may cause side effects
  - Weight gain & depression (serotonergic effects)
  - Extrapyramidal symptoms (dopaminergic effects)
  - G.I. upset


Marriage and Private Practice are the two extinguishers of science
Betahistine

*Trusted therapy for more than*

41 million

*Vertigo patients worldwide*

At twenty the will rules

At thirty the intellect

At forty the Judgment
Betahistine - Summary

• **Pharmacokinetics:** Rapid and complete absorption after oral route

• **Pharmacology:** It is a H1 agonist and H3 receptor antagonist. It increases cochlear and cerebral blood flow and regulates firing activity of vestibular nuclei.

• **Dose:** 24-48 mg /day

• **Indication:** vertigo, meniere’s syndrome

• **Contraindications:** not known

• **Precaution for use:** pheochromocytoma, peptic ulcer, bronchial asthma

“The True Art of Memory is The Art of Attention”
### Global evaluation (n=29)

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Excellent-good rating in % of patients by</th>
</tr>
</thead>
<tbody>
<tr>
<td>Efficacy</td>
<td>100%</td>
</tr>
<tr>
<td>Tolerance</td>
<td>100%</td>
</tr>
<tr>
<td>Effect on associated symptoms</td>
<td>95%</td>
</tr>
</tbody>
</table>

*Patients* | *Physicians* |
------------|--------------|
100%        | 100%         |
100%        | 100%         |
95%         | 95%          |

*Bradoo RA, Ind. J. Otolaryngol H N S, 2000, 52 (2), 151-8*

Whatever the Mind can conceive and believe, the mind can achieve.
Figure I: The top row images show **HYPOPERFUSION** in the left temporal lobe prior to therapy & the bottom row images of the same patient show complete **NORMALISATION OF PERFUSION** after 4 weeks of Betahistine therapy 16 mg three times daily.

**Krishna BA, Kirtane MV, Neurology India, 2000, 48, 255-9**
Figure II: The top row images show HYPOPERFUSION in the right inferior cerebellar region prior to therapy. The bottom row images show almost complete NORMALISATION OF PERFUSION following 2 weeks of Betahistine therapy 16 mg three times daily.

Reference Image

Pre-Betahistine Therapy (27.02.1998)
No. 791

Post-Betahistine Therapy (10.03.1998)
No. 1950

Krishna BA, Kirtane MV, Neurology India, 2000, 48, 255-9
Figure III: The top row images show a well-defined focalized HYPOPERFUSION in the right parieto-occipital region prior to therapy. The bottom row images show almost complete NORMALISATION OF PERFUSION of this region after 3 weeks of Betahistine therapy 16 mg three times daily.
Do’s and don’ts in encouraging vestibular compensation

**Encourage**
- Alertness
- Active & passive movements
- Large Support Surface
- Fine motor task
- Visual stimuli
- General care

**Avoid**
- Sedation
- Immobility
- Dark environment
- Solitude standing


Many Ideas grow better when transplanted into another mind than in the one where they sprang UP
LISTEN not to contradict or confute
Nor to Believe and Take for Granted
but TO WEIGH AND CONSIDER

THANK YOU