Vertigo
Moving beyond labyrinthine suppression

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Vertigo

Moving beyond labyrinthine suppression

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- One of 10 Global Medical “Hotspots”
  
  *Newsweek, October 30, 2006
  
  “India’s best public hospital. Last year the government-run hospital, with about 2,000 beds, treated 3.5 million people, achieving mortality and infection rates comparable to the best facilities in the developed world….”
Vertigo

Moving beyond labyrinthine suppression

“The patho-physiological approach to Vertigo”
Vertigo

Moving beyond labyrinthine suppression

“The patho-physiological approach to Vertigo”

-- Patho-physiological approach to Dx

-- Patho-physiological approach to Rx
Dizziness & Vertigo

Challenges

A clinical diagnosis

limited knowledge base

multi-specialty origins

varied interpretations
different things to different people
VERTIGO - causes

OTOLOGIC
- BPPV
- Menieres
- Endolymphatic Hydrops
- Vestibular neuronitis
- Labyrinthitis
- Vascular
- Autoimmune
- Trauma
- Iatrogenic

NEUROLOGICAL
- Vascular
  - Migraine
  - VBI
  - CVA
- Epilepsy
- Rx (iatrogenic)
- Tumor/trauma/thyroid
- Infections
- Glial diseases
- Ocular

MEDICAL
- Haematological
- Cardiovascular
- Metabolic
- Iatrogenic
- Hyperventilation

MISCELLA EOS
- Ocular
- Cervical
- Multisensory deficit syndrome
Pathophysiological Approach to Diagnosis - I

- Is it Vertigo?
- Is it Central or Peripheral?
- What is the patho-physiological mechanism?
- what is the etiology?
Pathophysiological Approach to Diagnosis

- Is it Vertigo?
- Is it Central or Peripheral?
- What is the patho-physiological mechanism?
- What is the etiology?

1. Avoid the Top Down Approach
Pathophysiological Approach to Diagnosis - I

- Is it Vertigo?
- Is it Central or Peripheral?
- What is the patho-physiological mechanism?
- what is the etiology?

XXX 1. Avoid the Top Down Approach

2. Separate the wheat from the chaff
   xxx --- secondary Anxiety
   xxx -- pharmacologic vestibular suppression
Pathophysiological Approach to Diagnosis - I

- Is it Vertigo?
- Is it Central or Peripheral?
- What is the pathophysiological mechanism?
- What is the etiology?

---------------------------------------------------------------------------------------------------------------------------

- Hallucination of motion
- or
- Disagreeable sensation of loss of spatial orientation

Syncope
Epilepsy
Periodic paralysis
Cataplexy
Ataxia
Pathophysiological Approach to Diagnosis - I

- Is it Vertigo?
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- What is the etiology?
Pathophysiological Approach to Diagnosis

- Is it Vertigo?
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- What is the etiology?

---

**AFFERENTS**

- Visual
- Vestibular
- Proprioceptive/Somatosensory

**CENTRAL**

- Vestibular nucleus
- Brain Stem Reticular Formation

**EFFERENTS**

- Oculomotor
- Spinal motor
- Autonomic system
- Cerebral Cortex
Pathophysiologica}l Approach to Diagnosis

- Is it Vertigo?
- Is it Central or Peripheral?
- What is the pathophysiological mechanism?
- What is the etiology?

AFFERENTS
- Visual
- Vestibular
- Proprioceptive/Somatosensory

CENTRAL
- Vestibular nucleus
- Brain Stem Reticular Formation

EFFERENTS
- Oculomotor
- Spinal motor
- Autonomic system
- Cerebral Cortex
Peripheral or Central?

**Peripheral**
- Vertigo rotatory
- Autonomic Sx.
- assoc. Nystagmus
- Nystagmus
  - unidirectional
  - fast and slow phase
  - inhibition by fixation
- Hearing loss/ tinnitus
- compensation

**Central?**
- Imbalance / Rotatory vertigo
- Autonomic symptoms variable
- Nystagmus may be absent
- Nystagmus atypical
- diplopia/ dysarthria/ paraesthesia
  - aura
  - vascular disease (ABCD2)
- no compensation
Peripheral or Central?

Three Step Bedside Oculomotor Examination

- Head Impulse
- Nystagmus
  - unidirectional
  - fast and slow phase
  - inhibition by fixation

- Test for Skew
  (Cover-Uncover Test)
ACUTE VERTIGO

Peripheral or Central?

Three Step Bedside Oculomotor Examination

- Head Impulse
- Nystagmus
  - unidirectional
  - fast and slow phase
  - inhibition by fixation

Test for Skew
(Cover-Uncover Test)
Peripheral or Central?

Three Step Bedside Oculomotor Examination

- Head Impulse
- Nystagmus
  - unidirectional
  - fast and slow phase
  - inhibition by fixation

HINTS to STROKE

Acute Stroke (< 72 hrs)

- HINTS --- 100% Sensitive; 96% Specific
- DW MRI --- 72% Sensitive

Pathophysiologival Approach to Diagnosis - I

- Is it Vertigo?
- Is it Central or Peripheral?
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- What is the etiology?
Pathophysiological Approach to Diagnosis - I

- Is it Vertigo?
- Is it Central or Peripheral?
- What is the pathophysiological mechanism?
- What is the etiology?

HALLUCINATION OF MOVEMENT

LOSS OF SPATIAL ORIENTATION
Pathophysiologic Approach to Diagnosis

- Is it Vertigo?
- Is it Central or Peripheral?
- What is the pathophysiologic mechanism?
- What is the etiology?

HALLUCINATION OF MOVEMENT
Assymetry of vestibular afferent input or its central connections

LOSS OF SPATIAL ORIENTATION
Inadequacy of vestibular input
overload/mismatch of afferent inputs/lack of central integration
Pathophysiological mechanism

**HALLUCINATION OF MOVEMENT**

Assymetry of vestibular afferent input or its central connections

**LOSS OF SPATIAL ORIENTATION**

- Inadequacy of vestibular input
- Overload/ mismatch of afferent inputs/ lack of central integration
Pathophysiological mechanism

HALLUCINATION OF MOVEMENT

Assymetry of vestibular afferent input or its central connections

- momentary-
  - transient stimulation/depression

- hours -
  - reversible metabolic pathology

- days -
  - irreversible loss of vestibular function

LOSS OF SPATIAL ORIENTATION

- Inadequacy of vestibular input
- overload/ mismatch of afferent inputs/ lack of central integration
Pathophysiological mechanism

**Etiology**

**HALLUCINATION OF MOVEMENT**
Assymetry of vestibular afferent input or its central connections

- momentary -
  - *transient stimulation/depression*

- hours -
  - *reversible metabolic pathology*

- days -
  - *irreversible loss of vestibular function*

**Loss of spatial orientation**

- Inadequacy of vestibular input
- Overload/ mismatch of afferent inputs/ lack of central integration

**Causes**

- Canalolithiasis- BPPV
- Vertebrobasilar ischaemia
- Endolymphatic hydrops
- Labyrinthitis
- Vestibular neuronitis
- Ototoxicity
- Multi-sensory deficit syndrome
- Brainstem disorder
Pathophysiological Approach to Diagnosis - II

**Disease evolution**

**ACUTE VESTIBULAR LESION**
Pathophysiological Approach to Diagnosis - II

Disease evolution

ACUTE VESTIBULAR LESION

RESOLUTION

COMPENSATION
Pathophysiological Approach to Diagnosis - II

Disease evolution

ACUTE VESTIBULAR LESION

RESOLUTION

COMPENSATION
  Sensory Substitution
  Tonic rebalancing of neural activity
  Recalibration
  Habituation
Pathophysiological Approach to Diagnosis - II

Disease evolution

ACUTE VESTIBULAR LESION

RESOLUTION

RECURRENCE

COMPENSATION

BREAKDOWN of COMPENSATION
Pathophysiological Approach to Diagnosis - II

**Disease evolution**

- **ACUTE VESTIBULAR LESION**
  - RESOLUTION
  - RECURRENCE
  - COMPENSATION
    - BREAKDOWN of COMPENSATION

**Secondary Effects**
- Anxiety ; Depression
- Labyrinthine suppression consequent to vestibular sedatives.
Clinical Presentations
- Acute lesion
- Acute lesion with partial or complete resolution; Recurrent Activity
- Acute lesion with partial or complete compensation; Breakdown of compensation
- Secondary Effects - Pharmacologic Labyrinthine Suppression/Anxiety/Depression

Pathophysiologic Approach to Diagnosis - II

Disease evolution

ACUTE VESTIBULAR LESION

RESOLUTION

RECURRENT

COMPENSATION

BREAKDOWN of COMPENSATION
Vestibular Diagnosis

Dx- Patho-physiological rather than etiological
45 M, no risk factors,
sudden rotatory vertigo lasting 10 days, vomiting ++
  gradual improvement since, some residual giddiness, some positional vertigo
  HINTS-( HI +/-, N - , Skew -) Cerebellar signs -ve , Cr. Nerves –ve, Dix-Hallpike +ve

Dx- Destructive peripheral lesion
  with inadequate compensation
  and canalolithiasis

D/D ? Labyrinthine infarct
  ? Vestibular neuronitis
Vestibular Treatment

Rx - Patho-physiological rather than etiological

Dx - Destructive peripheral lesion
    with inadequate compensation
    and canalolithiasis

D/D ? Labyrinthine infarct
    ? Vestibular neuronitis
Patho-physiology based Dx

I
- Is it Vertigo?
- Is it Central or Peripheral?
- What is the pathophysiological mechanism?
- what is the etiology?

II

ACUTE VESTIBULAR LESION

RESOLUTION

Disease Evolution

COMPENSATION

RECURSENCE

BREAKDOWN of COMPENSATION
Patho-physiology based Rx
Patho-physiology based Rx

ACUTE VESTIBULAR LESION  Labyrinthine suppression

RESOLUTION

COMPENSATION

RECURRENCE

BREAKDOWN of COMPENSATION
Patho-physiology based Rx

ACUTE VESTIBULAR LESION Labyrinthine suppression

RESOLUTION No RX COMPENSATION

RECURRANCE BREAKDOWN of COMPENSATION
Intratympanic gentamycin
-When?

ACUTE VESTIBULAR LESION
Labyrinthine suppression

RESOLUTION

RECURRENCE

COMPENSATION
BREAKDOWN of COMPENSATION

Disease modifying treatment
Intratympanic gentamycin

- When?

ACUTE VESTIBULAR LESION

Labyrinthine suppression

RESOLUTION

COMPENSATION

BREAKDOWN of COMPENSATION

Recurrence

Disease modifying treatment
Intratympanic gentamycin -When?

**ACUTE VESTIBULAR LESION**

- Labyrinthine suppression

**RESOLUTION**

**RECURRANCE**

**COMPENSATION**

**BREAKDOWN of COMPENSATION**

---

*Disease modifying treatment*

**Removal of unstable labyrinth**

- Intratympanic Gentamycin
- Vestibular Neurectomy
Intratympanic gentamycin -When?

ACUTE VESTIBULAR LESION

RESOLUTION

RECURRANCE

COMPENSATION

BREAKDOWN of COMPENSATION

Labyrinthine suppression

Disease modifying treatment

Vestibular rehabilitation

Removal of unstable labyrinth

- Intratympanic Gentamycin
- Vestibular Neurectomy
Patho-physiology based Rx

**ACUTE VESTIBULAR LESION** Labyrinthine suppression

- **RESOLUTION**
- **COMPENSATION**
- **RECURRENCE**
- **BREAKDOWN of COMPENSATION**

*Disease modifying Rx*  
- BPPV  
- Migraine  
- Perilymph fistula

*Vestibular rehabilitation*

*Removal of unstable labyrinth*
- Intratympanic Gentamycin  
- Labyrinthectomy / Vestibular Neurectomy
BPPV
CUPULOLITHIASIS

CANALOLITHIASIS
CUPULOLITHIASIS

- no latency
- persistent vertigo (as long as position is maintained)
- Semont manoeuvre

CANALOLITHIASIS

- latency
- transient
- Epley manoeuvre and Semont manoeuvre
62 yr. Male

sudden severe vertigo- lasted few days

-folowed by intermittent transitory vertigo ppt by change in position

- PTA-moderate left SN loss

Dx- left destructive lesion with probable Canalolithiasis
  ? Vascular
  ? Labyrinthitis

Dix- Hallpike –ve
HSCC canalolithiasis
HSCC canalolithiasis
HSCC canalolithiasis
HSCC canalolithiasis

- HSCC particle repositioning manoeuvre
  "Barbecue manoeuvre"

- Lie with diseased ear up- Vanucchi Manoeuvre
HSCC cupulolithiasis
Letters to the Editor

A warning to users of disposable tonsillectomy instruments

Dear Sirs,

We wish to draw readers’ attention to a potential hazard of using disposable tonsillectomy instruments.

Growing concern regarding the transmission of new variant Creutzfeldt-Jakob disease during tonsillectomy has resulted in the widespread adoption of disposable tonsillectomy sets. One of the instruments used routinely is the Bowles-Davis mouth gag, which has a tongue blade that is split in the middle to accommodate the endotracheal tube. The distal ends of the split tongue blades are held together by a welded metal loop, which prevents herniation of the endotracheal tube (Figure 1).

We experienced a case where this metal loop became detached intra-operatively, and came to lie within the posterior pharynx. Fortunately, the problem was discovered intra-operatively, and the loop was removed atraumatically and the patient suffered no harm.

We, however, feel that this highlights a possible weakness in the manufacture of these instruments, which creates the potential for aspiration of a loose foreign body after extubation. Furthermore, a missing instrument component could easily be missed, unless specifically looked for.

We wish to strongly advise readers to thoroughly inspect disposable tonsillectomy instruments both pre- and post-operatively, to avoid this potentially disastrous complication.

Department of Otolaryngology, Farnborough Hospital, Kent BR6 8ND, UK.

Clinical effect of canal plugging for paroxysmal positional vertigo.

A Letter Otol 2000;114:59-62

Dear Sirs,

We read with interest the article by Suzuki et al., as well as their previous experimental work on the subject. The report immaculately documented the nystagmus and its changes with position and time and correlated these with a pathophysiological explanation accounting for a diagnosis of right LSC cupulolithiasis. The patient was treated by plugging of the implicated LSC and this was claimed to have prevented further stimulation of the cupulolithiasis of the left LSC. However, the authors explained that “plugging creates a closed space” which prevents the canal between the plugged site and the cupula, thus effectively blocking the perilymphatic fluid. This effectively needed to be a specific explanation for positional vertigo secondary to cupulolithiasis. It is not clear as to how such a creation of a “closed space” would in any way inhibit the gravitational pull on a cupulolithiasis cupula which is loaded with otoconia and has a resultant removal specific gravity.

Going by the clinical results presented it is in fact quite possible that the patient’s clinical improvement resulted from the induced labyrinth suffering significant surgical damage leading to a loss of function of the right vestibular labyrinth LSC.

We are ourselves currently treating a similar patient with acute horizontal positional transient nystagmus, presumably secondary to a left LSC cupulolithiasis. The patient has remained refractory to conservative measures and repositioning manoeuvres. We have nevertheless proceeded to canal plugging due to the lack of a definite pathophysiological explanation for its efficacy in cupulolithiasis. We eagerly await the authors’ explanation regarding the same.

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References

Author’s reply

The main point of the question is whether or not the resolution of vertigo is due to dysfunction of the left semicircular canal (LSC). In fact the author had the same question, since left nystagmus was constantly seen some period after surgery. This indicates that the operated canal suffered from hypofunction and I was prepared for this before surgery. I understand that canal plugging to the human semicircular canal may well induce hypofunction of the more, or less, since the human membranous labyrinth is thin and hence may be easily affected by even manipulation. Therefore, I presume that mild hypofunction may have occurred after surgery in this particular case, too.

However, I do not think that the vertigo and rotational effect as I indicated in the paper, the position of the nystagmus and vertigo extremely important if the hypofunction of LSC should suppress these, it must be total or near-total loss of LSC function. Furthermore, considering that post-operative paralytic nystagmus and dizziness subsided in a rather short period of time, I think that the effect of suppression of inner ear function was not minimal.
Perilymph fistula
Perilymph fistula

ACUTE VESTIBULAR LESION

RESOLUTION ------No RX------
Perilymph fistula

**VARIED PRESENTATIONS**

**VERTIGO** - imbalance/ positional Sx/ severe vertigo

**HEARING LOSS** - Sensori-neural/ fluctuant/ conductive

**History**
- h/o trauma
- h/o pressure change
  - weight lifting
  - sneezing/ valsalva
  - flying/ diving

**Fistula test**
- Tymanometry
- Flood test
Disease modifying surgery

Superior Semi-circular canal dehiscence

Vascular loop
Vertigo syndromes and mechanisms in migraine

A. Thakar, F.R.C.S., C. Anjaneyulu, M.S., R. C. Deka, M.S.

Abstract
This paper attempts to define and categorize the vertigo associated with migraine. A retrospective chart review of 344 cases of vertigo identified 19 cases with headaches characteristic of migraine as per strictly defined criteria (International Headache Society, 1988). Four distinct types of vertiginous syndromes were noted. The commonest syndrome (Group I) manifested transient episodes of imbalance with additional momentary subjective rotary vertigo worsened by movement. The attacks lasted a few hours and evaluation in the inter-episode interval demonstrated no vestibular deficit. Group II manifested transient objective rotary vertigo of from 10 minutes to a few hours but no demonstrable permanent vestibular deficit. Group III displayed symptoms and signs characteristic of benign paroxysmal positional vertigo (BPPV) and Group IV manifested a permanent unilateral labyrinthine weakness. Causation of vertigo by migraine was implied in 10 of 19 cases where the headache and vertigo occurred simultaneously and in two other cases where the vertigo improved with anti-migraine prophylactic treatment.

Four distinct and characteristic vertigo syndromes have been noted with migraine. Their spectrum ranges from a transient reversible dysfunction to a more permanent destruction, and includes involvement of both the peripheral and the central vestibular systems.

Key words: Migraine; Vertigo
Migrainous Vertigo
Vertigo syndromes and mechanisms in migraine

A. Thakar, F.R.C.S., C. Anjaneyulu, M.S., R. C. Deka, M.S.

- abnormal neurologic processing of sensory inputs

- vasospasm
  (followed by vasodilatation)
Vertigo syndromes and mechanisms in migraine

A. Thakar, F.R.C.S., C. Anjaneyulu, M.S., R. C. Deka, M.S.

- Abnormal neurologic processing of sensory inputs
- Vasospasm
  (followed by vasodilatation)

- Episodic periods of sense of imbalance, motion provoked vertigo, excessive perception of motion, nausea
- Rotatory vertigo - few hours, with/without hearing loss/tinnitus
- Canalolithiasis
- U/L destructive lesion
Vertigo syndromes and mechanisms in migraine

A. Thakar, F.R.C.S., C. Anjaneyulu, M.S., R. C. Deka, M.S.

- Episodic periods of sense of imbalance, motion provoked vertigo, excessive perception of motion, nausea
- Rotatory vertigo - few hours, with/without hearing loss/ tinnitus
- Canalolithiasis
- U/l destructive lesion

Headache – throbbing/ hemicranial
Aura
Photophobia / phonophobia

Family history
Motion sickness

Therapeutic Response
Episodic periods of sense of imbalance, motion provoked vertigo, excessive perception of motion, nausea

- rotary vertigo - few hours, with/without hearing loss/ tinnitus

**TREATMENT**

1. Rx to Abort Attack
   - Sedation/ Sleep/
   - ??Steroids/ ?Oxygen

2. Avoid triggers

3. Prophylactic Medical Rx-
   - Propranolol

- Canalolithiasis

- u/l destructive lesion
Intratympanic gentamycin
Intratympanic gentamycin
-When?

ACUTE VESTIBULAR LESION

RESOLUTION

COMPENSATION

BREAKDOWN of COMPENSATION

Disease modifying treatment

Vestibular rehabilitation

Removal of unstable labyrinth

- Intratympanic Gentamycin
- Vestibular Neurectomy
Intratympanic gentamycin

-When?

**ACUTE VESTIBULAR LESION**

RESOLUTION

RECURRENCE

**COMPENSATION**

BREAKDOWN of **COMPENSATION**

*Disease modifying treatment*

Vestibular rehabilitation

*Removal of unstable labyrinth*

- Intratympanic Gentamycin
- Vestibular Neurectomy
Intratympanic gentamycin

-When?

- Any unstable labyrinth not responding to medical therapy

- attacks due to *labyrinthine activity* and not due to breakdown of compensation
Intratympanic gentamycin

-When?

- Any unstable labyrinth not responding to medical therapy

- attacks due to *labyrinthine activity* and not due to breakdown of compensation

- --------------------------------------------

XXX compensation expected to be poor

XXXX only hearing ear
Dosage schedule (based on hearing status)

- **Serviceable hearing**
  - (< 50 db)
  - weekly inj.
  - 4 injections

- **Poor hearing**
  - (> 50 db)
  - alternate day inj.
  - 4 injections

Hearing and vestibular evaluation prior to every injection
PTA worsening of > 10 db
SDS worsening of > 15%

Vestibular Symptoms
Spontaneous/ head-shaking nystagmus

-------------> defer next injection
how

Tuberculin syringe

0.65ml Gentamycin (40mg/ml) + 0.35ml Sodabicarb

26mg/ml pH- 6.8 “Buffered solution”

0.3-0.5ml (7.8-13mg)
Imbalance
(without relation to definitive episodes of vertigo)
Vertigo Control

Complete vertigo control = 88.1%

Control = \frac{\text{vertigo fr post Rx}}{\text{vertigo fr pre Rx}} \times 100

0 - Complete control
1-40 - Substantial
41-80 - Limited
81-120 - Insignificant
>120 - Worse

Complete vertigo control = 88.1%
Hearing Status

Hearing Worse - 7.1%
Intratympanic gentamycin—current place in therapy of vertigo

IT genta v/s

surgical labyrinthectomy/ Vest neurectomy

- Simple, “safe”, inexpensive
- vertigo control almost comparable
- hearing preservation often possible
Labyrinthectomy
Vestibular Neurectomy
Pathophysiological Approach to Diagnosis

- Is it Vertigo?
- Is it Central or Peripheral?
- What is the pathophysiological mechanism?
- What is the etiology?
Pathophysiological Approach to Diagnosis - II

ACUTE VESTIBULAR LESION

RESOLUTION

RECURRENCE

COMPENSATION

BREAKDOWN of COMPENSATION
Pathophysiological Approach to Treatment

ACUTE VESTIBULAR LESION

RESOLUTION

COMPENSATION

RECURRENCE
Disease modifying Rx
Removal of unstable labyrinth

BREAKDOWN of COMPENSATION
Vestibular rehabilitation
Thank You

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“3rd window” syndrome / SCC dehiscence syndrome
Outpatients microscope supine head turned to opp. side

Ø post inf quadrant
Ø carbolic acid application
Ø penetrate with needle - inject SLOWLY - 0.3-0.5 ml

Ø patient maintains position for 20 minutes

Tuberculin syringe
26G spinal needle
RESULTS
Dr. Sonia Suprabha, Prof. R. C. Deka. Dr. A. Thakar

42 patients

Age-18 - 65 yrs (mean:39.8 yrs)

DOSAGE SCHEDULE

daily/ alternate day- 20
weekly -22

No. of injections

4 injections 35
3 injections 3
8 injections( 2 courses) 4

Follow up : 3 mths -18 mths

(6 mths - 35 patients)
Why? -“Selectively” vestibulotoxic

Dose related actions

- vestibular sensory cells
- dark cells in crista ampullaris
- ?? Stria vascularis
- outer hair cells

Idiosyncratic reaction

Pharmacokinetics basis of selective vestibulotoxicity

Vestibular Ablation v/s Vestibular Alteration
pharmacokinetics

- Inner ear penetration-
  - round window
  - annular ligament of OW
  - preformed pathways ME-Inner ear

  **VARIABLES** - thickness of rw membrane, adhesions in rw niche, position of head and dependency of rw niche, patency of eustachian tube, rate of endolymph and perilymph turnover

- Perilymph $t_{1/2}$ 12 hours

  -----------------------------------------------

delayed effect

vestibulotoxic - and cochleotoxict -effects usually manifested 4-5 days following instillation
Dosage protocol

Toth & Parnes (1995)

**Shotgun protocol**
- inpatients
- three daily injections X 4 consecutive days
  - 21 cases
  - vertigo control 80%
  - hearing deterioration 51%
  - dead ear - 5/21

**Titration protocol**
- outpatients
- 4 weekly injections - Rx stopped or deferred if hearing loss
  - 16 cases
  - vertigo control 80%
  - hearing deterioration 17%
  - dead ear - 1/16
VESTIBULAR REHABILITATION THERAPY  Cawthorne (1944) and Cooksey (1946)

Head And Eye Movements in a Seated Position
1. Look upwards and downwards
2. Look to the right and to the left
3. Move your index finger towards the eye and then away from the eye (first slowly and then quickly)
4. With your eyes open, drop your head downwards and then roll it backwards (first slowly and then quickly)
5. With your eyes open, roll your head to the left and to the right (first slowly and then quickly)
6. Repeat exercises 4 and 5 with your eyes closed

Head and Body Movements in a Seated Position
1. Place an object on the floor and pick it up, bending and extending your torso (looking at the object the entire time)
2. Bend your torso and move the object to the front and behind your knees.

Exercises in an Orthostatic Position
1. Sit and then rise to the orthostatic position with your eyes open
2. Repeat exercise 1 with your eyes closed
3. Repeat exercise 1 making a turn to the right in the orthostatic position
4. Repeat exercise 1 making a turn to the left in the orthostatic position

Activities for Improving Balance
1. Walk while rotating your neck to the left and to the right
2. In the orthostatic position, make brisk 90 degree turns with your body (first with your eyes open and then closed)
3. Go up and down the stairs (using the railing if necessary)
4. In the orthostatic position, stand on one foot (first with the right foot and then the left), with your eyes open and then closed
5. Stay in the orthostatic position upon a soft surface
6. Walk upon a soft surface
7. Make baby steps with your eyes open and then closed
8. Repeat exercise 4 upon a soft surface
VESTIBULAR REHABILITATION THERAPY  Cawthorne (1944) and Cooksey (1946)

**Sensory Substitution**

Tonic Rebalancing of Neural Activity

Habituation

---

**Principle**

- **Neural Plasticity**
- **Constant Feedback and recalibration in the brain to environmental stimuli**
VESTIBULAR REHABILITATION THERAPY  Cawthorne (1944) and Cooksey (1946)

Sensory Substitution
Tonic Rebalancing of Neural Activity
Habituation

Technique
• Expose the patient to stimuli that provoke vertigo
• Challenge areas of deficiency
VESTIBULAR REHABILITATION THERAPY  Cawthorne (1944) and Cooksey (1946)

Sensory Substitution
Tonic Rebalancing of Neural Activity
Habituation

Stages of Rehabilitation
• Uncompensated
• Static Compensation
• Dynamic Compensation
VESTIBULAR REHABILITATION THERAPY

Most elderly patients would not achieve full compensation

• MAXIMISE SENSORY INPUT

• MINIMISE HAZARDS
VESTIBULAR REHABILITATION THERAPY

Most elderly patients would not achieve full compensation

• MAXIMISE SENSORY INPUT
  • Good lighting
  • Avoid bifocal lenses

• Flat shoes, bare foot
• Avoid thick carpets, irregular surfaces

• Walking stick
• Hand rails – stairs/ bathroom
VESTIBULAR REHABILITATION THERAPY

Most elderly patients would not achieve full compensation

• MINIMISE HAZARDS
  • Avoid low furniture/ clutter/ wires on floor
  • Avoid rugs
  • Avoid polished floors

• Avoid night darkness
  • Night light
  • water next to bed

• Accessible telephone
Causes of Dizziness in Elderly

A small abnormality say in one vestibular labyrinth may produce symptoms which suddenly increase in severity if vision deteriorates or arthritis develops in the neck.\(^1\)

1. Age and Ageing, 1981;10;105-109
Disease modifying surgery

Superior Semi-circular canal dehiscence

Vascular loop
Vestibular Neurectomy

Gentamycin failure
Perilymphatic Fistula

- Cochleo-vestibular developmental anomalies

Internal Acoustic Meatus

Perilymphatic duct
Perilymph fistula

23, M

Severe giddiness at age of 10 yrs lasting 1 month

Falls / Drop Attacks/ persistent imbalance- 2mth
Perilymph fistula

23, M

Severe giddiness at age of 10 yrs lasting 1 month

Falls / Drop Attacks/ persistent imbalance- 2mth

dizziness increased with rt. ear down
Perilymph fistula

23, M

Severe giddiness at age of 10 yrs lasting 1 month

Falls / Drop Attacks/ persistent imbalance- 2mth

dizziness increased with rt. ear down

FLOOD TEST
Perilymph fistula

23, M

Severe giddiness at age of 10 yrs lasting 1 month

Falls / Drop Attacks/ persistent imbalance- 2mth

dizziness increased with rt. ear down

FLOOD TEST

Conservative Rx- recurrent drop attacks

Surgical Rx  xxxxx- only hearing ear

Rt Tympanotomy- sealing of OW leak  -> cessation of DROP ATTACKS