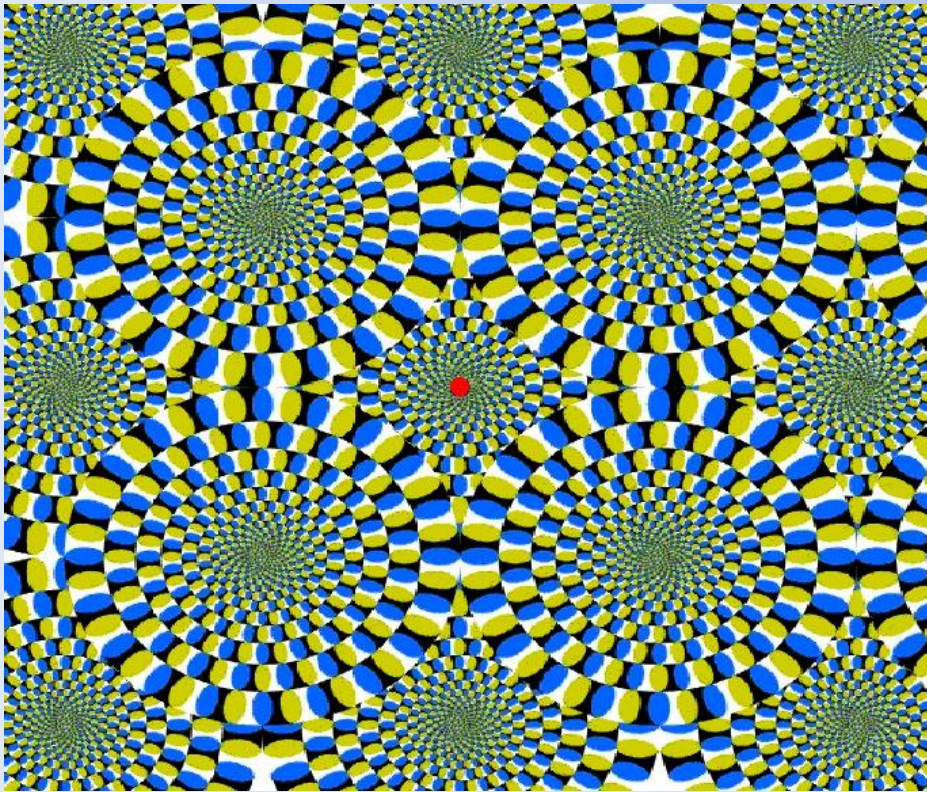


Workshop 3C:

Dizziness in Older Adults



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AGILE Vice-Chair

Dizziness in Older Adults: Aims

1. Describe the problem of dizziness and begin to unpick what it means
2. Describe potential causes of dizziness in older adults
3. Focus on vestibular causes:
 - Review basic anatomy of the vestibular system
 - Briefly discuss BPPV – theory, testing and treatment
 - Describe unilateral vestibular hypofunction
 - Basic vestibular rehabilitation: exercises that may complement PSI, OEP and other exercise programmes

Dizziness



- Common complaint to GPs “I feel a bit dizzy doctor!”
- Increased likelihood with age
- More women affected

Related to:

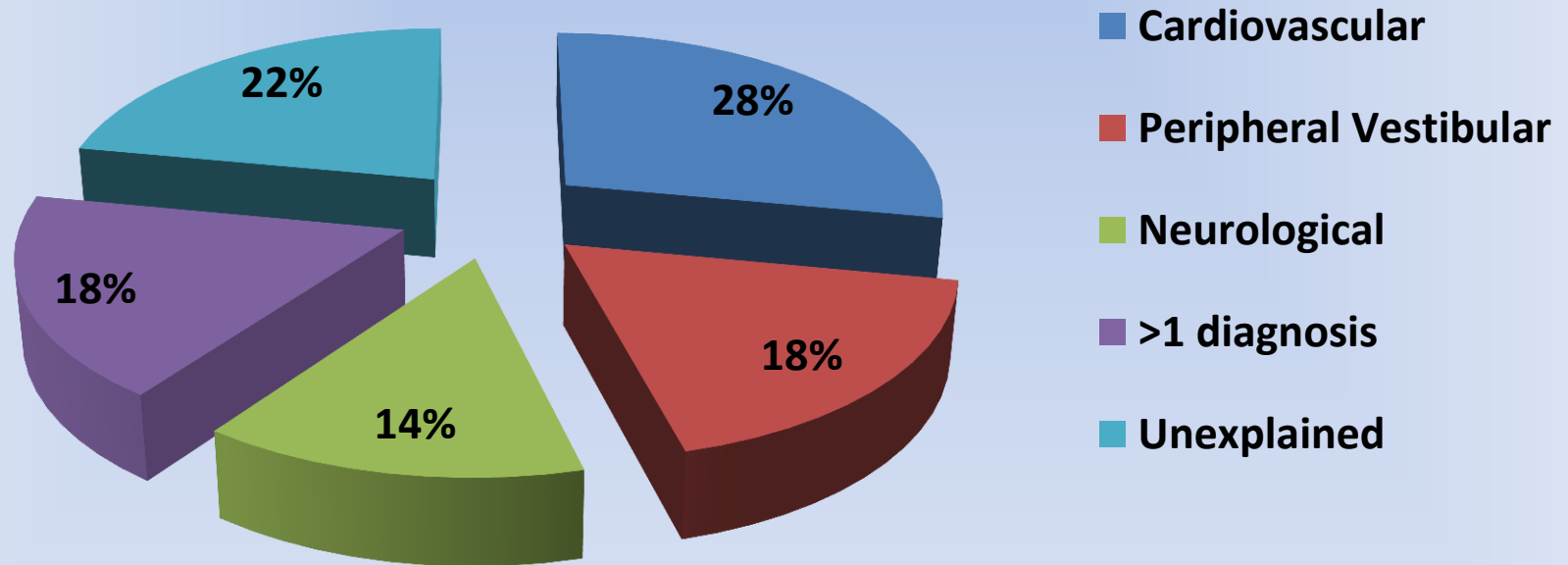
- Reduced ADL's
- Difficulty with ambulation
- Depression
- Falls and injuries

Prevalence of dizziness

- Nearly 20% of community-dwelling adults aged 60 or over reported having 'suffered dizziness significant enough to result in a doctor's visit, taking a medication, or significantly interfering with normal activities within the previous year' (Lawson, 2005)

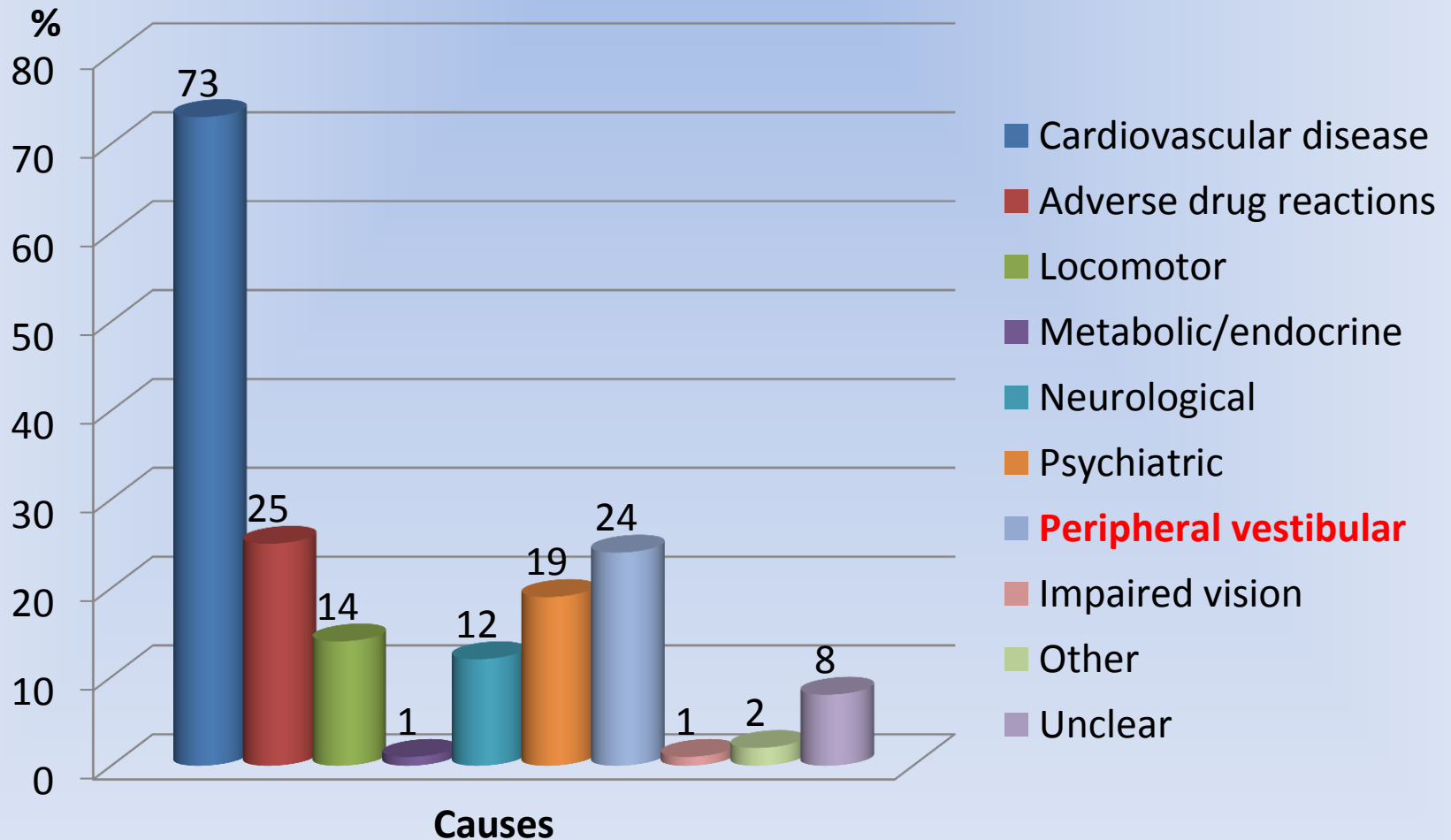
Diagnosis of geriatric patients with severe dizziness in general practice

Lawson *et al* 1999 n=50 people aged >60 yrs



Persistent dizziness in elderly patients

Maarsingh *et al* 2010 Primary Care n=417 aged 65-95 years



Dizziness in older adults is often multi-dimensional

In the Maarsingh study
61% patients had 2 or
more contributing causes
of dizziness



Assessment is

- 70% history
- 20% clinical examination
- 10% laboratory tests

What, where, when, how, duration, latency, symptoms.....

- Different descriptions and taking an accurate history will lead you to the basis for diagnosis, which can then be confirmed by the correct clinical examinations
- ?Orthostatic hypotension
- ? Poor strength and balance
- ? BPPV
- ?Vestibular hypofunction
- ? Central problem
- ? Vascular problem

First things first.....

- What does the person mean by dizziness?

Light headed

ROOM IS SPINNING

giddy

UNSTABLE

Unsteady

WHOOSH!

Blackness

Sick

Drunk

Questions to ask

- Onset – sudden or gradual?
- What are the symptoms?
- What are the precipitating factors?
 - Spontaneous, movement, position, stress, visually complex situations. Do they avoid provoking situations?
- How often does it happen – daily, weekly, clusters etc
- Duration and easing factors
- Associated factors
- Medical history, medications
- Limiting function?
- What does the person think is happening?

Red flag symptoms associated with dizziness

- Associated hearing loss
- Numbness or tingling
- Weakness
- Slurred speech
- Loss of consciousness
- Rigidity or tremors (*although can occur with syncope)
- Diplopia
- Altered heart rate or rhythm, or breathing pattern
- Headaches, memory loss



Dizziness of the cardiovascular system

Syncope is defined as a transient, self-limited loss of consciousness and postural tone (which often leads to falling). The onset is rapid, of short duration, and recovery is spontaneous, complete and prompt. The underlying mechanism is an abrupt cerebral hypoperfusion



Cardiovascular causes

1. **Presyncope:** light-headedness, blurred/black vision, loss of concentration, pale and sweaty, malaise, strong desire to lower body
2. **Vasovagal syncope:** prolonged standing, hot room, presyncope, pale, relieved by lying down
3. **Reflex syncope:** situational e.g.. Bleeding, cough, defaecation, micturition, exercise, pain, emotion (needles!)
4. **Carotid sinus syndrome:** usually have cardiac history, stimulated sensitive carotid sinus results in bradycardia, hypotension and asystolic response

5. Arrhythmias: previous cardiac disease, palpitations, +/- chest pain, no warning, can happen in sitting and lying (unlike vasovagal syncope)

6. Structural cardiac disease

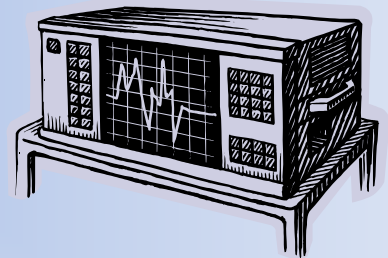
7. Orthostatic hypotension



ECG tracing of a normal heart rhythm.



In atrial fibrillation, the tracing shows tiny, irregular "fibrillation" waves between heartbeats. The rhythm is irregular and erratic.



Neurological disorders

- MS
- Ataxia/ cerebellar disorders
- TIA
- Brain tumours
- Migraine
- Labyrinthine infarct
- Trauma
- Intoxication

Medications:

Many drugs can cause dizziness. Common culprits include:

- Anti-convulsants
- Anti-depressants
- Anti-anxiety drugs
- Sedatives, including hypnotics
- Strong analgesics
- Muscle relaxants
- NSAIDs
- Anti-arrhythmias
- (anaemia)

Gait/ locomotor

- Dizziness vs unsteadiness
- Peripheral neuropathy
- Msk problems
- Visual impairment
- Cautious gait
- Poor postural control

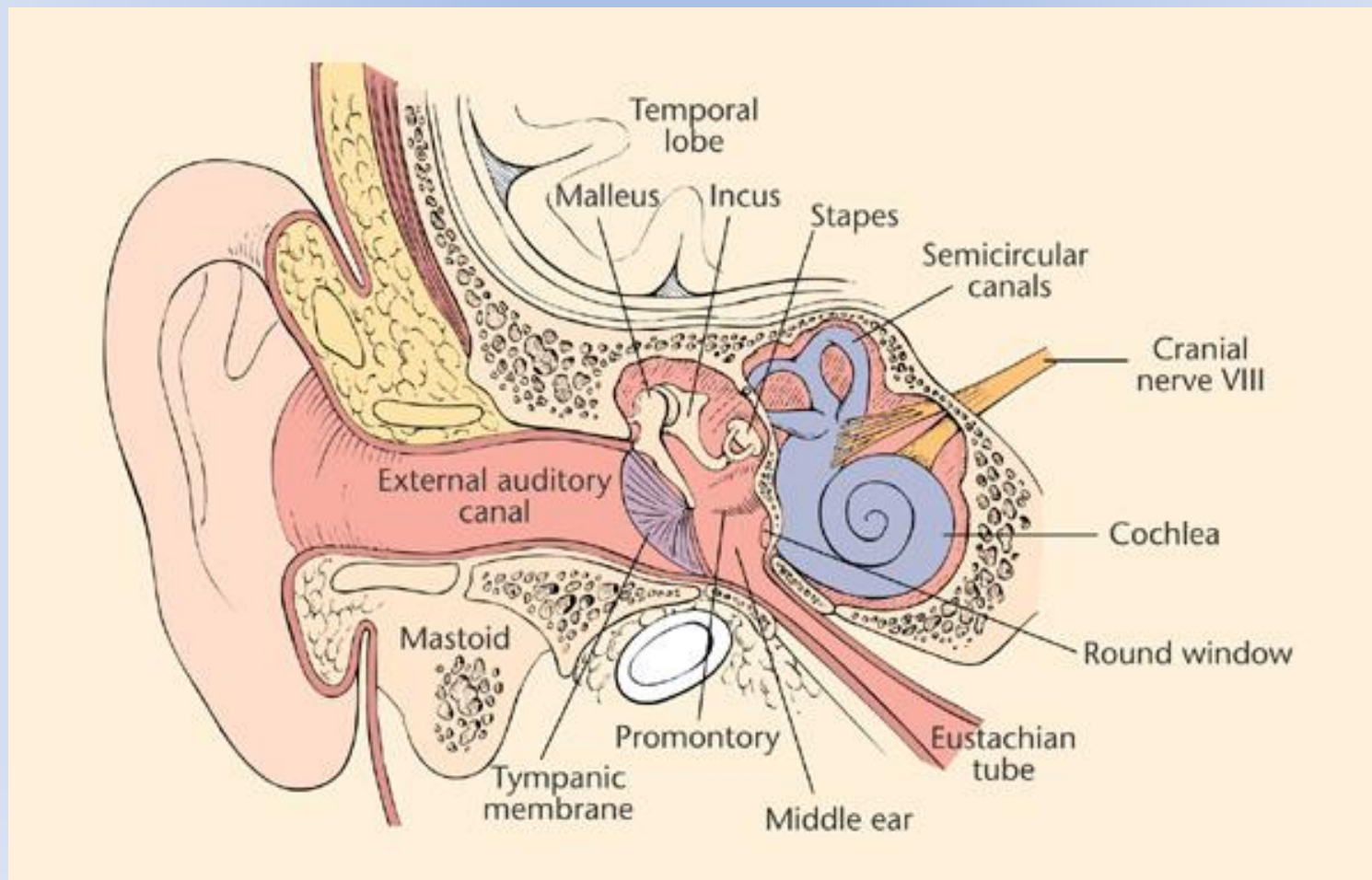


Vestibular disorders

- Common in older people, and in older people who fall (80% of people attending A&E with unexplained falls had symptoms suggestive of vestibular problems - Pothula, 2004)
- BPPV
- Meniere's Disease
- Vestibular neuritis
- Viral labyrinthitis
- Bacterial labyrinthitis
- Acoustic neuroma
- Perilymph fistula

Anatomy of the Vestibular System

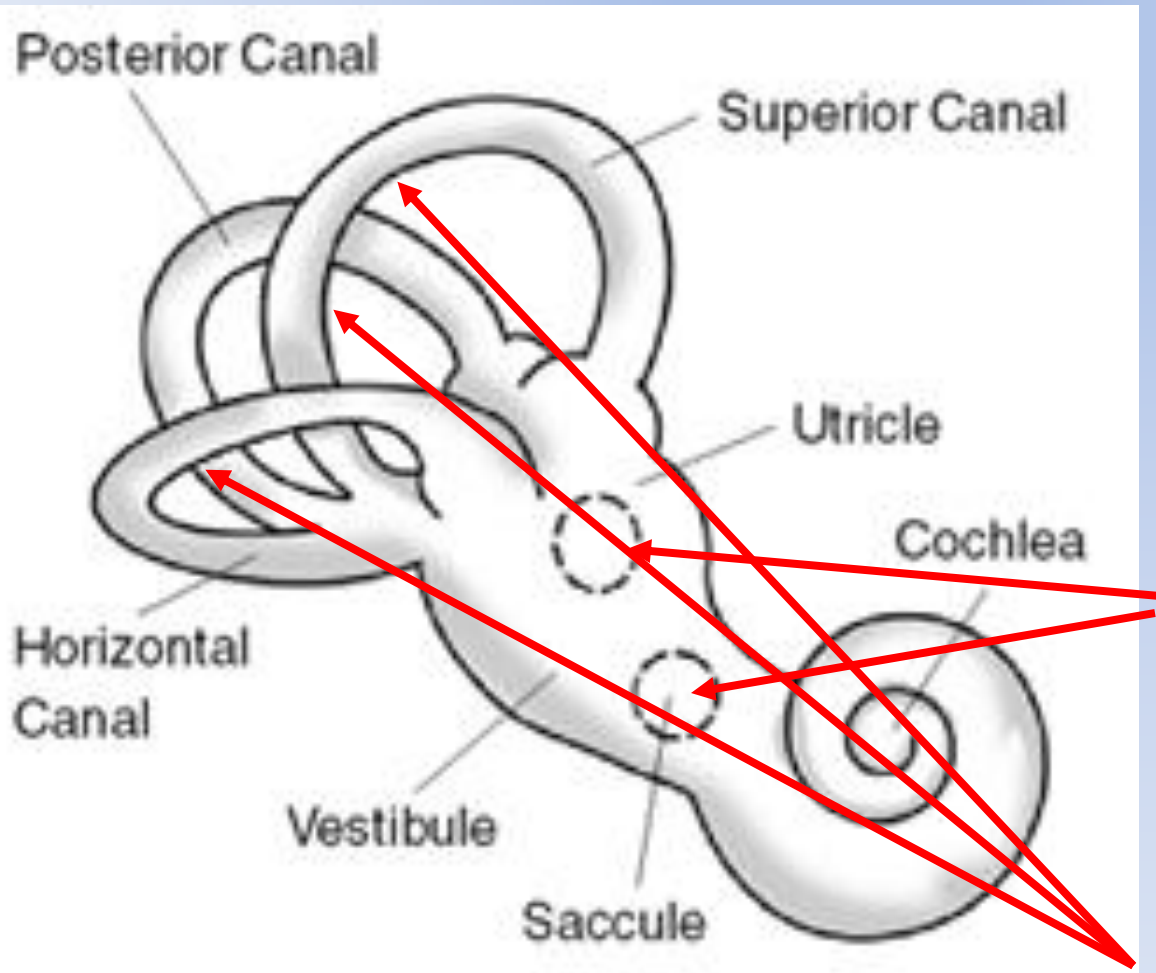
Normal Ear anatomy



Vestibular system anatomy

- VS is responsible for sensing motion of the head
- Maintains stability of images on the fovea of the retina and postural control during that motion
- Accurate representation of the head in 3 dimensions
- This info is then used by the central vestib pathways to control reflexes and perceptions mediated by the VS
- Detects head motion – velocity, acceleration and frequency
- VOR short latency

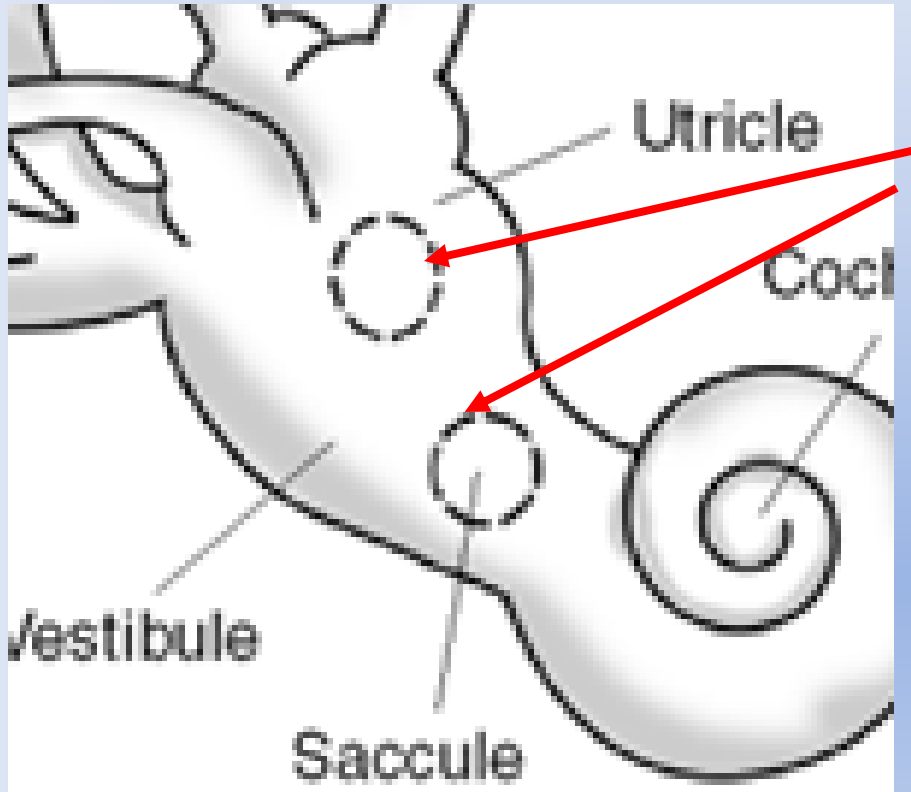
The Vestibular System



- Within bone
- Membranous labyrinths
- Filled with endolymph
- Right and Left
- 5 receptor organs

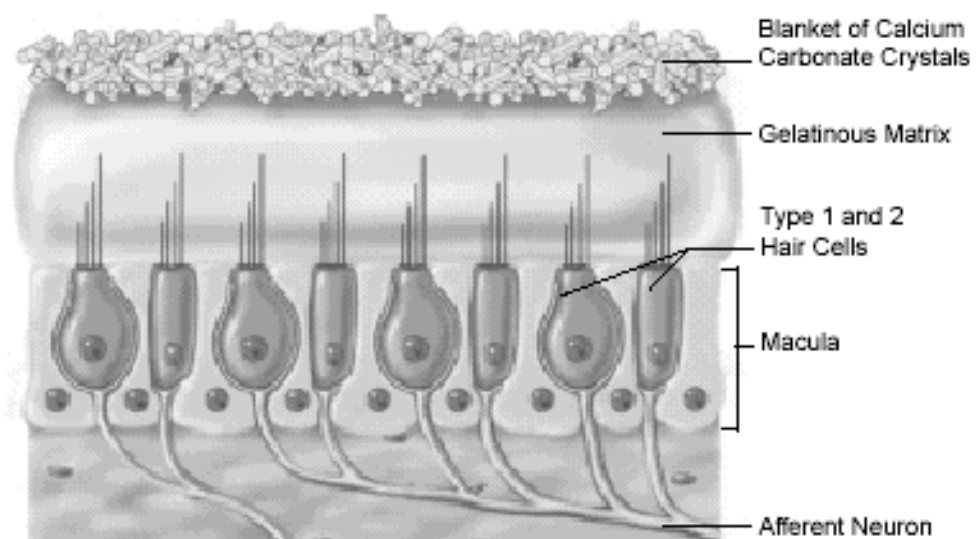
Utricle and Saccule
(otoliths)- linear
acceleration

Semicircular canals-
angular acceleration

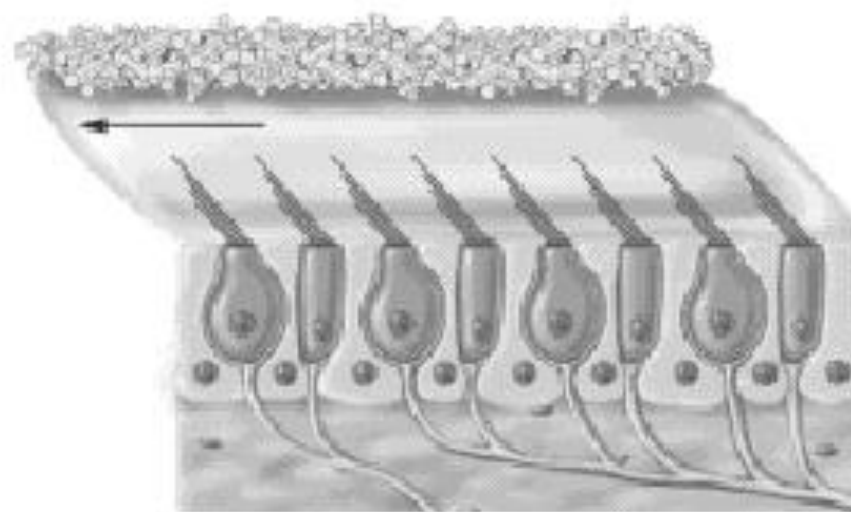


Otoliths

- Utricle = horizontal
- Saccule = Vertical
- Otoconia (calcium carbonate) moves separately to the head to detect linear acceleration.
- Displaces hair cells so that signal can be produced for any physiological plane of movement



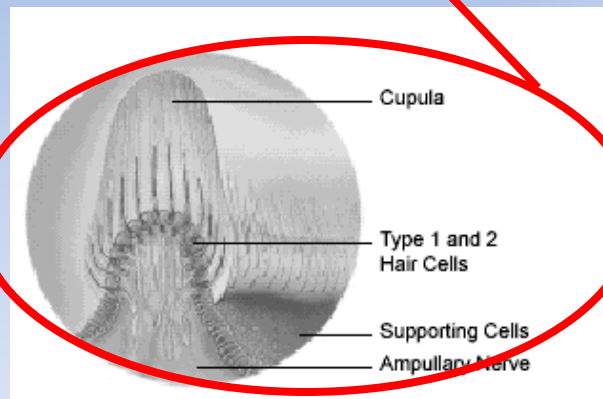
At Rest

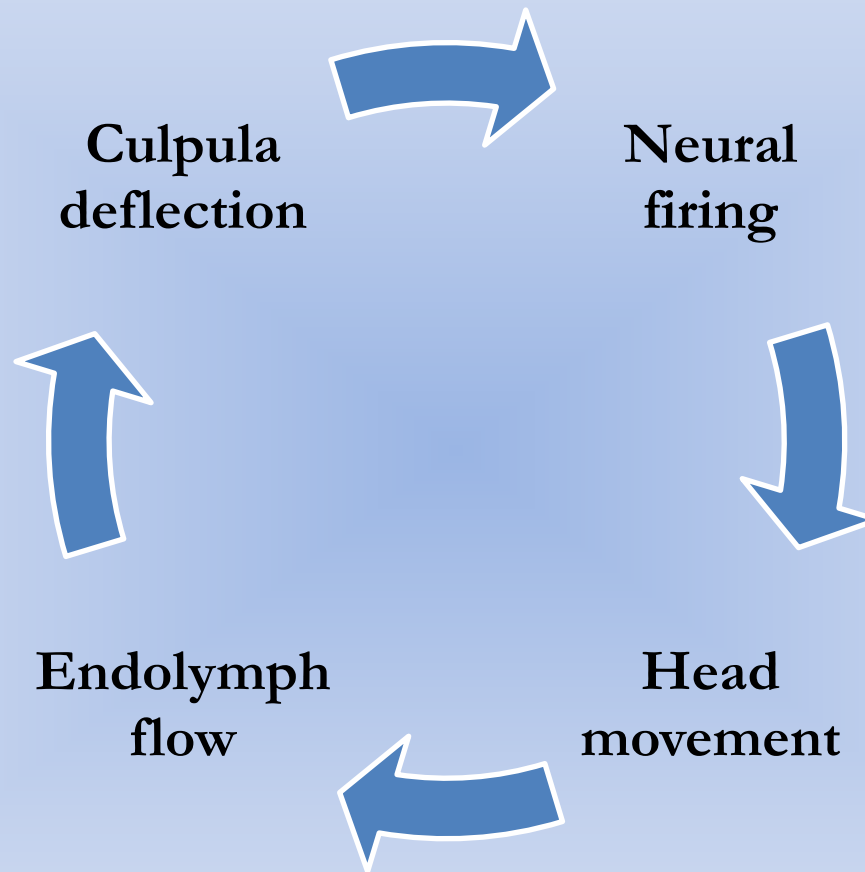


With Otolithic Displacement

Semicircular Canals

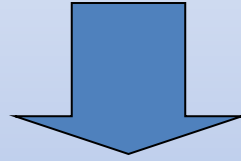
- Detect angular acceleration
- Posterior, anterior and horizontal



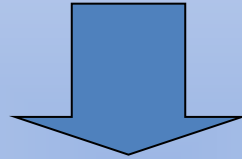


- The 6 SCC's become 3 coplanar pairs
- 1. RIGHT & LEFT Horizontal
- 2. LEFT Anterior & RIGHT Posterior
- 3. LEFT Posterior & RIGHT Anterior
- When head movement occurs, the endolymph of each coplanar pair is displaced in the opposite direction with respect to the ampulla => INCREASED firing of one vestibular nerve and DECREASED in the other
- They work as matched pairs in a “push-pull” situation

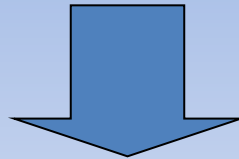
Turn head to RIGHT



Excitation of RIGHT SCC



Decreased excitation of LEFT SCC



**Firing of RIGHT Vestibular Nerve & inhibition of
LEFT**

Central Processing of Vestibular Input

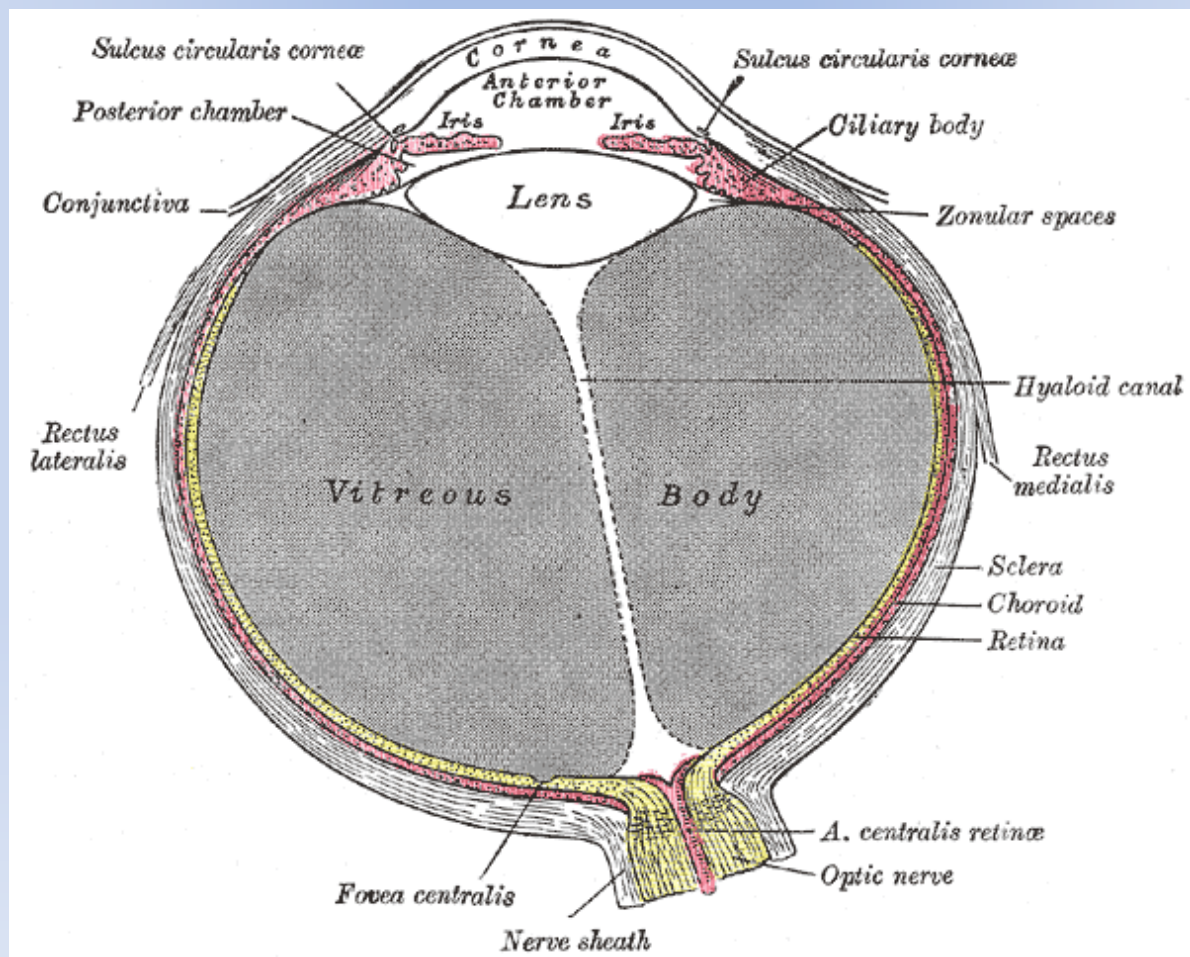
- Primary afferents synapse in the
- 1. Vestibulonuclear complex
 - Pons & medulla
 - Primary processor of vestibular input
 - Fast connections between incoming afferent info and motor output neurons
- 2. Cerebellum
 - Adaptive processor – monitors vestibular information
- At both locations input is processed with somatosensory and visuosensory input

Motor Output of Vestibular System

Neurons: 1. Vestibular Ocular Reflex

- VOR = the ability of the eyes to fix on an object whilst head turning by stimulating nerve input to the extra ocular muscles of the eye
- Stimulation of the SCC's => reflex eye movt in the opposite direction to the perceived head movt
- Main function = to control eye position during transient head movts to maintain a stable visual image
- Moving head to the right causes eye movement to the left
- This co-ordination of eye movement with head movement is necessary for clear vision and normal balance

- Fovea is the point where visual images become perfectly clear.
- Brain uses information from the fovea to clear images, and fill in the blind spot next to the fovea
- Saccadic eye movement = gaze shift to fast movement
- Smooth pursuit = tracking



Motor Output: 2. Vestibulospinal Reflex

- Output neurons are found in the AHC of spinal cord grey matter which drive skeletal muscle
- Complex connection
- Strategies include lateral vestibulospinal tract => antigravity muscle; medial vestibulospinal tract => cervical muscles; reticulospinal tract => activates trunk and proximal limb muscles

Nystagmus

- “Involuntary rhythmic oscillation of the eyes”, usually having a clearly defined slow and fast component.
- The vestibular system drives the slow component (VOR)
- The direction of the fast component defines the direction of the nystagmus
- VERTIGO = dizziness with nystagmus, “illusion of movement”

Types of Nystagmus

Physiological

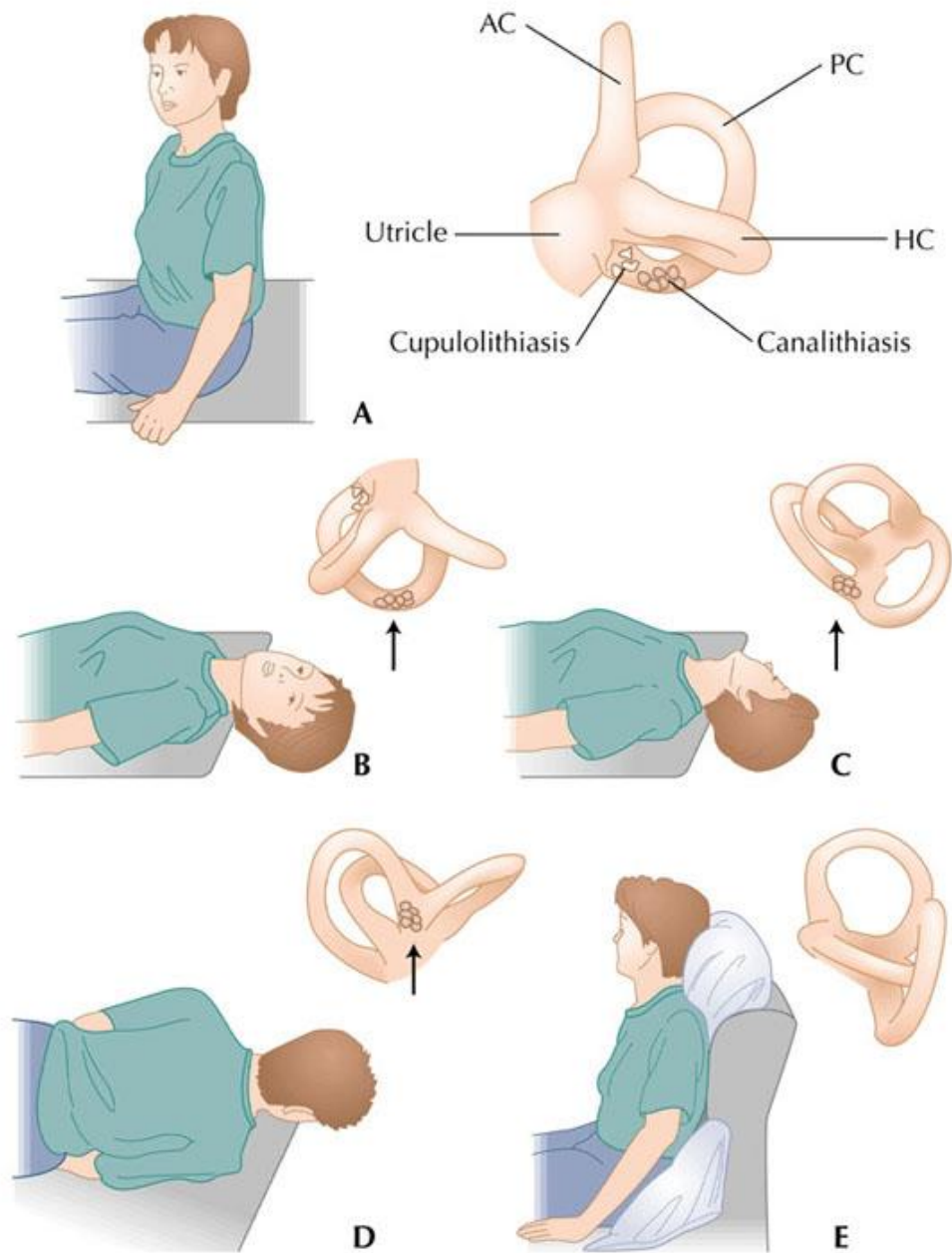
- Rotational induced
- Caloric induced
- Optokinetic
- End-point

Pathological

- Spontaneous
- Gaze-evoked/holding
- Positional
- Congenital

BPPV

- Benign Paroxysmal Positional Vertigo
- Most common peripheral cause of vertigo
- Posterior canal most frequently affected
- Females > males
- Commonly presents between 4th and 7th decade, increased incidence with age
- Condition often missed in the elderly
- 20% cases occur post trauma e.g. whiplash, HI, fall



Subjective Findings in BPPV

- Vertigo looking up or bending down
- Vertigo when turning in bed
- Vertigo when lying down
- +/- loss of balance
- +/- nausea
- Feeling of support surface tilting
- Vague sensations e.g. light headed, floating

Objective Findings in BPPV (Nystagmus + Vertigo)

- Nystagmus **CONCURRENTLY** with patient in the provoking position (Dix-Hallpike)
- Occurs in the position rather than during the movement (due to latency of onset of symptoms)
- Latency of nystagmus – 1 or more seconds
- Crescendo then decrescendo of nystagmus
- Nystagmus lasts ½ - 1 minute
- Reversal of nystagmus on sitting up
- Reduction in symptoms on repeat testing
- +/- positive balance test

Unilateral Vestibular Loss (UVL)

- Under-functioning vestibular sensory organ
- Includes conditions such as BPPV, Meniere's disease, post surgery (e.g. AN), labyrinthitis, vestibular hyponeuritis
- Symptoms include dizziness/vertigo, balance deficits and visual gaze disturbance

UVL is characterised by

- 1. Balance defecits:** Feeling unsteady, falls, difficulty walking in a straight line, and tendancy to veer in one direction, difficulty turning
- 2. Visual/ gaze disturbance:** Blurred vision specifically associated with movement (e.g. when walking), may be very subtle.

Vestibular compensation and pathophysiology

- Fortunately much disease process of the vestibular labyrinth is self limited and spontaneous recovery is expected
- This process is **VESTIBULAR COMPENSATION**
- “It will get better over the next few weeks”



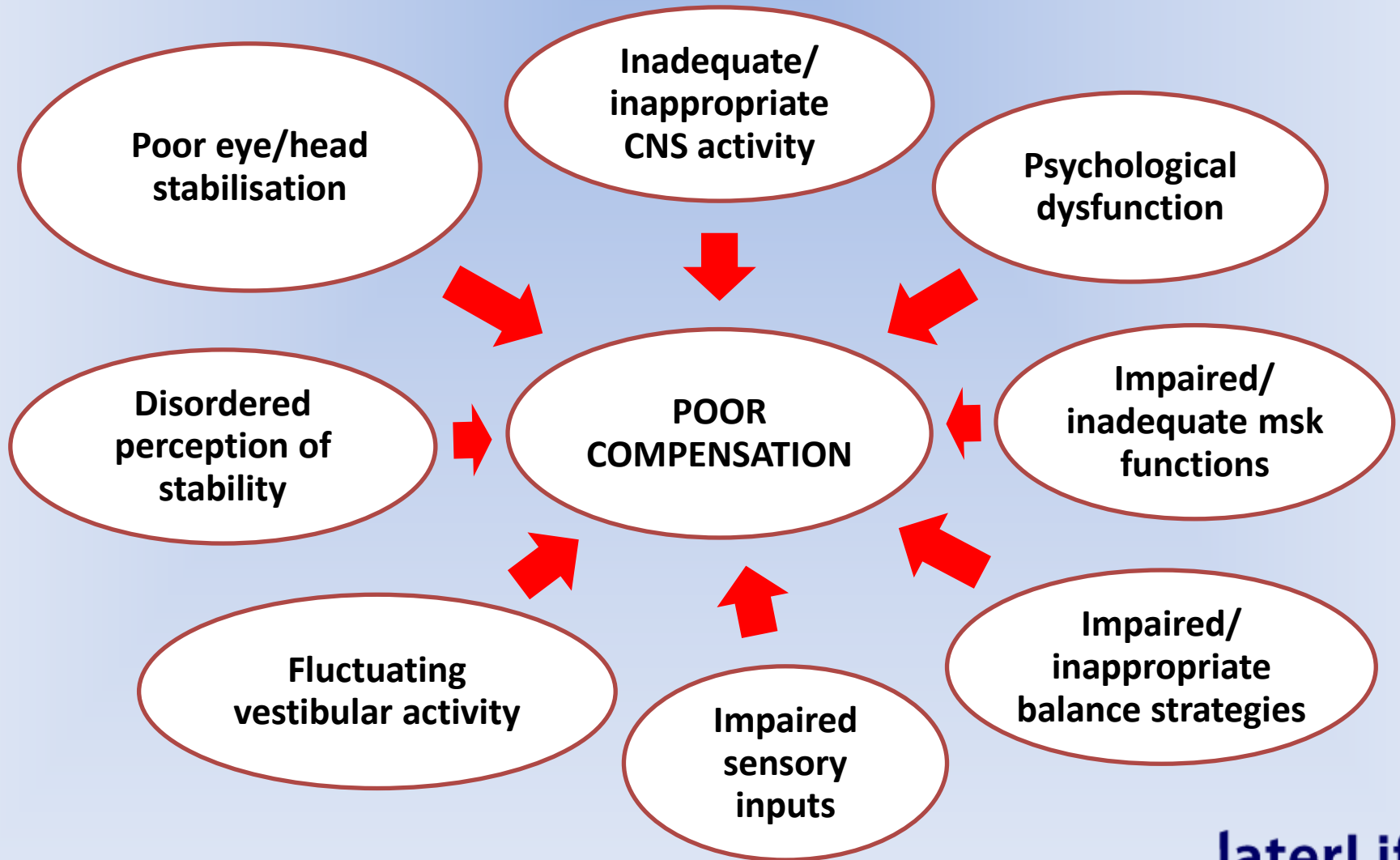
Recovery

- Static components recover spontaneously 3-14 days after lesion
- In the cerebellum, firing of vestibular nuclei neurons become rebalanced. There is recovery in firing rate on the lesion side and dampening down on the normal side.
- This occurs independent of vision

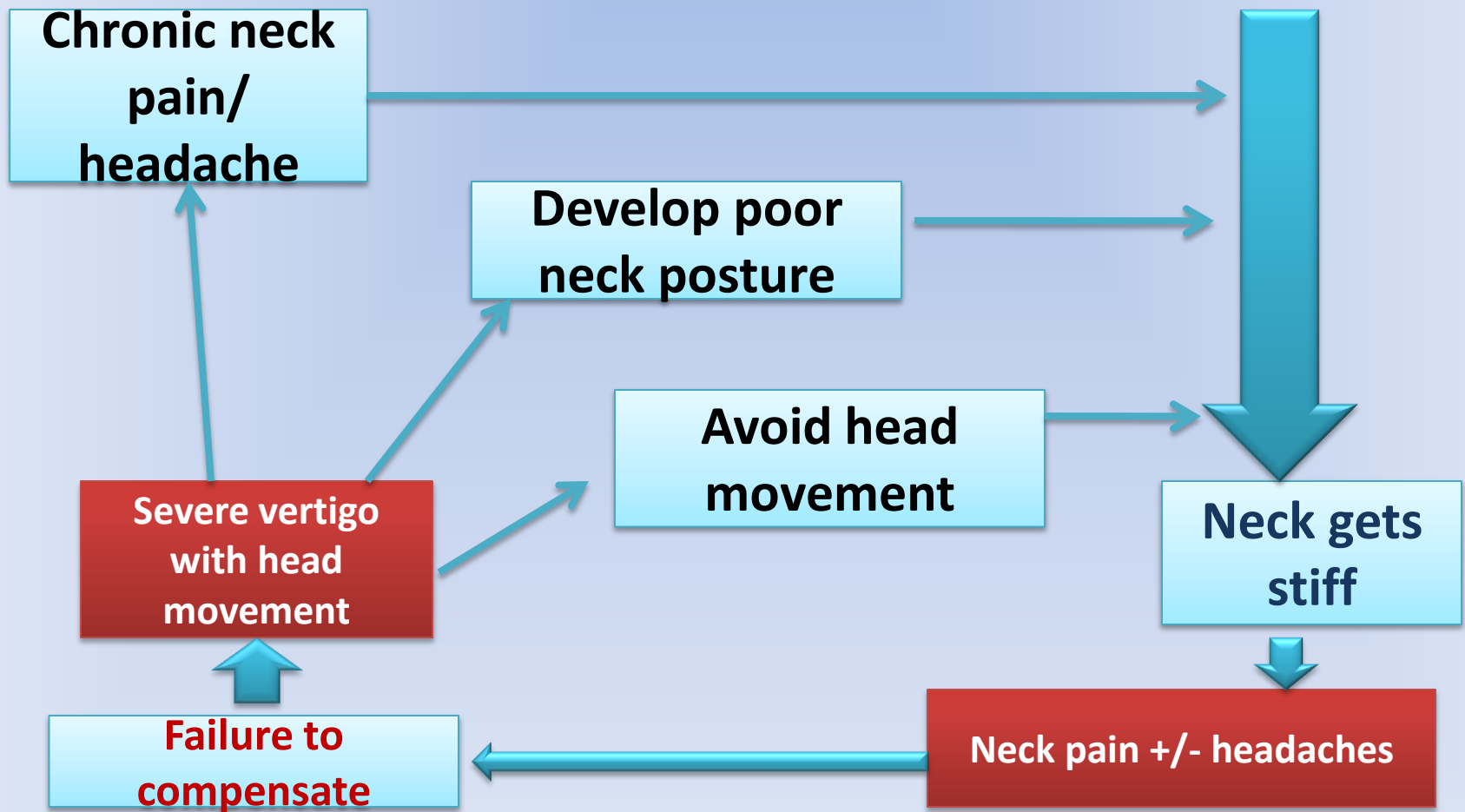
BUT.....

- Dynamic vestibular function recovery is more variable and subtle problems may persist e.g. image motion or retinal slip
- Adaption or recalibration of the VOR is one of the most important recovery mechanisms
- This is when VR has a role to play
- The VOR is the only reflex in the body which can be changed or adapted with exercise

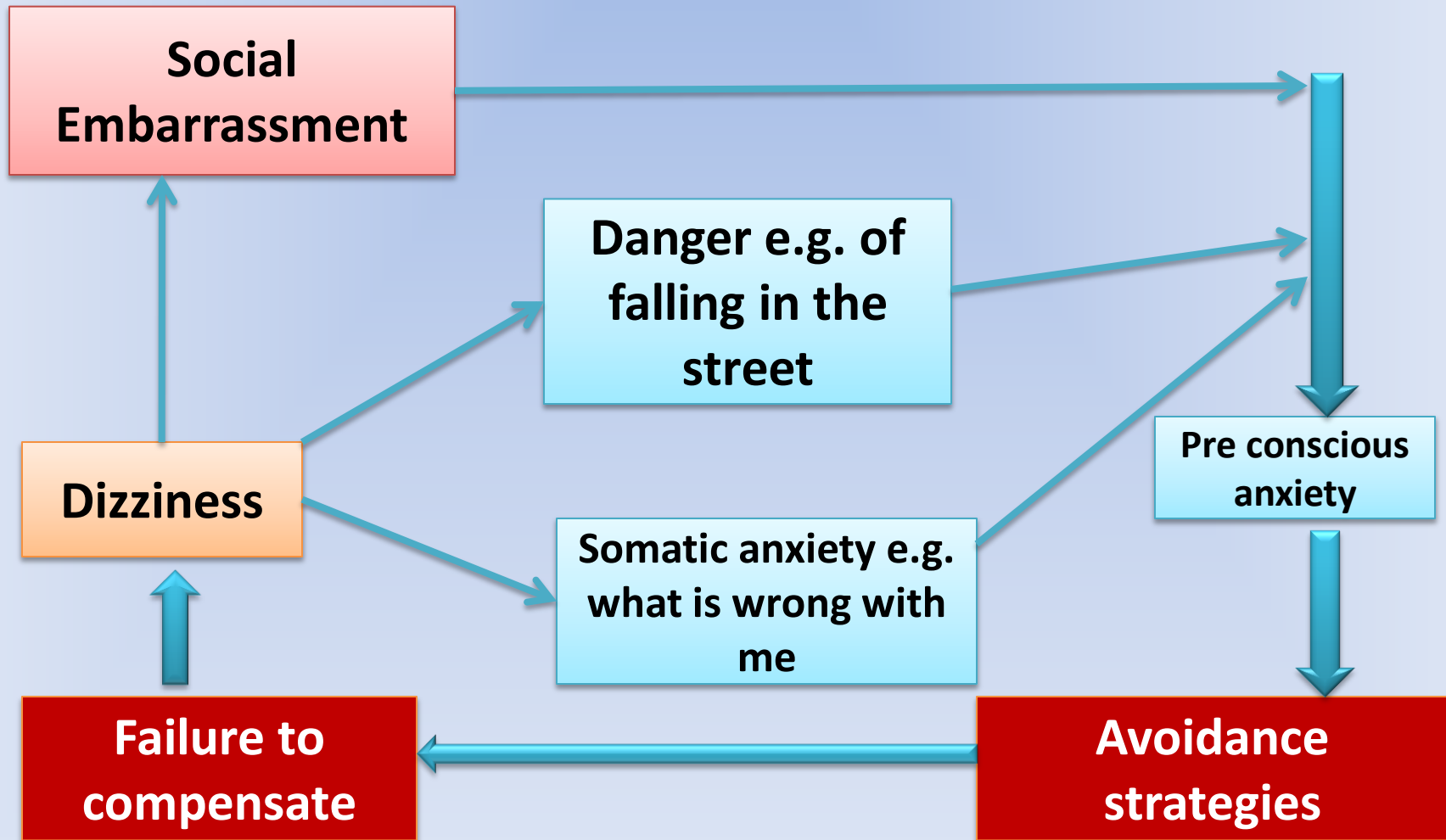
Reasons for poor vestibular compensation:



The dizziness vicious cycle 1



The dizziness vicious cycle 2:



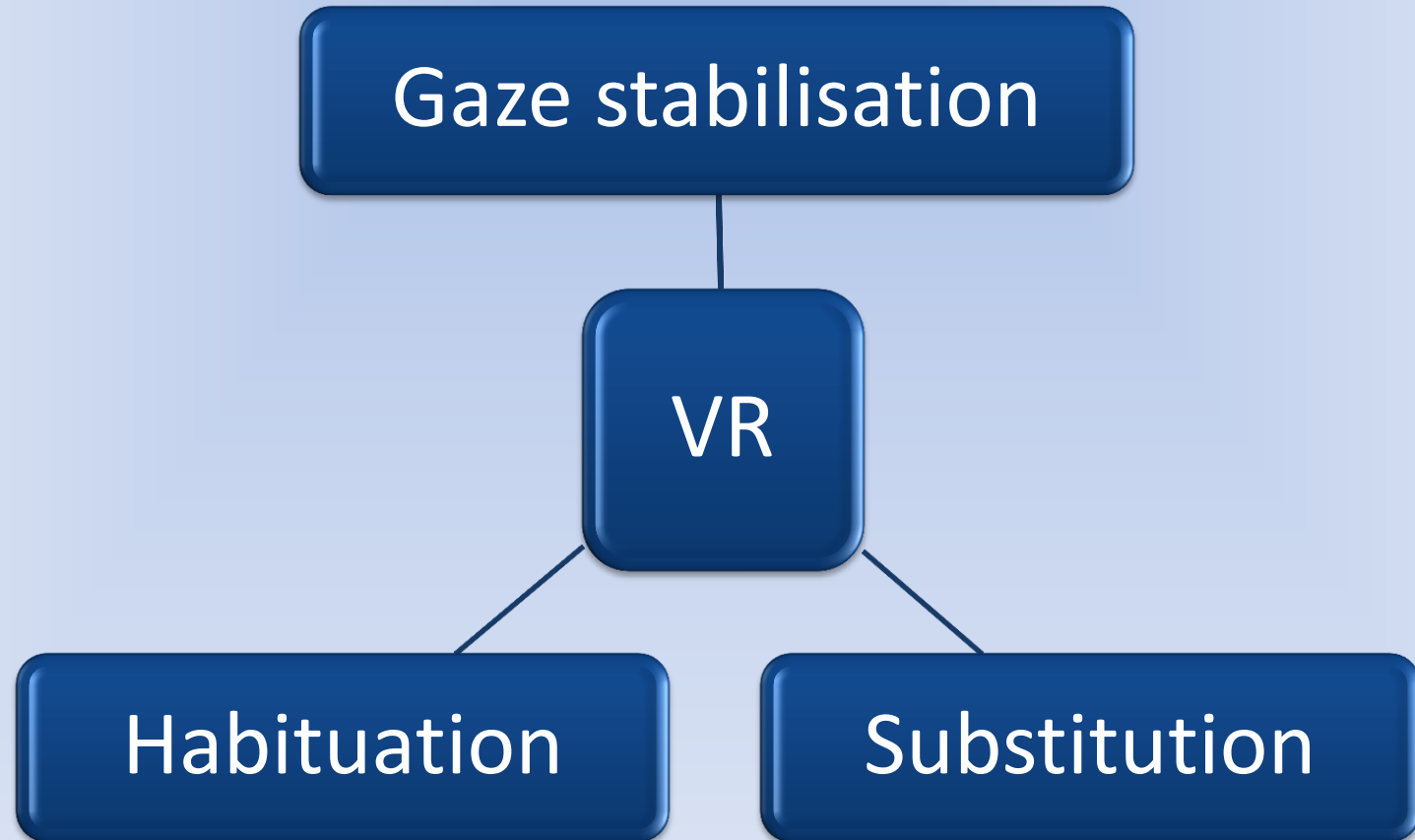
Rationale of Customized Exercise Programmes

- Identify and concentrate on specific tasks
- Work at limit of patients ability

Goals of Treatment

- Improve static or dynamic balance
- Improve ability to see clearly with head movement
- Decrease sense of disequilibrium
- Improve overall activity level and physical condition
- Decrease social isolation

3 main components to VR



Treatment Progression

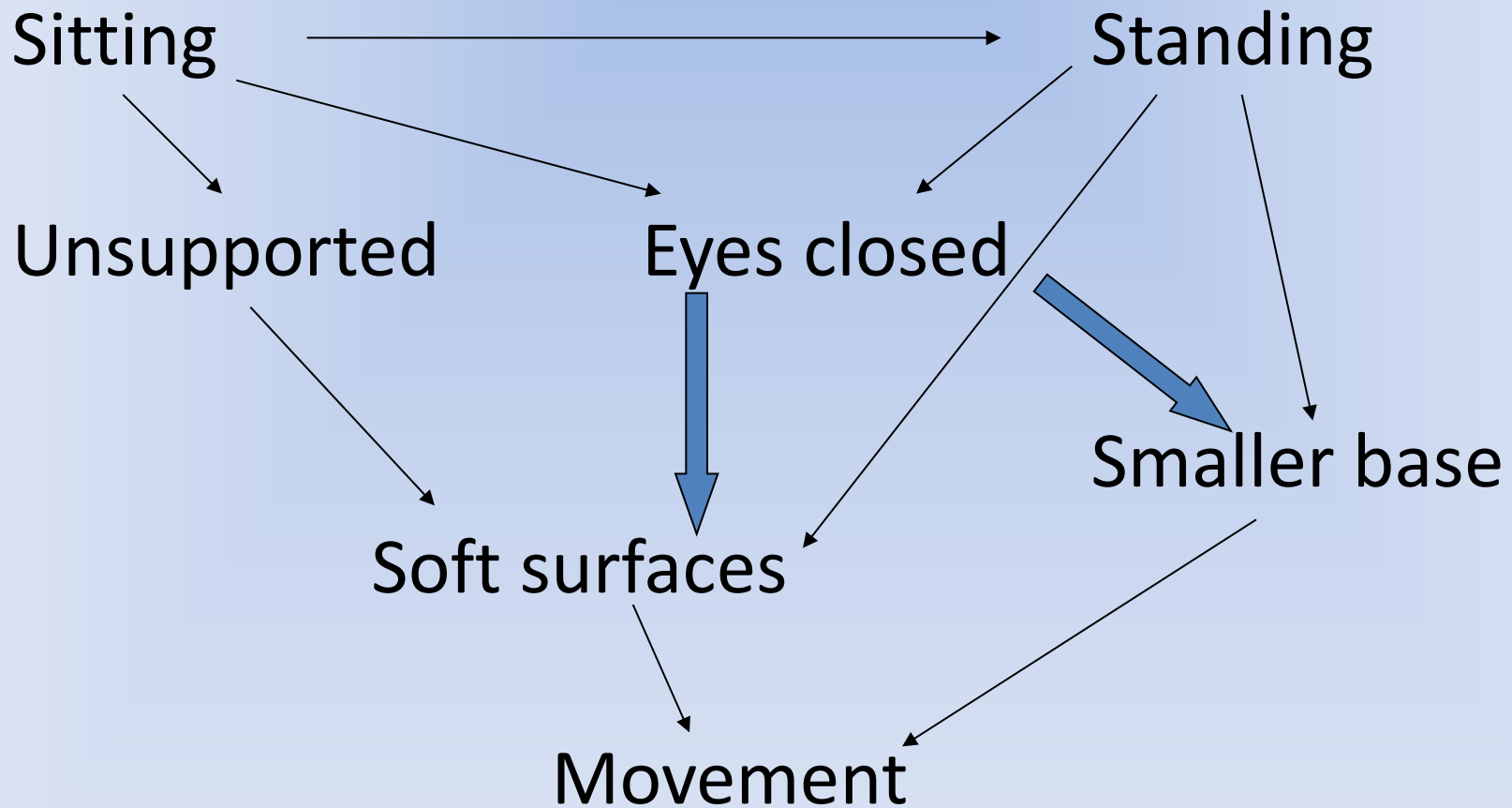
Stable  Unstable

Simple  Complex

Slow  Faster

Non-functional  Functional

Stability Examples



Simple	Complex
Basic VOR gaze stabilisation	VOR with ■ Patterns ■ Mirrors ■ Movements
Marching on the spot	Marching with ■ Eyes closed ■ Head movement ■ Movement around them
Walking	Walking with ■ Combination of above ■ Other movements ■ Specific context or environments

Other Exercise Variables

- Speed, distance, environment, backgrounds
- Specific work movements
 - Standing and twisting at conveyor belt
 - Using a computer
- Specific Activity
 - Hanging washing on a line
 - Driving



Specific sports movements

- Bending for indoor bowls
 - Line dancing
- Find the component the patient can't do and practice



Yardley et al 2004

- Single blind RCT (based in UK)
- VR compared to usual care
- Reassessment at 3/12
- After reassessment usual care group receive VR
- Reassessment at 6/12
- Measured; Vertigo Symptom Scale, Dizziness Handicap Inventory, Balance Performance Monitor, Provoked symptoms on movements.

Yardley et al 2004 - Results

- 170 randomised, mean age 62, ♀ 60%.
- Significant improvement in all outcome measures at 3 months.
- At 3 months 23% VR: no provoked symptoms compared to 6% usual care
- At 6 months VR maintained improvements and control (had 3/12 VR) had improved to the same level
- 71% carried out the exercises most days of the week.

Intervention

- Nurse training
- Patient information
- Schedule for sessions
- One 45 minute face to face session
- Telephone follow-up 1 and 3 weeks after this.
- Encourage presence of family member or friend who can provide support

Implementation

- Go through exercise with patient helping to fill out chart and copying current exercises

6 Exercises:

1. Shake head
2. Nod head
3. Shake head with eyes closed
4. Nod head with eyes closed
5. Shake head and stare at finger
6. Nod head and stare at finger

Therapy Monitoring Sheet

Telephone _____

3 = strong symptoms

1-wk Follow-up

3 conditions:

1. Sitting
2. Standing
3. Walking

3-wk Follow-up

Shake (eyes closed)

Nod (eyes closed)

Shake/stare

Nod/stare

Special
exercises

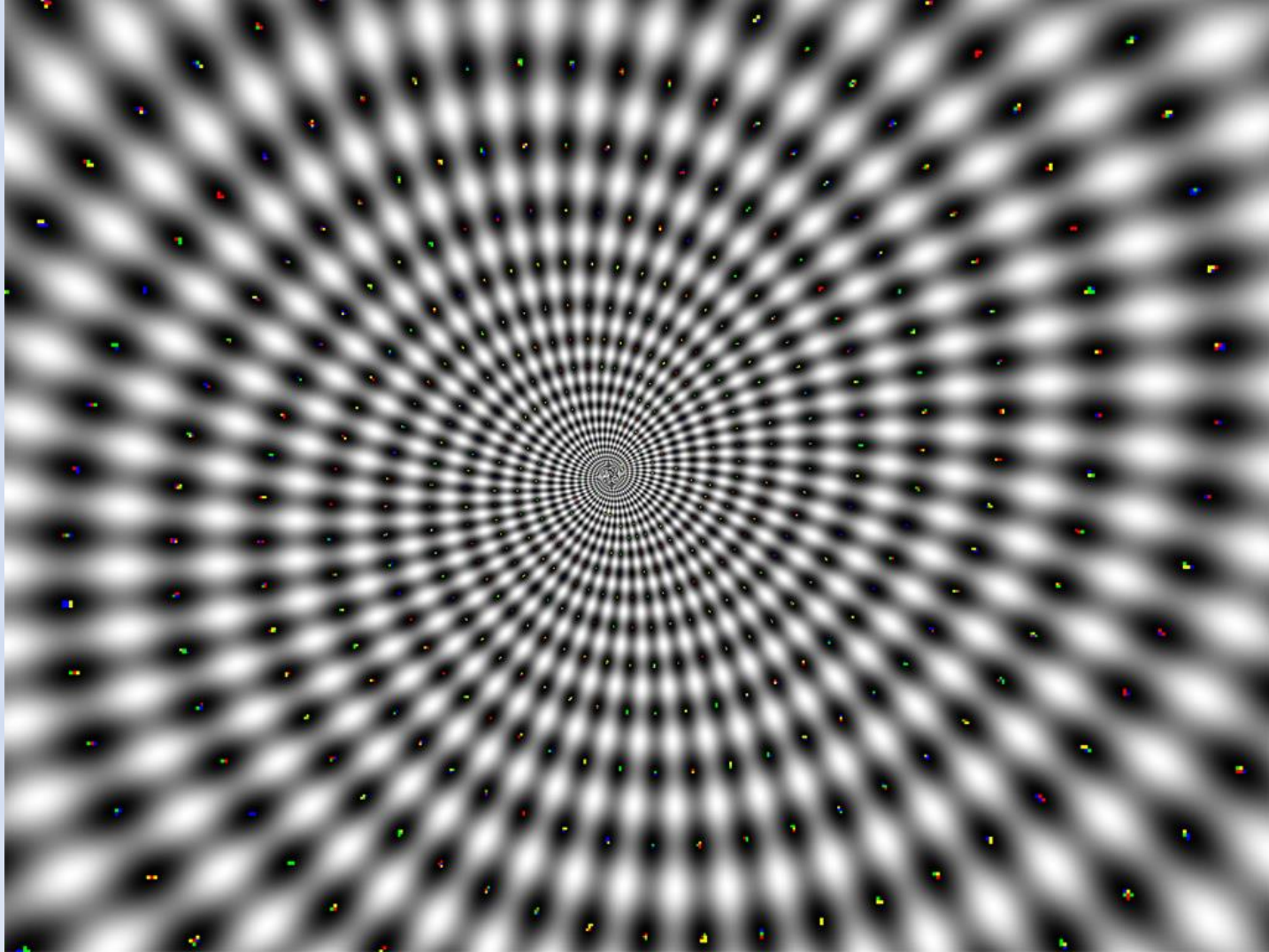
General
activities

General comments: (including any particular patient concerns, goals, or problems, nonadherence, setbacks)

Rate symptoms:

0. No symptoms
1. Very slight symptoms
2. Mild symptoms
3. Strong symptoms

Have a go!



Yardley (2012)

- N=276
- Usual care vs booklet vs booklet and telephone support
- “Booklet based vestibular rehabilitation for chronic dizziness is a simple and cost effective means of improving patient reported outcomes in primary care”.
- A copy of the booklet used in this study (**Balance Retraining and Controlling Your Symptoms by Professor L Yardley**) may be obtained from the Meniere's Society; email info@menieres.org.uk or call them on 0845 120 2975

Case studies

1. Relatively active 65 year old female. 1 episode of acute viral labyrinthitis 18 months ago now resolved with some residual symptoms. On assessment dizzy on sit to stand, bending and reaching, hence 2 falls in the kitchen. Higher level balance deficit. Nauseous on head turning. She is attending your PSI group – what exercises may help and what homework would you give? Progressions?
2. Frail 94 year old female. Several falls outdoors and has now stopped going out. Acute episode of vertigo, diagnosed with BPPV by GP and treated with Epley PRM. Frightened by vertigo episode as happened during the night and she felt as though the bed was tipping her out. Some residual dizziness and feels unsteady. She has started using a walking frame and balance is poor. Design an exercise programme – what exercises could she do on her own and what could she do in your group?
3. 85 year old man. Fell at home and banged his head 4 months ago. Not going out now as feels unsteady when trying to cross the road and doesn't like walking past brick walls. He often veers to the right. Has stopped doing his ironing as it makes him dizzy. OK when rolling in bed. Right ear feels a bit “blocked” and has some hearing loss. Design a rehab programme to complement his Otago (home based) exercises. You have access to a support worker.

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