Doctor! Why do I feel dizzy all the time?

รศ. นพ. จันทร์ชัย เจรียงประเสริฐ

ภาควิชาโสต ศอ นาสิกวิทยา

คณะแพทยศาสตร์ โรงพยาบาลรามาธิบดี

Chronic dizziness: a practical approach

- Chronic dizziness: 0.3% of adult population
- About 10% of new patients in general neurology clinic
- Description of *current* symptom
- *Evolution* of symptoms , how they started and how they have changed?
- What is the new *Criteria of diagnosis*?
- How many *PE & laboratory testing* need?
- How many *treatment modalities* exist?

Pract Neurol, 2010

Persistent postural-perceptual dizziness (PPPD)

- Diagnostic criteria for PPPD: Consensus document of the committee for the Classification of Vestibular Disorders of the B´ar´any Society
- International Classification of Vestibular Disorders - ICVD

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Functional dizziness: from PPV and CSD to PPPD

- The ICVD differentiates disorders: by
  - Duration of symptoms (acute, episodic, and chronic syndromes)
  - Proven or presumed pathophysiologic mechanisms (structural, functional, and psychiatric conditions)
- Functional disorders: conditions or disorders 'arising from changes in the mode of action (functioning) of (a) organ system(s)', unrelated to structural or cellular deficits
- Functional & psychiatric cause of vestibular disorders:
  - Common & can be identified by readily recognizable patterns of symptoms
  - Now considered separately from one another & from structural vestibular diseases, occur independently

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Functional dizziness: from PPV and CSD to PPPD

New functional causes of vestibular symptoms:

- Termed 'persistent postural perceptual dizziness' (PPPD) based on 3 decades of research on phobic postural vertigo (PPV), space-motion discomfort (SMD), visual vertigo (VV), and chronic subjective dizziness (CSD)
- Prevalence as primary causes of vestibular symptoms: estimated at 8–10% of all pts in specialty neuro-otology centers
- Coexisting anxiety & depressive disorders: common in functional dizziness, but PPV & CSD can occur w/o psychiatric comorbidity

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Functional dizziness: from PPV and CSD to PPPD

- Nearly 50% of all pts active psychiatric disorder
- Highest rates: VM (49-65%), MD (57%), Vestibular paroxysmia (51%)
- Lower rates: VN (22-37%), bilateral vestibular failure (24%), BPPV (15%)
- CSD (N=107): clinically significant anxiety symptoms 60%, depression symptoms 45%, no psychiatric morbidity 25%
- Prevalence of psychiatric morbidity in general pop about 20%

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In 1870 s, 3 German physicians

Described

- Syndromes of dizziness and discomfort in motion rich environments, accompanied by autonomic arousal, anxiety, and avoidance of provocative circumstances
- Benedikt: neuro-ophthalmologic process in *Platzschwindel* (vertigo in a plaza or square)
- Cordes: a psychological genesis in *Platzangst* (fear in a plaza or square)
- Westphal: postural control, locomotion, conscious appraisal of spatial orientation, and threat assessment as “part of one process” in *Die Agoraphobie* (fear of the marketplace)

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In early 20th century

- As otology, neurology, and psychiatry matured into separate specialties:
- *Platzschwindel* and *Platzangst* faded from use
- *Agoraphobia* became a psychiatric disorder, losing its space and motion context

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A century later

- Small case series published describing various syndromes of spatial disorientation and aberrant motion sensations, including supermarket syndrome, space phobia, motorist's vestibular disorientation syndrome, visually induced motion symptoms, and physiologic height vertigo

In 1980s

- In 1986, Brandt and Dieterich defined *Phobischer Attacken-Schwankschwindel (phobic postural vertigo, PPV)* as a diagnosable clinical syndrome of postural dizziness & fluctuating unsteadiness accompanied by mild anxiety and depression in obsessive compulsive personality traits (OCP)
- In mid-1980 s, Jacob et al potential links b/w anxiety symptoms, persistent dizziness, and vestibular dysfunction in pts anxiety disorders. In 1989, described & subsequently validated symptom of *space-motion discomfort (SMD)* as a combination of uneasiness about spatial orientation & increased awareness of motion stimuli

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Late 20th – early 21st century

- In 1995, Bronstein described *visual vertigo (VV)* in pts following acute PVD or CVD, manifested w sensations of unsteadiness or dizziness on exposure to complex or moving visual stimuli, often persisted despite recovery from acute vestibular deficits
- Visual cues triggered VV overlapped w environmental stimuli that activated SMD
- In 2004, Staab et al described *chronic subjective dizziness (CSD)* based on observations, defined more explicitly in 2007; dx similar to PPV, focused primarily on physical not psychological symptoms

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Structural, functional, and *psychiatric* conditions

- PPPD – last for months to years making chronic vestibular disorder
- Studies of *PPV, VV, SMD, & CSD* identified a number of functional alterations in vestibular & balance mechanisms associated w clinical entities
- Additional investigations separated them from 1ry psychiatric disorders, may co-exist

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CSD – A

3 groups:

(1)Otogenic CSD: 1ry neuro-otologic conditions *triggering 2*ry anxiety

(2)Psychogenic CSD: anxiety alone *causing* dizziness

(3)Interactive CSD: neuro-otologic conditions *exacerbating* preexisting anxiety

Arch Otolaryngol HNS, 2005

Table 1
Features of PPV, SMD, VV, and CSD that informed the definition of PPPD

	PPV [13]	SMD [39]	VV [15]	CSD [79, 81]
Primary Symptoms (criteria A.1–3)				
Dizziness	✓✓	✓	✓✓ [22, 23]	✓✓
Unsteadiness	✓✓	✓✓	✓✓	✓✓
Non-spinning vertigo	✓✓	✓✓	✓✓	✓
Temporal profile (Criteria A.1–3)				
	Fluctuating with momentary flares	Situational (provoked)	Situational (provoked), Persistent [23]	Persistent with diurnal variability [27]
Provocative factors (Criteria B.1–3)				
Upright posture	✓✓			✓ [75]
Active or passive motion	✓	✓	✓	✓✓
Moving visual stimuli or complex patterns	✓	✓	✓✓	✓✓
Precipitants (Criterion C.1)				
Vestibular syndromes	✓	✓	✓	✓
Other medical illnesses	✓			✓
Psychological distress	✓	✓		✓
Course of illness (Criteria C.1.a-b)				
	Long-standing, waxing/waning [18]	May be long-standing	May be long-standing	Chronic
Physical exam and laboratory findings (Criterion E)				
	Normal	Somatosensory dependence on posturography [41]	Central or peripheral vestibular deficits	Abnormalities related to comorbid conditions [75]
Features not incorporated into PPPD				
Anxiety	Part of PPV	Associated with SMD [41]	Associated with prolonged VV [23]	May be comorbid with CSD [80]
Depression	Part of PPV			May be comorbid with CSD [80]
Personality traits	Obsessive-compulsive traits are part of PPV			Neurotic, introverted traits may be risk factors for CSD [76]

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Criteria for the diagnosis of PPPD

PPPD is a chronic vestibular disorder defined by criteria A-E below. All five criteria must be fulfilled to make the diagnosis.

A. One or more symptoms of dizziness, unsteadiness, or non-spinning vertigo are present on most days for 3 months or more.

1. Symptoms last for prolonged (hours long) periods of time, but may wax and wane in severity.

2. Symptoms need not be present continuously throughout the entire day.

B. Persistent symptoms occur without specific provocation, but are exacerbated by three factors:

1. Upright posture,

2. Active or passive motion without regard to direction or position, and

3. Exposure to moving visual stimuli or complex visual patterns.

C. The disorder is precipitated by conditions that cause vertigo, unsteadiness, dizziness, or problems with balance including acute, episodic, or chronic vestibular syndromes, other neurologic or medical illnesses, or psychological distress.

1. When the precipitant is an acute or episodic condition, symptoms settle into the pattern of criterion A as the precipitant resolves, but they may occur intermittently at first, and then consolidate into a persistent course.

2. When the precipitant is a chronic syndrome, symptoms may develop slowly at first and worsen gradually.

D. Symptoms cause significant distress or functional impairment.

E. Symptoms are not better accounted for by another disease or disorder.

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Precipitating conditions

- Most common:
  - Peripheral or central vestibular disorders (PVD or CVD) 25–30% of cases
  - Attacks of vestibular migraine (VM) 15–20%
  - Panic attacks or anxiety manifest prominent dizziness 15% (each)
  - Traumatic brain injuries (TBI) or whiplash injuries of neck 10–15%
  - Autonomic disorders 7%
  - Less common: cardiac dysrhythmias, ADR ~3%
- Majority of conditions precede PPPD: acute or episodic in nature
- Onset of chronic symptoms of PPPD following acute illnesses
- Precipitants: generalized anxiety disorder, autonomic disorders, & peripheral or central degenerative conditions may develop insidiously

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PPPD

- May co-exist w other diseases or disorders
- Evidence of another active illness not exclude Dx of PPPD
- Clinical judgment must be exercised to determine the best attribution of pt's vestibular symptoms to all identified illnesses
- All symptoms susceptible to exacerbation with upright posture, motion, and exposure to complex visual stimuli
- Symptoms not increase immediately on standing, moving, or entering visually stimulating environments, tend to build throughout continued exposure
- Symptoms usually not return to baseline immediately on cessation of exposures, may last for hours or more

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Upright posture: active or passive movement

- Minimize adverse effects of upright posture by touching fixed objects, using gait aids, or holding onto other people, do not have to hold tightly to support their weight
- Rather, a light touch is sufficient to obtain the stabilizing effect of somatosensory input
- High velocity movements – prolonged or repeated are universally provocative whether encountered actively or passively

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Visual stimuli

Most troublesome

- Full field visual flow exacerbate symptoms for hours, even after brief exposures e.g., passing traffic especially at high speed on a highway or motorway, large crowds of people milling about
- Large complex patterns e.g., an expanse of busy carpeting, large store displays
- Wide-open spaces w distant or indistinct visuospatial reference points e.g., large fields, warehouses, atria
- Smaller visual targets at close distance e.g., books, mobile electronic devices
- Performing tasks that require precision visual focus even when sitting still e.g., using a computer, or watching television
- Increasing need to view information on electronic screens in the modern world: a bane for many PPPD

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Clinical course

- Most cases, PPPD develops as acute symptoms of precipitating conditions remit, no experience symptom-free intervals, as acute vertiginous symptoms fade, develop characteristic chronic symptoms of PPPD
- Other, a stuttering onset experience PPPD-like symptoms lasting days to weeks, until recurrences settle into a persistent course, short-lived precipitants, recurrent events (e.g., attacks of BPPV, migraine, or panic)
- Least often, gradual onset e.g. generalized anxiety, autonomic disorders, and degenerative diseases of PVD or cerebellum; chronic conditions develop slowly
- Most patients describe an acute, subacute, or stuttering onset of illness
- Clinical histories of gradually worsening chronic vestibular symptoms or balance problems have no identifiable starting points

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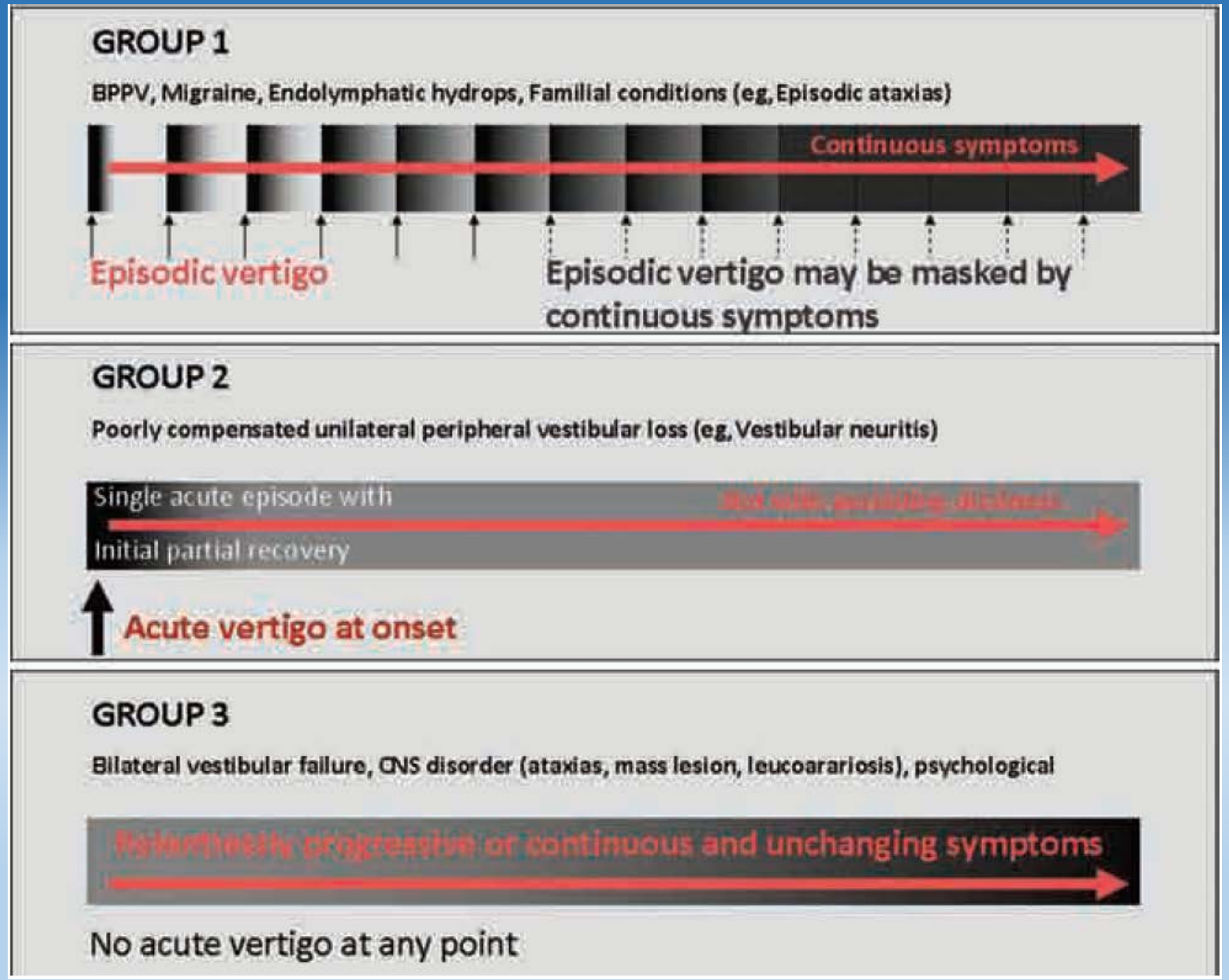

Stuttering onset

Acute vertiginous

Gradual onset

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Dx of PPPD

- Dx made by gathering clinical hx relevant to Criteria A-D
- No findings on PE, lab testing, or dx imaging pathognomonic of PPPD
- PE – If PPPD is the best diagnosis, either alone or in combination with other diseases or disorders (Criterion E)
- PPPD is not a diagnosis of exclusion

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DDx of PPPD

- Chronic sequelae of acute precipitants,
- Recurrent attacks of episodic precipitants,
- Ongoing manifestations of chronic precipitants,
- Other chronic vestibular syndromes, medical or psychiatric disorders that produce persistent unsteadiness or dizziness, and
- Adverse effects of regularly consumed prescription or non-prescription medications

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Chronic sequelae of acute precipitants

- Acute disorders potential for chronic symptomatic complications (e.g., VN or stroke leading to persistent uncompensated vestibulopathies)
- Hx of persistent non-vertiginous dizziness & unsteadiness provoked by upright posture, pts' own movements, & exposure to visual motion stimuli plus PE & lab evidence of good compensation (e.g., no spontaneous nystagmus or negative HIT, headshake, or stepping tests); indicates PPPD only active dx
- In contrast, presence of ongoing episodes of head motion-provoked vertigo or unsteadiness w/o persistent dizziness & PE incomplete compensation; argues against PPPD
- 3rd possibility: persistent dizziness & motion sensitivity plus head motion-provoked symptoms & PE incomplete compensation; indicate coexisting PPPD & uncompensated vestibulopathies

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BOX 1 Factors which may interfere with recovery following vestibular lesions (slightly modified from Bronstein and Lempert[31])

- Age
- Active migraine
- Brain lesions (particularly cerebellar)
- Peripheral neuropathy
- Visual disorders
  - Reduced visual acuity
  - Modified optics (eg, cataract operation)
  - Strabismus
- Factors reducing head movements (eg, neck stiffness)
- Psychosocial problems
- Medical interventions
  - Insufficient/inadequate counselling
  - Antivertiginous drugs
  - Tranquilisers
- Lack of mobility
  - Orthopaedic (eg, hip arthritis)
  - Excessive bed rest or patient advised not to move
  - Fear (eg, of vertigo or falling)
  - Avoidance of symptom provoking situations

Pract Neurol, 2010

Recurrent attacks of episodic precipitants

- When PPPD co-exists w these disorders, proper diagnosis rest on identifying characteristic symptoms of each active disorder
- Episodic disorders add distinctive vestibular symptoms to the background of PPPD
- VM – acute attacks of vertigo plus cephalalgia, photophobia and phonophobia, w or w/o visual aura
- BPPV – short-lived positional vertigo
- MD – attacks of vertigo, tinnitus, & fluctuating hearing

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Chronic dizziness

- Vestibular mechanisms control eye movements & pts' symptoms mediated by two separate but linked systems: brainstem & perceptual (presumably cortical) system
- In acute vertigo: eye movements & perception strongly coupled
- In chronic vertigo: eye movements correlate poorly w symptoms
- Whatever cause of failure to compensate, anxiety and/or depression, associated w long term symptoms & disability

Pract Neurol, 2010

Ongoing manifestations of chronic precipitants

- Some precipitants of PPPD = chronic conditions themselves (e.g., anxiety & depressive disorders, post-concussive syndrome, autonomic disorders, & heart diseases)
- Cause persistent unsteadiness or dizziness w or w/o precipitating PPPD
- When present alone: not greatly affected motion provocations of Criterion B as when PPPD exists

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Chronic anxiety and depressive disorders

- Chronic anxiety: generalized anxiety disorder, agoraphobia, social phobia, OCD, & traumatic stress disorders manifest w persistent dizziness
- Depressive disorders also may cause dizziness
- The 7-item Generalized Anxiety Disorders Scale (GAD-7) screen for pathological anxiety
- The 9-item Patient Health Questionnaire (PHQ-9) screen for depression
- The 14-item Hospital Anxiety and Depression Scale (HADS) covers both
- Positive results indicate anxiety or depressive disorder is likely, either as the sole cause of vestibular symptoms or co-existing w PPPD

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Postconcussive syndrome

- Following TBI or whiplash injury often experience chronic dizziness in addition to headache, insomnia, cognitive symptoms, and mood lability
- The presence or absence of other sequelae of injury will determine if additional diagnoses are warranted, e.g., BPPV, fracture temporal bone, perilymph fistula, cervical vertigo

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Autonomic disorders

- Most likely to precipitate PPPD, based on CSD research:
- Postural orthostatic tachycardia syndrome (POTS) & type 1 neurocardiogenic (vasovagal) syncope, most encountered in adolescents & young adults
- Orthostatic intolerance w or w/o hypotension from neurologic and cardiovascular illnesses (e.g., autonomic neuropathy) more common in older adults, part of multi-factorial dizziness
- Vital signs during autonomic challenges & sensitivity to visual stimuli when seated at rest best distinguish PPPD from autonomic disorders, recognizing potential for two problems co-exist

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Other chronic vestibular syndromes

- Bilateral vestibulopathy
- Neurodegenerative disorders (e.g., downbeat nystagmus syndrome and other cerebellar diseases)
- Mal de debarquement syndrome (MdDS)

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Bilateral vestibulopathy

- Best distinguished by characteristic findings on PE & lab testing, such as bilaterally positive HIT, diminish responses on caloric irrigation, or sinusoidal stimulation in a rotary chair
- PPPD not cause oscillopsia, but oscillopsia present in 30–40% of bilateral vestibulopathy
- PPPD susceptible to exacerbations when exposed to complex visual stimuli even when sitting still, whereas bilateral vestibulopathy have minimal symptoms when seated and stationary

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Bilateral vestibulopathy

BOX 2 Common causes of bilateral vestibular failure[25]

- Idiopathic (either recurrent vertigo or slowly progressive presentations)
- Gentamycin ototoxicity
- Post-meningitis
- Neurological: cranial/ peripheral neuropathies, cerebellar degeneration

- Oscillopsia during walking & balance problems in the dark after a course of gentamycin (not deaf) or meningitis (usually deaf)
- Often miss idiopathic cases (at least as common): only history slowly progressive balance difficulty (sometimes very mild) and oscillopsia during walking, running or riding a vehicle
- Some cases: episodes of vertigo or spontaneous paroxysmal oscillopsia gradually leading into this syndrome

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Chronic neurologic disorders

- Neurodegenerative disorders affect posture and gait: PD, cerebellar degeneration, including DBN syndrome & small vessel white matter disease may manifest w dizziness or unsteadiness when standing or walking before motor signs detected on PE
- The gradual onset of these complaints in relative absence of difficulties with complex or moving visual stimuli arouse suspicion: PPPD not correct diagnosis
- The best approach in this situation: period of prospective monitoring (typically 6–12 months)

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Mal de debarquement syndrome

- Persistent unsteadiness triggered by traveling on boats, aircraft, or automobiles, usually for at least a few hours
- Symptoms characteristically decrease during passive motion (e.g., riding in a car) & increase again when motion ceases
- "Spontaneous onset" version of MdDS: most pts had migraine or anxiety disorders: known precipitants of PPPD
- Major difference b/w MdDS VS PPPD: extent of tx response
- MdDS generally has a limited response to VR or medications, whereas tx of PPV, SMD, VV, CSD, and PPPD, itself, showed significant improvements w vestibular habituation or serotonergic antidepressants

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Adverse effects of medications

- Prescription medications, OCT preparations, & dietary supplements may cause dizziness, unsteadiness, or vertigo
- Vestibular symptoms caused by newly administered medications or changed doses of existing medications may precipitate PPPD

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Other functional forms of vestibular symptoms

- Persistent vestibular symptoms not fit dx criteria of either PPPD or other well defined chronic vestibular syndromes include constant, invariant vertigo, unsteadiness, or dizziness, complex body motions in multiple directions simultaneously, and kaleidoscopic swirling movements of large portions of the visual field
- Pts w these complaints often report a lack of provoking or mitigating factors
- Many pts: these functional forms of vestibular symptoms accompanied by other chronic physical complaints such as fatigue & pain, raising possibility manifestation of a broader somatic symptom disorder or bodily distress disorder

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Gait disorders, falls, and near falls

- Clinical evidence of significant changes in gait or recurrent falls or near falls indicates the presence of a structural or functional gait disorder
- PPPD may co-exist with these disorders

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Estimates of the prevalence of PPPD

- 15–20% among all pts presenting for evaluation of vestibular symptoms, the most common dx among young adults and the second most common among all adults, trailing only BPPV
- Average duration of illness at time of tertiary consultation = 4.5 yrs w some pts experiencing symptoms for decades
- Average age of presenting for evaluation = mid-40 s, range from adolescence to late adulthood, female predominance
- Disability varied: from few limitations in ADL to those severely impaired & unable to work

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Estimates of the incidence of PPPD

- PPPD-like chronic dizziness or persistent VV – 25% of pts after 3–12 months FU, despite adequate compensation or recovery from initial illnesses
- A long-term FU study of pts w PPV: found only minority experienced spontaneous resolution of symptoms
- Most: chronic waxing and waning course and ¾ developed anxiety or depressive comorbidity
- Majority PPPD: likely to remain symptomatic w/o tx, regardless of initial precipitant

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Possible pathophysiologic processes underlying PPPD

- *Possible risk factors:* anxiety-related personality traits or a personal or FmHx of anxiety disorders may develop PPPD following relevant precipitants
- *Initial reactions:* possibility highly anxious response to precipitating events – pivotal initial pathophysiologic process in development of PPPD, & early symptom-specific interventions might counter this effect (cognitive behavioral therapy – CBT)
- *Possible alterations in postural control:*
- *Possible association with visual dependence:*
- *Possible changes in activity and connectivity of crucial brain regions:*

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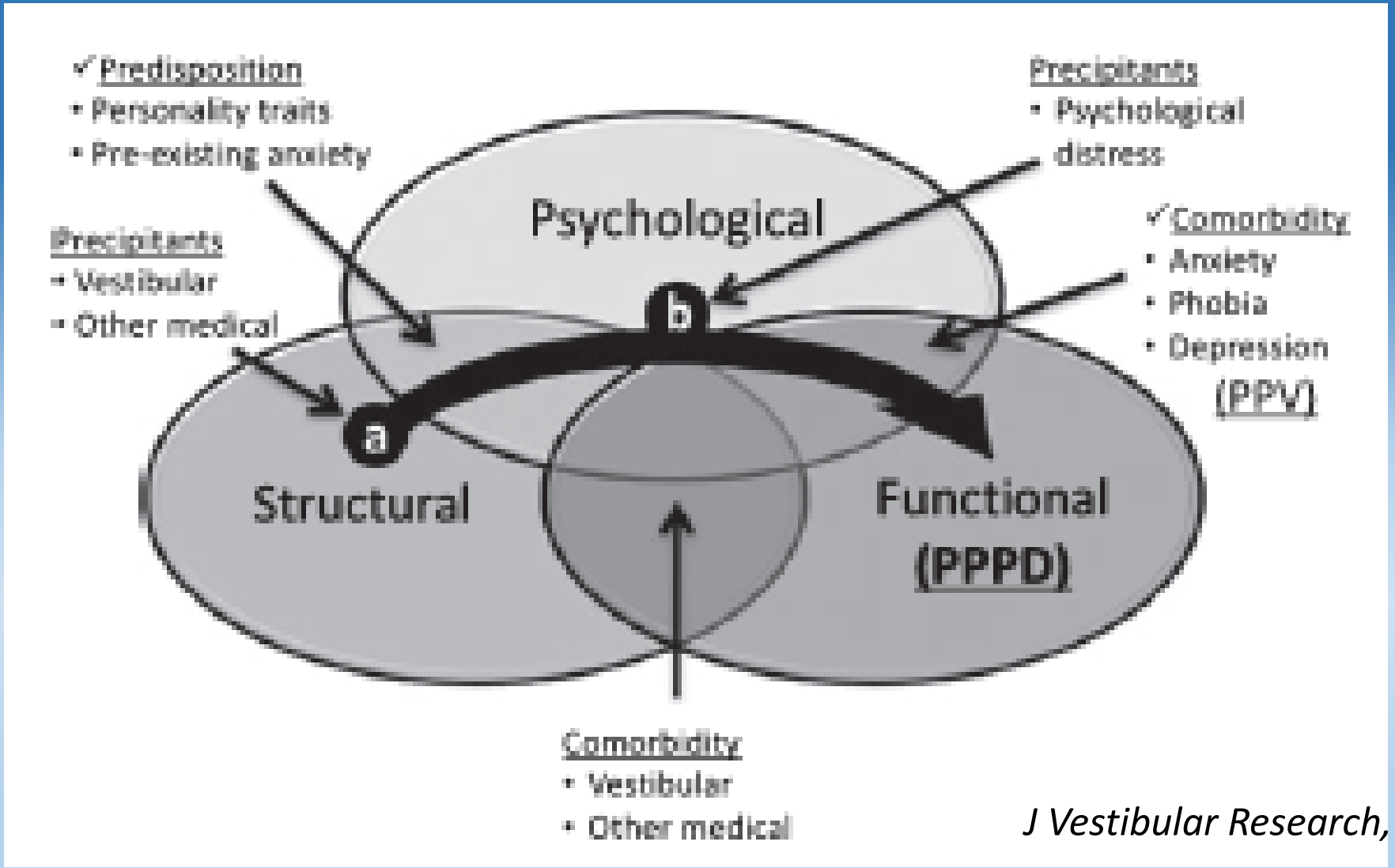
Putative mechanisms of PPPD



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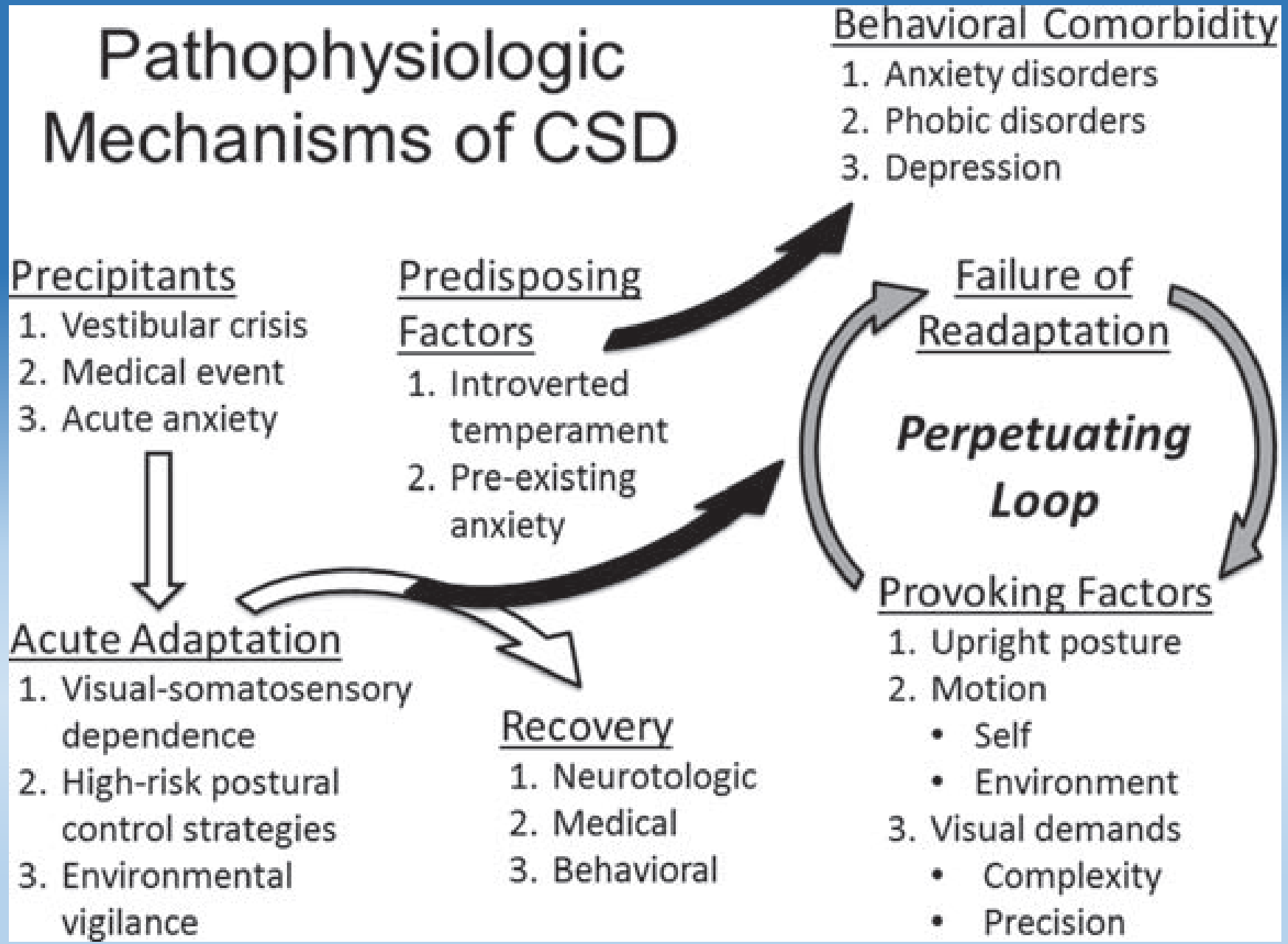
Pathophysiologic Mechanisms of CSD



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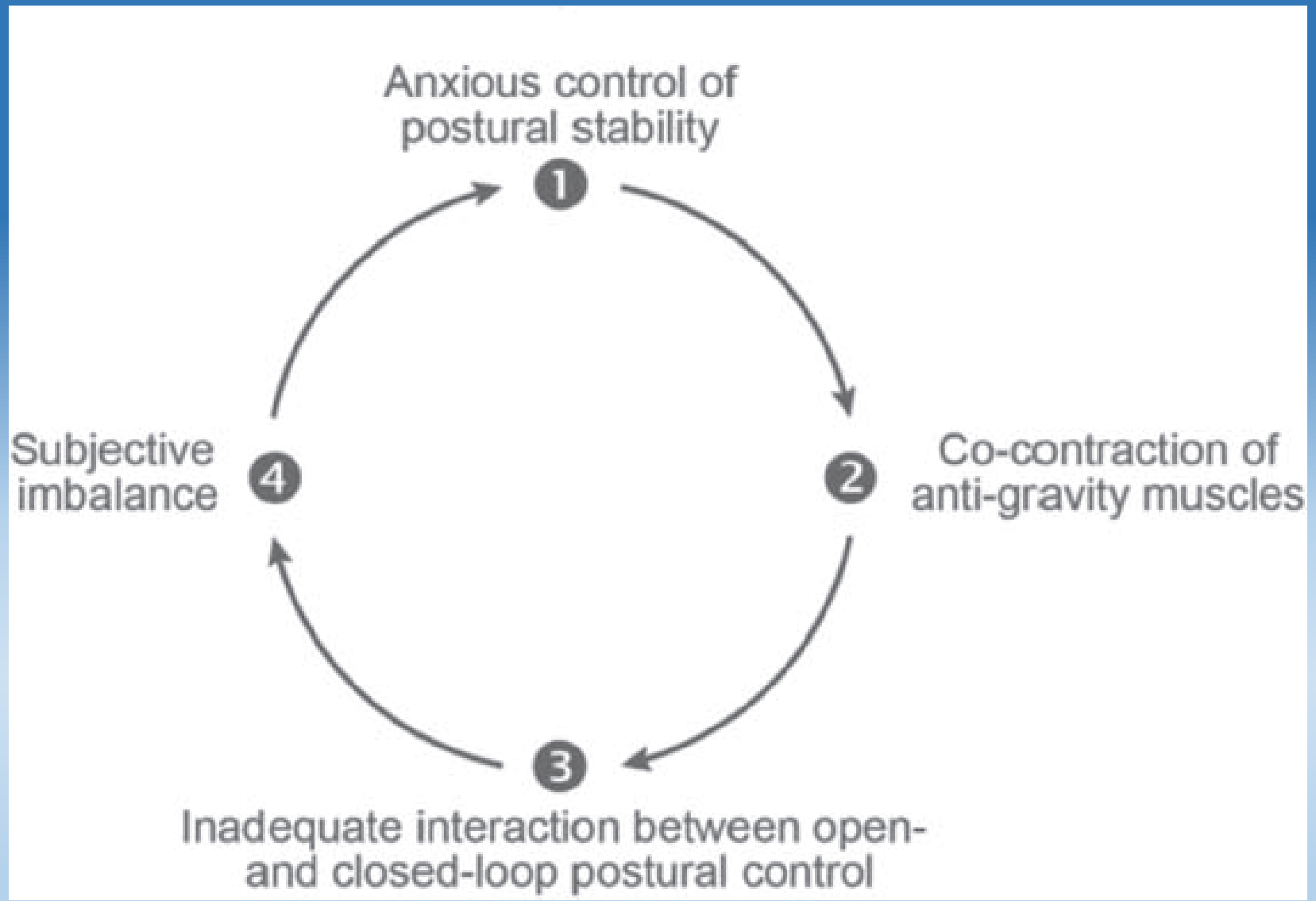

Circular cascade of symptoms

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Disability status & psychological traumatization

- First belief: disability status, healthcare utilization & pt dissatisfaction w medical care are greater among pts w functional disorders than w structural diseases
- Functional or psychiatric disorders no more impaired than structural illnesses
- Second belief: experiences of psychological traumatization and adverse life events differentially cause functional and psychiatric presentations of neurologic symptoms
- Childhood or adulthood adversity no more common among pts w functional or psychiatric than structural vestibular disorders
- A history of adversity increased likelihood of severe symptoms & handicap, not help DDx of vestibular symptoms
- Instead, marked poorer outcomes regardless of final dx

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Cocontraction of leg antigravity muscles

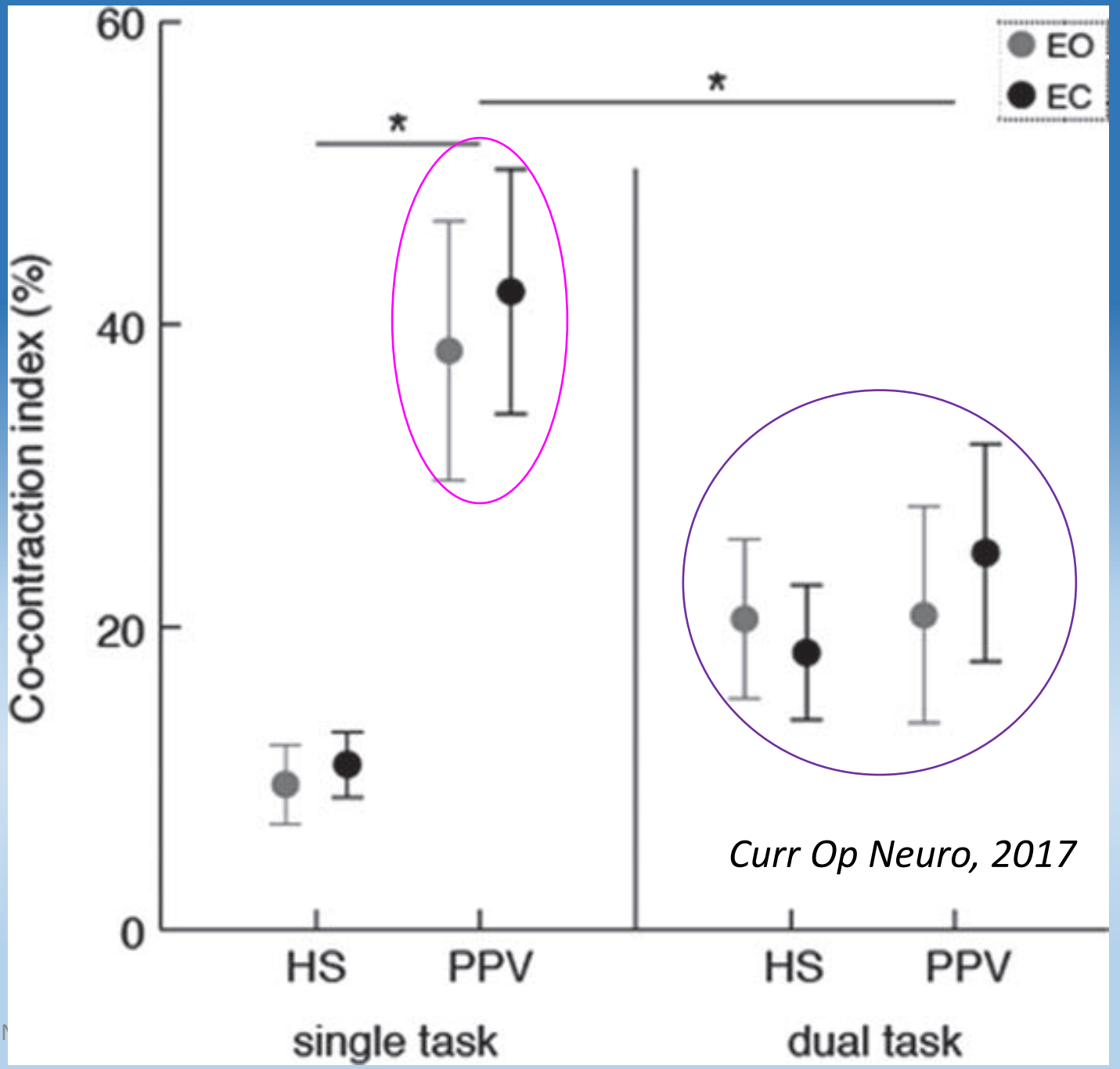
Cocontraction indices of examined leg muscle pair (i.e., tibialis anterior and soleus muscle) in healthy subjects (HS) & pts w PPV for 4 stance conditions

Single task: enhanced cocontractions

- standing w eyes open (EO)
- standing w eyes closed (EC)

Cognitive dual task (dual distracted task): normalized performance

- standing w eyes open
- standing w eyes closed



Neuro-physiological findings in Functional Dizziness

- Postural control of acrophobia (fear of heights) similar to PPV
- Share two criteria: dissociation b/w subjective & objective risk of falling and no apparent increase of actual falls compared w normal controls
- Inadequate strategy of balance regulation & muscle cocontraction in PPV considerably improves & normalizes to that of HS when perform cognitive tasks
- Distraction: an effective coping strategy for preventing PPV attacks in susceptible pt
- Anxiety also affects ocular motor reflexes & gaze control as in CSD
- Acrophobia tend to freeze their gaze to the horizon when exposed to heights

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Assessment

[1] A thorough balance & vertigo assessment, symptoms — (dys)function and (dis)abilities per se — rather than just making an aetiological diagnosis

[2] Problems following vestibular pathology, e.g., muscular pain from increased muscle tension (neck), stress, fatigue & chronic anxiety; affect ability to participate in rehabilitation program

- Inactivity: from bed rest, fear, anxiety or other factors; also delays and impairs complete compensation

Pract Neurol, 2010

Assessment

- *Functional assessment:* sequential actions be rated or timed, e.g, 'Get up and Go' test = quick screening tool for detecting balance problems in elderly or Dynamic Gait Index examines ability to adapt to various task demands
- *Systems assessment:* Somatosensory, visual and motor function assessed w conventional neuro-exam, type & effectiveness of postural reactions examined w gentle or vigorous pushes/pulls to trunk to elicit ankle, hip or stepping motor postural strategies
- *Symptom assessment: 1*ry vestibular symptom, associated autonomic & psychological correlates (require additional reassurance or tx) if pts develop anxiety or hyperventilation

Pract Neurol, 2010

Assessment

- *Dizziness triggers:* e.g., visual stimuli such as large field stimuli (a moving train) or self-motions (travel sickness)
- Critical in guiding tx since the principle 'we'll work on whatever turns your dizziness on' (desensitization tx)
- VR exercises for pts will largely be based on the findings

Pract Neurol, 2010

TABLE 1 Relevant questions and investigations in the patient with chronic disequilibrium (slightly modified from Bronstein and Lempert[31])

Question	Possible diagnosis	Procedure
Oscillopsia?	Downbeat nystagmus syndrome Bilateral loss of vestibular function	Brain MRI Vestibular tests (bedside+laboratory)
Neck or arm pain? Sphincter dysfunction? Long tract signs?	Cervical cord compression/canal stenosis	MRI spine
Memory loss? Incontinence?	Hydrocephalus, or white matter 'small vessel' disease	MRI/CT head
Slowness, tremor?	Parkinsonism	Neurological examination DAT scan
Motor incoordination? Speech disorder?	Cerebellar ataxia	Neurological examination+brain MRI, paraneoplastic and genetic tests
Vascular risk factors? Strokes?	White matter 'small vessel' disease (leucoaraiosis)	Brain MRI/CT Cardiovascular assessment
Distal numbness?	Peripheral neuropathy	Neurological examination+clinical neurophysiology

Laboratory testing

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Chronic Dizzy: Management

4 equally important components management of any patient w dizziness or vertigo, whether acute, recurrent or chronic:

- Treatment of specific vestibular condition if there is one (e.g., BPPV, VM, MD etc.)
- Short term non-specific pharmacological treatment of vertigo and associated nausea
- Physical (vestibular) rehabilitation
- Provision of information, counselling & reassurance plus psycho-education, CBT

Pract Neurol, 2010

Disease specific treatment

- Not all patients require action in all four domains
- An active underlying vestibular disorder identified: BPPV, MD, needs to be treated because, each vertigo attack, chronic symptoms tend to increase
- Particularly important to treat migraine: both cause vertigo and be triggered by it
- BPPV: usually require repositioning treatment and nothing else
- Vestibular migraine: if no interictal or chronic symptoms, just require antimigraineous drugs but no rehabilitation
- In chronic dizziness typically require counselling and rehabilitation, but no drugs

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Non-disease specific pharmacological treatment

- Vertigo or nausea reduced with medications only for acute attack or recurrent episode
- Long term vestibular suppressants, tranquilizers: harmful to the process of vestibular compensation, only be used for truly acute vertigo and stopped as soon as vertigo begins to recede

Pract Neurol, 2010

General support

- Reassurance, information and counselling
- Pts w long term dizziness often wandered from clinic to clinic, from specialist to specialist, for months or years
- On basis of normal brain scans some doctor likely to said
- 'there's nothing wrong with you', or
- 'there's nothing that medical science can do for you' or,
- worse, 'it's all in your mind'
- This is not only the wrong approach but it is usually not true

Pract Neurol, 2010

TABLE 2 Summary of our approach to the patient with chronic vestibular symptoms (slightly modified from Bronstein and Lempert[31])

Approach	Specific goals—try to establish:
Attempt retrospective diagnosis	Did it all start as BPPV, vestibular neuritis, recurrent vertigo (eg, migraine, Ménière's disease), brainstem stroke? Are the original symptoms still present? Or are we only dealing with chronic dizzy symptoms?
Other potentially important problems	Is vestibular compensation impeded due to additional problems? Fluctuating vestibular disorder, recurrent vertigo Visual problems such as squint, cataract operation Proprioceptive deficit such as peripheral neuropathy (diabetes/alcohol) Neurological problems such as 'small vessel' white matter disease Orthopaedic problems and lack of mobility Loss of confidence, fear of falling, psychological disorders Age: all of the above possible but try to identify which one(s)
Treatment is multidisciplinary	Treat any episodic vertigo specifically: BPPV: repositioning manoeuvres Vestibular migraine: migraine prophylaxis Ménière's disease: low salt diet, diuretics,betahistine Rehabilitation (and simple counselling): all patients Treat underlying complicating factors: eg,orthopaedic, depression, diabetes, migraine Do *not* prescribe vestibular suppressants or tranquilisers, stop/reduce them if possible
Make sure the 'chronic dizziness' is not a gait disorder	'Is your problem a head problem or a leg problem?' Ask about falls Observe: gait (including heel to toe), postural reactions and Romberg's sign Eye movement and neurological examination: Bilateral vestibular failure: oscillopsia, unsteady in the dark, abnormal doll's head/head thrust test Cerebellum: abnormal eye movements, gait/limb ataxia Parkinsonism: rest tremor, increased tone, akinesia Spasticity: increased reflexes, Babinski's sign Peripheral neuropathy: distal weakness (cannot walk on heels or toes) and sensory loss Frontal disorder/hydrocephalus: gait 'ignition' failure, gait apraxia, shuffling

BPPV, benign paroxysmal positional vertigo.

Pract Neurol, 2010

Table 1. Dosing Schedule for Selective Serotonin Reuptake Inhibitors

Drug	Starting Daily Dose, mg	Target Daily Dose by Week 4, mg	Subsequent Daily Increases (2- to 4-wk Intervals), mg	Maximum Daily Dose, mg
Fluoxetine hydrochloride	5-10	20	20	80
Sertraline hydrochloride	12.5-25	50	50	200
Paroxetine hydrochloride	5-10	20	20	60
Citalopram hydrobromide	5-10	20	20	40
Escitalopram oxalate	5	10	10	20

Table 2. Selective Serotonin Reuptake Inhibitor Treatment Outcome by Pattern of Illness for All Patients

		Positive Response		No Benefit	
Pattern of Illness	No. of Patients	Remission (CGI-I Score of 1)	Partial Response (CGI-I Score of 2)	No Response (CGI-I Score of ≥3)	Not Tolerated
Otogenic	28	15	3	4	6
Psychogenic	31	16	7	4	4
Interactive	29	5	13	5	6

Table 3. Mean Improvement for Patients Who Completed Selective Serotonin Reuptake Inhibitor Treatment

Pattern of Illness	No. of Patients	CGI-I Score, Mean ± SD
Otogenic	22	1.50 ± 0.80
Psychogenic	27	1.59 ± 0.84
Interactive	23	2.04 ± 0.77

SSRI in CSD

Arch Otolaryngol HNS, 2005

CSD - Treatment

- Interactive group experienced a clear reduction in symptoms not respond as vigorously to SSRI treatment, significantly fewer pts experienced a full remission compared w other groups
- Long-standing nature anxiety diathesis: limited extent to benefit from short-term, single-modality therapy
- Interactive illness need adjunctive or alternative therapies — pharmacologic, psychotherapeutic, surgical, and VR to completely resolve their symptoms

Arch Otolaryngol HNS, 2005

Treatment

- Posturography used to monitor therapeutic effects of psycho-education, CBT, medication & physiotherapy
- CBT in combination w VR significant improved symptoms of small groups chronic nonspecific dizziness & PPV but transient positive effect after 1 yr
- Recent interventions addressed dysfunctional illness beliefs and behaviors in pts w chronic vestibular morbidity
- Study suggested combined approach using psycho-education, CBT, VR, & antidepressant drugs, when needed, may reduce dysfunctional illness behavior and dizziness
- 78% pts reported sustained reductions of dizziness symptoms after at least 1 yr of flexible treatment (average 32 months)

Curr Op Neuro, 2017

Important lessons

[1] Individualized use of currently available therapies benefits pts who receive evaluations thorough enough to guide multidisciplinary tx plans

[2] A systematic approach to tx over several weeks to months may be needed to achieve favorable outcomes

[3] Treatment gains can be sustained

- Beneficial efficacy of serotonergic antidepressants, VR exercise & CBT been investigated in pts w CSD in open-label, retrospective FU, and RCT
- Additional studies of these tx in cohorts of pts w chronic nonspecific dizziness

Curr Op Neuro, 2017

Conclusion

- Functional & psychiatric disorders cause vestibular symptoms (vertigo, dizziness, and unsteadiness) more common than many well known structural vestibular disorders
- Treatment plans include pt education, VR, CBT & medication substantially reduce morbidity and can potentially sustain remission if applied systematically (Integrative Medicine)

Curr Op Neuro, 2017