MAYO CLINIC

MOTION SICKNESS

Pathophysiology and Clinical Presentation

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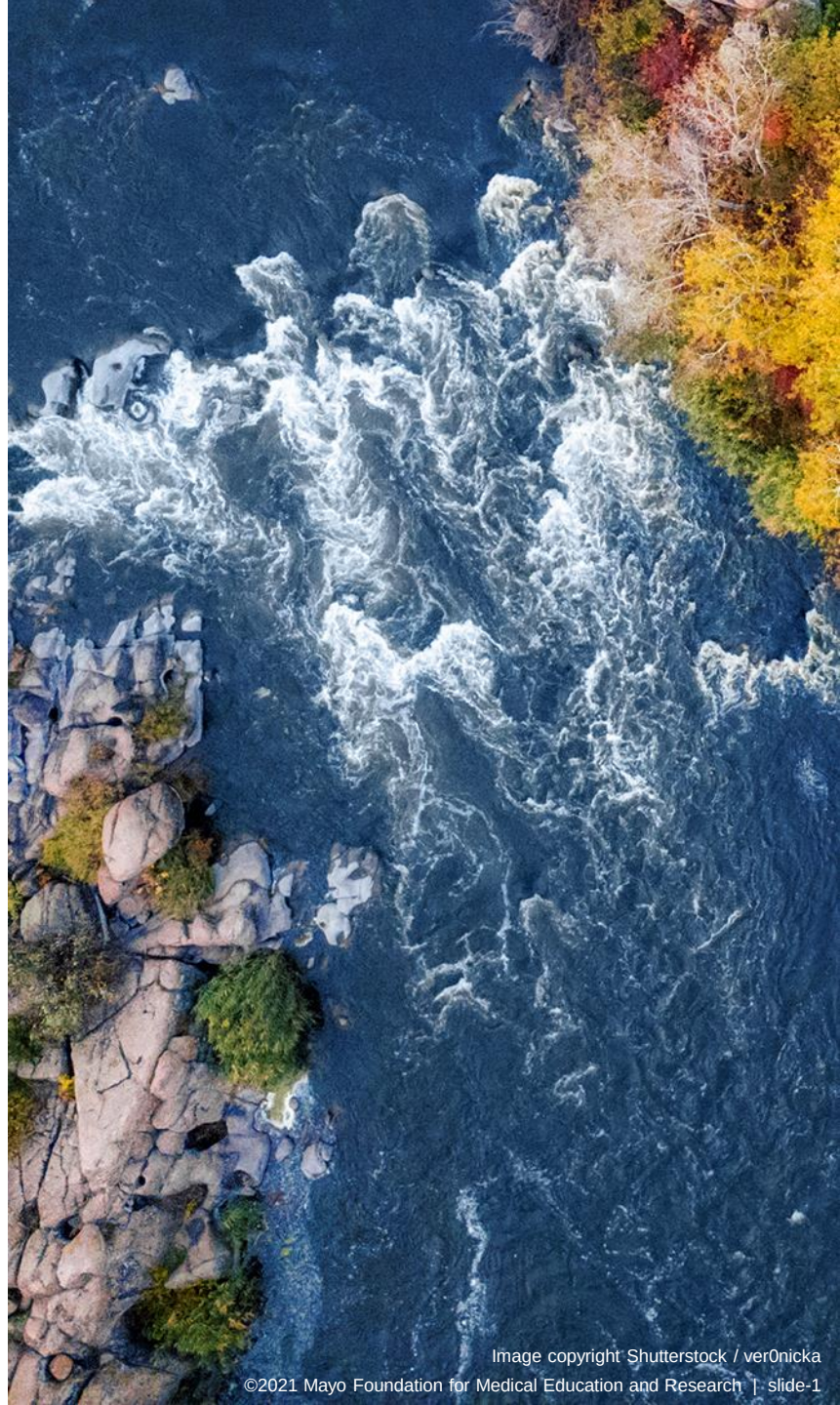
Canadian Academy of Audiology
13 October 2023



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DISCLOSURES

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TODAY

- Identify the presumed pathway for motion sickness generation
- Review available clinical tools for motion sickness
- Describe how motion sickness may present in various vestibular pathologies

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WHAT IS MOTION SICKNESS?

- *Kinetosis*
- Originally described by Hippocrates: “sailing on the sea proves that motion disorders the body”
- **Illness caused by motion**

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SYMPTOMS

- Nausea
- Vomiting
- Cold sweat
- Headache
- Dizziness
- Fatigue
- Loss of appetite
- Increased salivation

Takov & Tadi 2019; Hromatka et al 2015

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MOTION SICKNESS

- Seasickness
- Carsickness
- Airsickness
- Centrifugal motion sickness
- Dizziness due to spinning
- Virtual reality
- Space motion sickness

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MOTION SICKNESS

- **Seasickness**
- Carsickness
- Airsickness
- Centrifugal motion sickness
- Dizziness due to spinning
- Virtual reality
- Space motion sickness

- Most common form of motion sickness
- 25% of passengers on large ships develop motion sickness within 2-3 days
- Increased incidence in smaller ships, adverse weather – up to 60%

Koch et al 2018

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MOTION SICKNESS

- Seasickness
- **Carsickness**
- Airsickness
- Centrifugal motion sickness
- Dizziness due to spinning
- Virtual reality
- Space motion sickness

- Up to 4%
- Increased prevalence in high velocity situations (e.g., rally cars), sitting in the back seat, when reading

Koch et al 2018; Golding & Gresty 2015

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MOTION SICKNESS

- Seasickness
- Carsickness
- **Airsickness**
- Centrifugal motion sickness
- Dizziness due to spinning
- Virtual reality
- Space motion sickness

- <1% of travelers in pressurized commercial cabins
- 10-31% of student aviators – decreases with exposure

Murdin et al 2011

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HOW COMMON IS MOTION SICKNESS?

- **Very**
  - *Anyone can be motion sick with the "right" stimulus.*
  - 1/3 of individuals are highly susceptible
  - Most everyone will experience motion sickness at least once.

Takov & Tadi 2019; Herron 2010

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HOW COMMON IS MOTION SICKNESS?

- Susceptibility associated with gene-environment interaction
- Gender
  - More common in women, especially during menstruation and pregnancy
- Race / ethnicity

Takov & Tadi 2019; Zhang et al 2016; Golding 2016; Koch et al 2018; Schmal 2013; Lawther & Griffin 1988; Paillard et al 2013; Golding 2006; Zhang et al 2016; Stern et al 1996; Reavley et al 2006; Murdin et al 2011; Hromatka et al 2015

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HOW COMMON IS MOTION SICKNESS?

- Family history
  - 2x risk if parent had pediatric motion sickness
  - Monozygotic twins generally concordant for motion sickness
  - Heritability estimates: 55-70%
  - Hromatka et al (2015) studied >80k individuals, finding 35 single-nucleotide polymorphisms associated with motion sickness
  - Genes mapped to chromosome 4

Prieson 2019; Takov & Tadi 2019; Zhang et al 2016; Golding 2016; Koch et al 2018; Schmal 2013; Lawther & Griffin 1988; Paillard et al 2013; Golding 2006; Zhang et al 2016; Stern et al 1996; Reavley et al 2006; Murdin et al 2011; Hromatka et al 2015;

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HOW COMMON IS MOTION SICKNESS?

- Age
  - Rare <2 years assumed due to insufficient visual input, recumbent position
  - 6-12 years – most susceptible! Peak symptoms noted 9-10 years
  - Susceptibility declines through puberty and adulthood, perhaps associated with habituation
  - Rare >50 years

Gahlinger 1999; Schmal 2013; Golding, 2016; Koch et al 2018; Takov & Tadi 2019; Murdin et al 2011; Reavley et al 2006; Paillard et al 2013

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HOW COMMON IS MOTION SICKNESS?

- Other associations
  - Increased prevalence with migraine, vertigo, Meniere's disease
  - Increased with sleep deprivation
  - Blind individuals are as susceptible as sited individuals with vision denied
  - Less prevalence found in dancers, rope walkers, acrobats, vestibular hypofunction

Takov & Tadi 2019; Paillard et al 2013; Bertolini & Straumann 2016; Cuomo-Granston & Drummond 2010; Drummond 2005; Golding & Patel 2017; Murdin et al 2015; Kaplan et al 2017; Lackner 2014; Golding 2016; Graybiel 1970

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HOW COMMON IS MOTION SICKNESS?

- Psychological component
  - Symptoms can begin even before sailing, or even at the sight of a ship
  - When one individual develops motion sickness, others are likely to develop symptoms
  - 45% benefit from placebo

Schmal 2013; McIntosh 1998

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MOTION SICKNESS

Onset

- Insidious onset
- Follows exposure to unfamiliar motion, inciting event

Early

- General discomfort, malaise, reduced alertness, increased lethargy, apathy

Mid

- Nausea
- Increased with full stomach, physical illness, stuffy atmosphere, tobacco smoking, offensive odor, sight of vomiting, fear, excitement

Takov & Tadi 2019; Brainard & Gresham 2014

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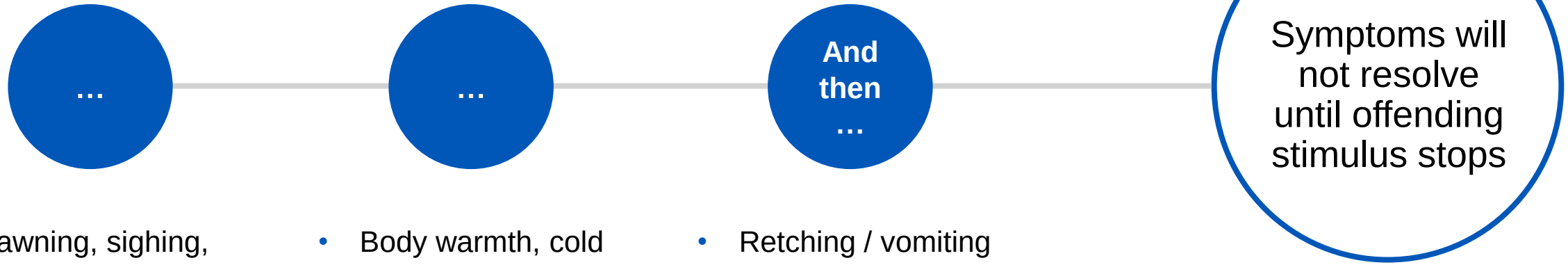

- Yawning, sighing, anorexia, increased salivation, burping, headache, eye strain, blurred vision, non-vertiginous dizziness, drowsiness, spatial disorientation, difficulty focusing /concentrating, hyperventilation

- Body warmth, cold sweats, pallor
- Decreased blood pressure

- Retching / vomiting

- Symptoms resolve completely within 24 hours

Golding 2016; Matsangas & McCauley 2014a; Matsangas & McCauley 2014b; Javaid et al 2019; Chan et al 2006

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POST-MOTION

- Aren’t we done?!
- Any parents in the room?

SOPITE SYNDROME

- Symptoms associated with prolonged periods of motion
- May be the sole manifestation of motion sickness
- May occur before / after other signs of motion sickness
- Drowsiness, yawning, disinclination for work, lack of social participation, mood changes, apathy, sleep disturbances, other fatigue-related symptoms

Graybeil & Knepton 1976; Lawson 1998

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- We can adapt and reduce symptoms over time – usually over the course of a few days.
- Some don’t habituate or may develop atypical compensation (i.e., mal de debarquement)

Golding 2016; Shankar Kikkeri & Siddiqui 2018

Motion sickness = unpleasant.

Why does this mechanism exist?

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THEORY

- Why do we experience motion sickness?

SENSORY CONFLICT

- Discontinuity between visual, vestibular, somatosensory input OR between semicircular canal and otolith input

NEURAL MISMATCH

- Discontinuity between ongoing perception and long-term memory; focus on limbic system function

DEFENSE AGAINST POISONING

- Discontinuity between vestibular / visual perception interpreted as hallucination (i.e., poisoning)

NYSTAGMUS HYPOTHESIS

- Vestibular stimulation of extra-ocular muscles leads to stimulation of vagus nerve

Kohl 1983; Lackner 2014; Treisman 1977; Ebenholtz et al 1994

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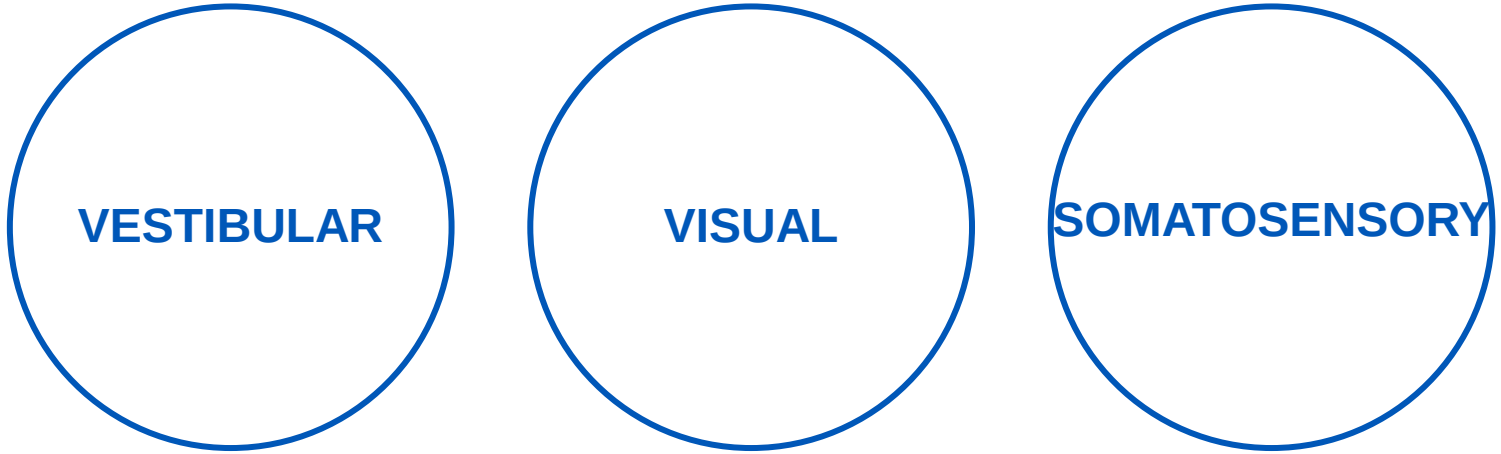
SENSORY CONFLICT

- Most commonly evaluated and accepted theory of motion sickness
- Describes conflict between various sensory systems, especially vestibular / visual.
- Numerous environments could lead to sensory conflict.



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SENSORY CONFLICT

Why does this happen?

Sensory information is different than expected based on past experience.

Inter-modality conflict – typically vision versus vestibular

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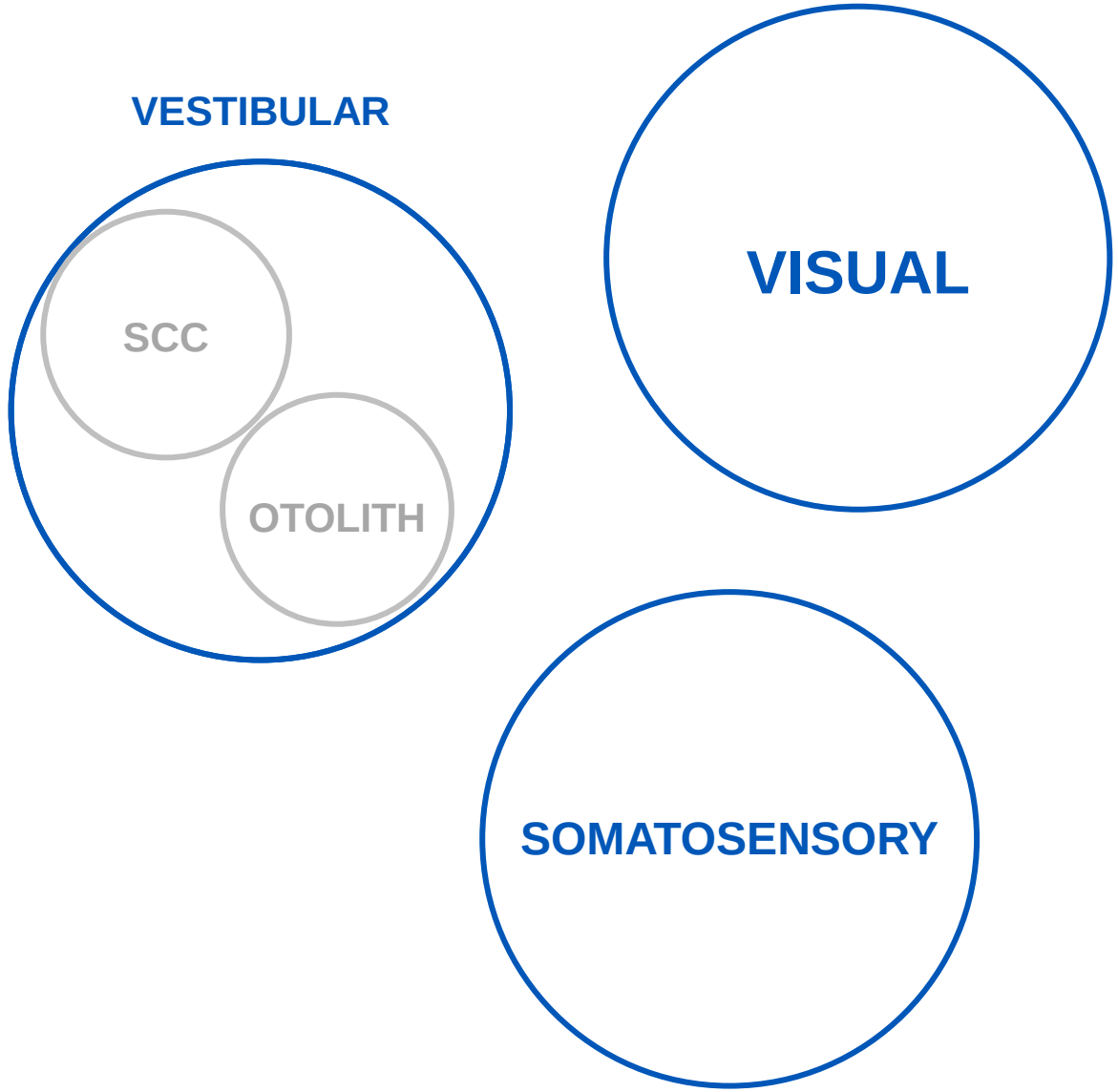
SENSORY CONFLICT

Why does this happen?

Intra-labyrinthine conflict



Previc 2018

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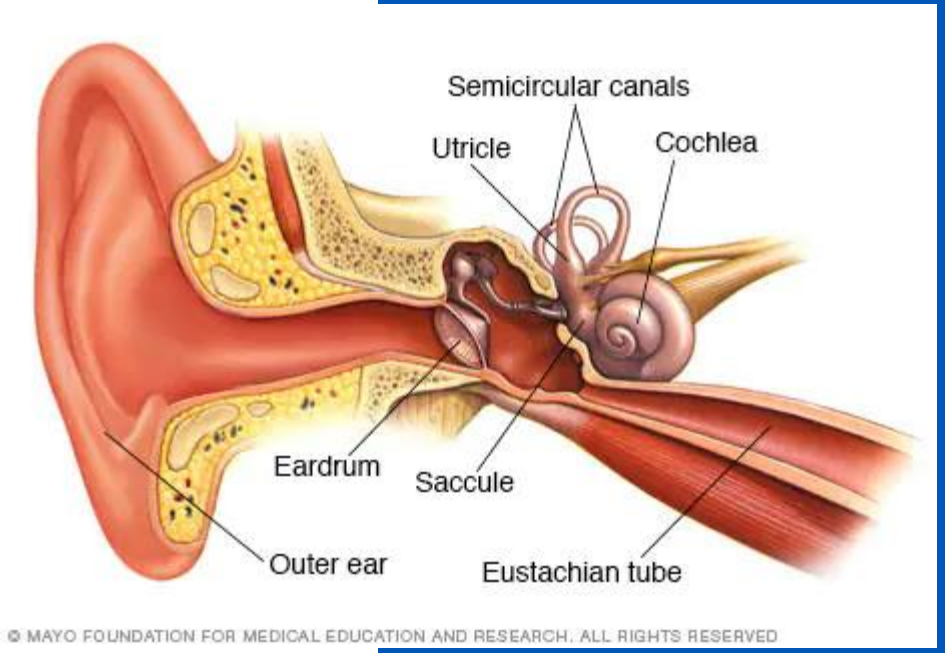
INTRA-LABYRINTHINE CONFLICT



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- If end organ afferent information shifts out of balance due to reduced otolith input, semicircular canal activity may be perceived as overactive and trigger the motion sickness cascade.
- Each end organ may have a different role in motion sickness.

Previc 2018

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PMCID: PMC5144688
PMID: 27385797

Contribution of intravestibular sensory conflict to motion sickness and dizziness in migraine disorders

Joanne Wang[1] and Richard F. Lewis[2,3,4]

Abstract Go to:

Migraine is associated with enhanced motion sickness susceptibility and can cause episodic vertigo [vestibular migraine (VM)], but the mechanisms relating migraine to these vestibular symptoms remain uncertain. We tested the hypothesis that the central integration of rotational cues (from the semicircular canals) and gravitational cues (from the otolith organs) is abnormal in migraine patients. A postrotational tilt paradigm generated a conflict between canal cues (which indicate the head is rotating) and otolith cues (which indicate the head is tilted and stationary), and eye movements were measured to quantify two behaviors that are thought to minimize this conflict: suppression and reorientation of the central angular velocity signal, evidenced by attenuation ("dumping") of the vestibuloocular reflex and shifting of the rotational axis of the vestibuloocular reflex toward the earth vertical. We found that normal and migraine subjects, but not VM patients, displayed an inverse correlation between the extent of dumping and the size of the axis shift such that the net "conflict resolution" mediated through these two mechanisms approached an optimal value and that the residual sensory conflict in VM patients (but not migraine or normal subjects) correlated with motion sickness susceptibility. Our findings suggest that the brain normally controls the dynamic and spatial characteristics of central vestibular signals to minimize intravestibular sensory conflict and that this process is disrupted in VM, which may be responsible for the enhance motion intolerance and episodic vertigo that characterize this disorder.

Keywords: vestibular, migraine, motion sickness, vertigo, eye movements

Evaluation of two ocular characteristics: 1) VOR attenuation with head tilt, 2) eye's rotational axis shifts toward gravity

INTRA-LABYRINTHINE CONFLICT

- Control / migraine subjects demonstrated evidence of canal-otolith resolution
- Vestibular migraine subjects did not.

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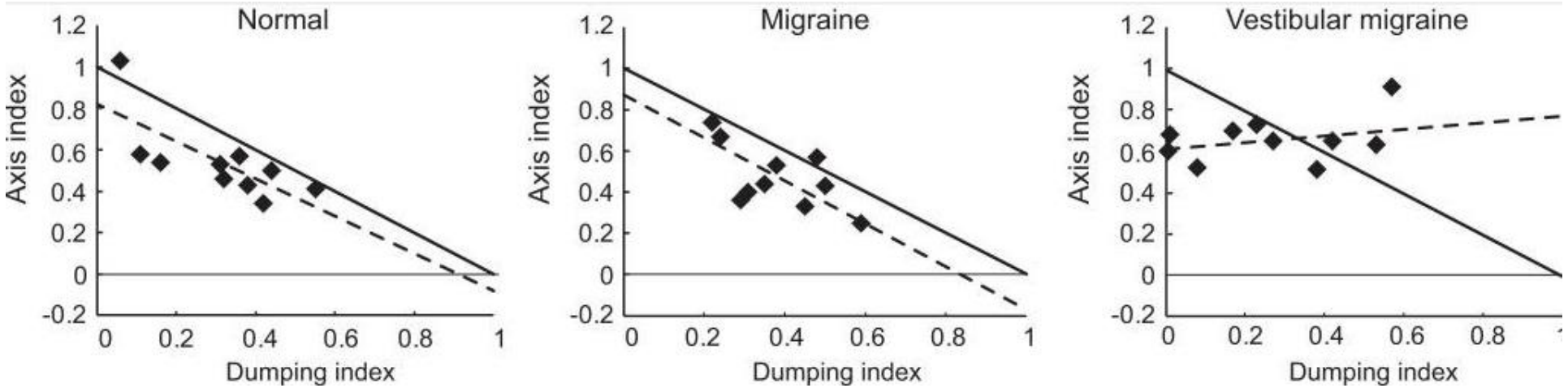
INTRA-LABYRINTHINE CONFLICT



- **Axis index** = [axis shift(tilted) – axis shift (upright)/45˚]; if the eyes rotational axis shifted by a magnitude equal to the size of the head tilt (e.g., aligned with gravity), then the brain interprets the rotational cue as occurring around an earth vertical axis
- **Dumping index** = [time constant(upright) – time constant(tilted)/T_c(upright)]; if tilting had no effect on time constant, then DI = 0 but if tilting eliminated VOR (i.e., suppressing velocity storage and resolving sensory conflict), DI = 1.
- 0 = no contribution to the resolution of canal-otolith conflict, 1 = complete resolution
- Solid lines = optimal conflict resolution
- Dashed lines = regressions

Wang & Lewis 2016

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INTRA-LABYRINTHINE CONFLICT

- Poor centrally-mediated conflict resolution may drive motion sickness

- Focus on cerebellum, vestibular nuclei
- Nodulus is key for integrating semicircular canal and otolith information
  - Encodes orientation to gravity, pitch and roll vectors
  - Inhibits velocity storage integrator

- Suppressing the velocity storage integrator stops motion sickness development if done before motion occurs

Wang & Lewis 2016; Yakushin et al 2003; Angelaki & Hess 1994; Angelaki & Hess 1995; Kitama et al 2014; Cohen et al 1992

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VESTIBULAR SYSTEM ROLE

KEY COMPONENTS

- Head / body motion
- Ability to sense spatial / gravitational vertical
- Ability to determine whether the body's orientation vector is misaligned with spatial vertical in roll

Motion sickness is a response to movement of the body's orientation vector (eigenvector away from gravity and/or gravito-inertial acceleration) in roll (Bles 1998, Cohen 2004)

Motion sickness in spaceflight is caused by the inability to sense gravity → inability to align the eigenvector of the body orientation vector (velocity storage) with the spatial vertical.

Bles 1998; Cohen 2004; Bertolini & Straumann 2016; Cohen 2011; Donohew & Griffin 2004; Joseph & Griffin 2008; Dai et al 2010

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VESTIBULAR SYSTEM

- Peripheral vestibular system is essential to producing motion sickness.
- Vestibulo-autonomic symptoms triggered from the velocity storage integrator in the vestibular nuclei and nodulus of the vestibulo-cerebellum.
- Velocity storage
  - Nystagmus / perception after rotation ends, description attributed to Mach (1875)
  - Velocity storage integrator: mechanism for altering input angular acceleration into a converted angular velocity signal.
  - This integrator holds the axis of eye rotation aligned with gravity and spatial vertical.

Tyler & Bard 1949; Bard & Woolsey 1947; Mach 1875; Robinson 1977; Raphan et al 1981; Raphan & Cohen 1985; Raphan & Cohen 2002; Dai et al 2003

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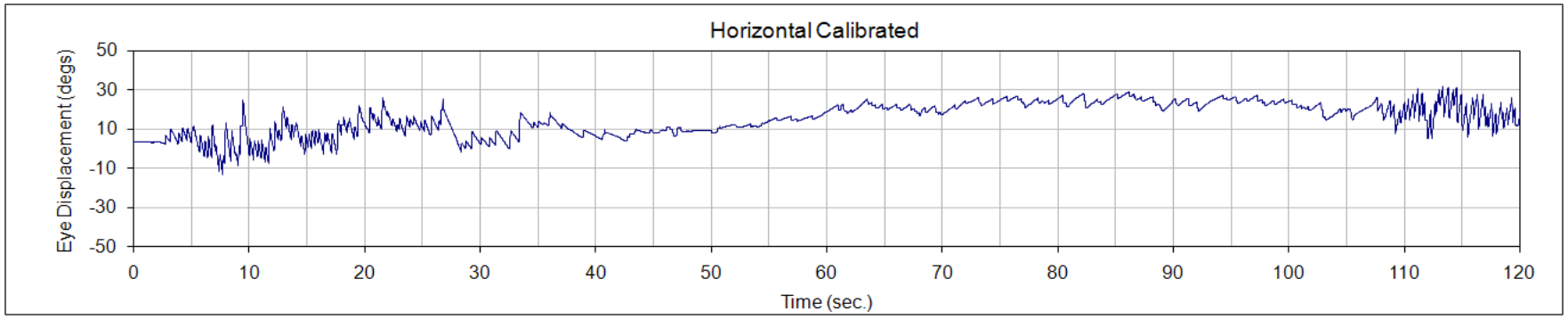
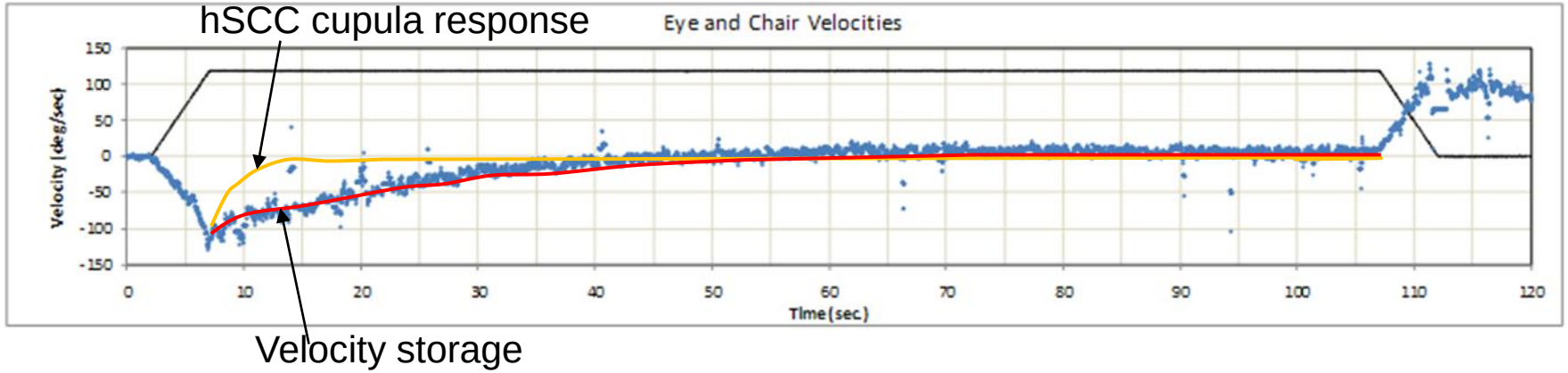
VELOCITY STORAGE





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VELOCITY STORAGE

- Purpose of this response?
  - Vestibular peripheral system is less sensitive to inputs <1 Hz due to limitations of cupular dynamics
  - Central vestibular system enhances poorer low frequency performance
  - Provides a positive feedback loop located within the vestibular nuclei, cerebellum
  - Integrates signals from the vestibular, other systems to *sustain vestibular activity beyond the primary afferent signal*

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CLINICAL PRESENTATION

Chua Wei De, Tze Ling 2020

- High motion sickness, ***prolonged velocity storage***

Jiaodan et al 2022

- ***No significant differences*** noted in migraine vs. controls

Clément, Reschke 2018

- No relationship between VOR gain and motion sickness level
- ***Prolonged velocity storage*** noted for those with more severe motion sickness

DiZio, Lackner 1991

- Parabolic flight
- Pattern noted between ***motion sickness and velocity storage***, suggesting relationship to otolith offloading

Should we expect prolonged time constants with trapezoidal/step testing?

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MOTION SICKNESS SCHEMATIC

Environment

Anxiety,
anticipation,
unpleasant sight,
smells

Motion stimuli

Heavy meal

Can we predict motion sickness from clinical findings?

Unfortunately, motion sickness is not so simple.

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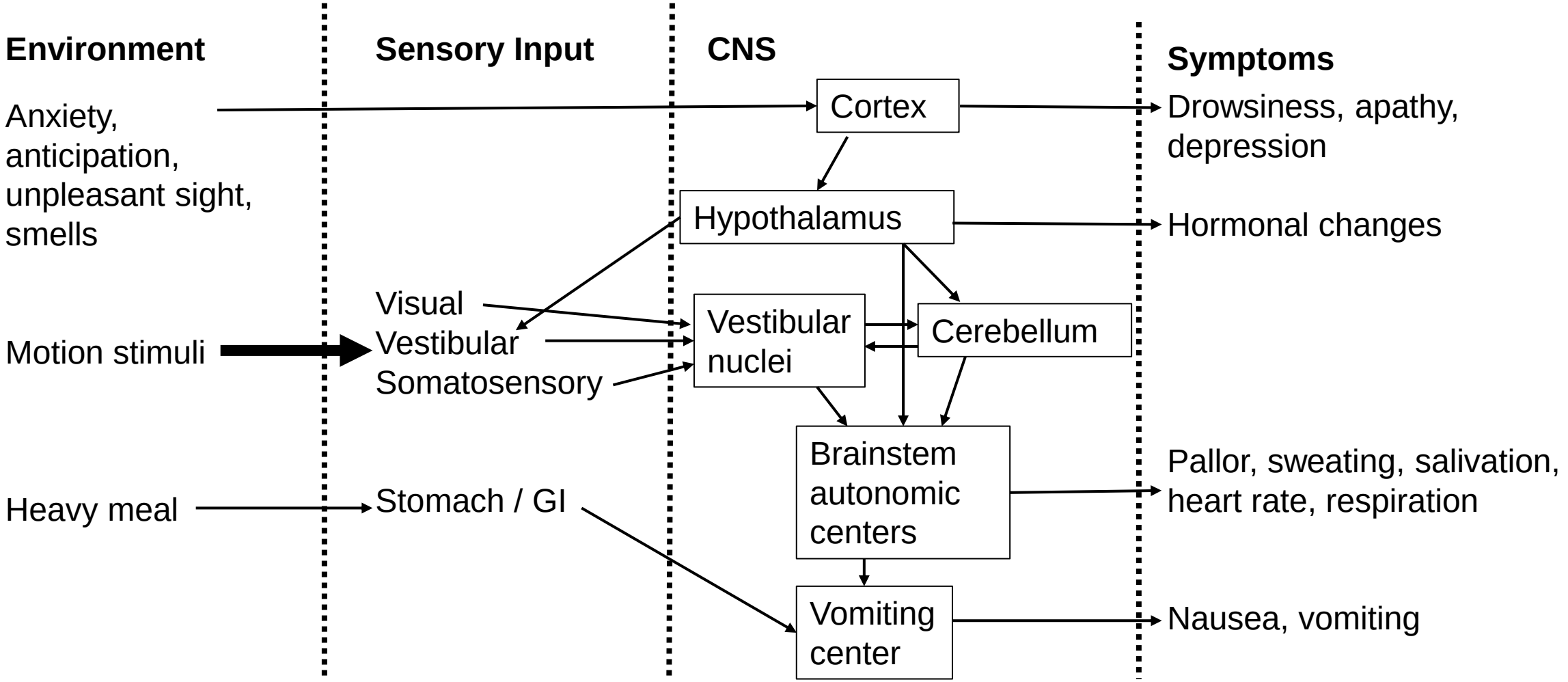
MOTION SICKNESS SCHEMATIC



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DIFFERENTIAL DIAGNOSIS

CLINICAL DIAGNOSIS

Consider Motion Sickness Susceptibility Questionnaire (MSSQ)

Laboratory testing usually not needed

- Differential diagnosis
  - Migraine
  - Pregnancy
  - Concussion
  - Intoxication
  - Hangover
  - Basilar artery occlusion
  - Cerebral vascular accident
  - Vestibulopathy
  - Hypoglycemia
  - Depression/anxiety

Golding 2016

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Motion Sickness Susceptibility Questionnaire Short-form (MSSQ-Short)

1. Please State Your **Age** ..... Years. *2.* Please State Your Sex (tick box) **Male** [ ] **Female** [ ]

This questionnaire is designed to find out how susceptible to motion sickness you are, and what sorts of motion are most effective in causing that sickness. Sickness here means feeling queasy or nauseated or actually vomiting.

Your CHILDHOOD Experience Only (before 12 years of age), for each of the following types of transport or entertainment please indicate:

3. **As a CHILD (before age 12),** how often you **Felt Sick or Nauseated** (tick boxes):

	Not Applicable - Never Travelled	Never Felt Sick	Rarely Felt Sick	Sometimes Felt Sick	Frequently Felt Sick
Cars					
Buses or Coaches					
Trains					
Aircraft					
Small Boats					
Ships, e.g. Channel Ferries					
Swings in playgrounds					
Roundabouts in playgrounds					
Big Dippers, Funfair Rides					
	1	0	1	2	3

Your Experience over the LAST 10 YEARS (approximately), for each of the following types of transport or entertainment please indicate:

4. **Over the LAST 10 YEARS,** how often you **Felt Sick or Nauseated** (tick boxes):

	Not Applicable - Never Travelled	Never Felt Sick	Rarely Felt Sick	Sometimes Felt Sick	Frequently Felt Sick
Cars					
Buses or Coaches					
Trains					
Aircraft					
Small Boats					
Ships, e.g. Channel Ferries					
Swings in playgrounds					
Roundabouts in playgrounds					
Big Dippers, Funfair Rides					
	1	0	1	2	3

Or– Do you / did you get motion sick?

Reason 1968; Golding 1998; Golding 2006

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CLINICAL PRESENTATION

- No significant differences noted in oculomotor performance → perception may be considered
  - Smooth pursuit, optokinetic stimuli
  - Dynamic visual acuity
- Caloric responses may demonstrate higher slow phase velocity, but within the range of expected values
  - No significant difference
  - Increased risk of nausea / vomiting
- No significant abnormalities noted for cVEMP
  - This may not hold for those with additional conditions, including vestibular migraine

Boldingh et al 2011; Buyuklu et al 2009; Fowler et al 2020; Fowler et al 2014; Gedik-Soyuyuce et al 2022; Mallinson & Longridge 2002; Sharon & Hullar 2014; Zaleski et al 2015

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ASSOCIATED CONDITIONS

- Vestibular migraine
- Meniere’s disease
- Other peripheral vestibulopathy

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VESTIBULAR MIGRAINE

- **Estimated 2/3 prone to motion sickness**
- Assumed genetic factors
- Dizziness triggered by visual stimulation suggesting atypical integration visual and vestibular information
- Both conditions demonstrate similar preponderance of females, increased symptoms with menstruation
- Overlapping mechanisms
  - GI symptoms (hypersensitive emetic center)
  - Shared brainstem/cerebellar pathways
  - Vascular mechanisms
  - Serotonin

Abouzari et al 2020; Meng et al 2022; Baloh 1997; Reavley et al 2006; Stern 1996; Drummond 2002; Drummond & Grandston 2004; Golding 2006; Grunfeld & Gresty 1998; Golding et al 2005; Takeda et al 2001; Menetrey & Basbaum 1987; Marcus et al 2005; Cuomo-Granston & Drummond 2010

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MENIERE'S DISEASE

- Higher MSQ scores than controls, but less than vestibular migraine
- May not report pediatric motion sensitivity, only after Meniere's onset
- Association noted between Meniere's disease and history of migraine – is the motion sensitivity related to this?

Sharon & Hullar 2014; Golding & Patel 2017; Pyykko et al 2019

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OTHER PERIPHERAL VESTIBULOPATHY

- No reported relationship between other peripheral vestibulopathy (e.g., BPPV, labyrinthitis) and motion sickness
- Bilateral areflexia? No motion sickness.

Graybiel 1963; Kennedy et al 1968; Cheung et al 1991

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COUNSELING FOR MOTION SICKNESS

HIGH PREVALENCE OF MOTION SICKNESS IN PATIENTS WITH VESTIBULAR SYMPTOMS

HOW CAN WE PREVENT / REDUCE THE EFFECTS?

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PREVENTION AND TREATMENT

Prevention is easier than curing.

- Avoid heavy meals, caffeine, alcohol, foods high in histamine (e.g., cheese, tuna)
- Avoid smoking, stuffy atmosphere
- Travel well-rested, stay hydrated
- Reduce head / body movement
- Reduce visual sensory conflict – avoid reading, screens, wear sunglasses
- Sit in front, maintain visual horizon, drive

Murdin et al 2011; Schutz et al 2014; Takov & Tadi 2019; Koch et al 2018; Bos et al 2006; Griffin & Newman 2004; Brainard &Gresham 2014; Rolnick & Lubow 1991

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ADDITIONAL RECOMMENDATIONS

- Controlled regular breathing activates the parasympathetic nervous system to inhibit the vomiting
- Habituate…
  - 50% can habituate to seasickness
  - >5% cannot habituate
  - Habituation is highly specific to the particular stimulus

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ADDITIONAL RECOMMENDATIONS

- Controlled regular breathing activates the parasympathetic nervous system to inhibit the vomiting
- Habituate…
  - 50% can habituate to seasickness
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PHARMACOTHERAPY

- Most effective prophylactically and when combined with other behavioral modifications
- **Anticholinergics: e.g., scopolamine**
  - Nonselective anticholinergic agent inhibiting input to the vestibular nuclei and vomiting center
  - Typically used as a 1mg transdermal patch behind the ear
  - Apply 4-8 hours before exposure, lasts 72 hours; replace as needed
  - Should not be used in <10 years, elderly, various medical conditions
  - Can be given orally, intramuscular injection, nasal spray
- Antihistamines: e.g., meclizine
- Sympathomimetics (catecholamine activators)

Golding 2016; Russel et al 2014; Yen Pik Sang et al 2003; Murdin et al 2011; Lackner 2014; Yen Pik Sang et al 2005; Schutz et al 2014; Takov & Tadi 2019; Schmal 2013; Tal et al 2013; Brainard & Gresham 2014; Gordon et al 2001; Spinks & Wasiak 2011; Simmons et al 2010; Zhang et al 2016; Nachum et al 2006; Bar et al 2009

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PHARMACOTHERAPY

- Most effective prophylactically and when combined with other behavioral modifications
- Anticholinergics: e.g., scopolamine
- **Antihistamines: e.g., meclizine**
  - Block histamine receptors in vomiting center; anticholinergic properties
  - Take 2 hours before motion
  - More sedating than scopolamine
  - Must be 1st generation antihistamine – 2nd generations are not effective
  - Safe to use during pregnancy (category B)
- Sympathomimetics (catecholamine activators)

Golding 2016; Russel et al 2014; Yen Pik Sang et al 2003; Murdin et al 2011; Lackner 2014; Yen Pik Sang et al 2005; Schutz et al 2014; Takov & Tadi 2019; Schmal 2013; Tal et al 2013; Brainard & Gresham 2014; Gordon et al 2001; Spinks & Wasiak 2011; Simmons et al 2010; Zhang et al 2016; Nachum et al 2006; Bar et al 2009; Estrada et al 2007; Paul et al 2005; Weinstein & Stern 1997; Cowings et al 2000; Nicholson et al 2002; Paule et al 2004; Cheung et al 2003; Kohl et al 1987

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PHARMACOTHERAPY

- Most effective prophylactically and when combined with other behavioral modifications

- Anticholinergics: e.g., scopolamine
- Antihistamines: e.g., meclizine
- **Sympathomimetics (catecholamine activators)**
  - Ephedrine, dextroamphetamine
  - Not superior to scopolamine, antihistamines
  - Used in combination to overcome drowsiness

Golding 2016; Russel et al 2014; Yen Pik Sang et al 2003; Murdin et al 2011; Lackner 2014; Yen Pik Sang et al 2005; Schutz et al 2014; Takov & Tadi 2019; Schmal 2013; Tal et al 2013; Brainard & Gresham 2014; Gordon et al 2001; Spinks & Wasiak 2011; Simmons et al 2010; Zhang et al 2016; Nachum et al 2006; Bar et al 2009; Estrada et al 2007; Paul et al 2005; Weinstein & Stern 1997; Cowings et al 2000; Nicholson et al 2002; Paule et al 2004; Cheung et al 2003; Kohl et al 1987; Buckey et al 2007; Weerts et al 2014; Murray 1997

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SUMMARY

- Motion sickness is a common condition in the general population and more so in certain vestibular system disorders.
- The motion sickness cascade **requires** peripheral vestibular function.
- Once the vestibulo-autonomic cascade is triggered, motion sickness will continue until the offending stimulus is stopped.

Currently there is no better cure than prevention.

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Prevention Recommendations

- Dizzy patients often experience motion sickness: vestibular migraine, other peripheral vestibulopathies
- Provide non-pharmacologic recommendations and information regarding management strategies.
- Encourage understanding of environmental triggers
  - Others to consider? Virtual reality, video games, autonomous cars, high speed trains, commercial spaceflight…

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Thanks!

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