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# Motion Sickness: Pathophysiology & Clinical Presentation

Jamie M. Bogle, AuD, PhD

# Jamie Bogle, AuD, PhD

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# Disclosures

- **Presenter Disclosure**: Financial: Jamie Bogle is employed by Mayo Clinic. Non-financial: Jamie Bogle is non-salaried faculty at the University of Colorado at Boulder, Gallaudet University, Salus University, Missouri State University. She is the American Academy of Audiology – ARC Conference Committee Chair; Co-Vice Chair, Guidelines & Strategic Documents and the Associate Editor for the American Journal of Audiology
  - In lieu of honorarium, a donation has been made on behalf of the presenter to the American Academy of Audiology Research Grants in Hearing and Balance.
- **Content Disclosure**: This learning event does not focus exclusively on any specific product or service.
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- As healthcare is rapidly evolving, new findings may change the validity of the information presented in this course. Providers should always consult the latest research and guidelines from professional associations.
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- Healthcare providers should apply cultural competence and sensitivity to ensure effective and respectful care based on their patients' diverse needs.
- Providers should always evaluate the limitations of diagnostic and screening tools, considering their potential for false results as well as any ethical implications.
- It is the responsibility of course participants to critically evaluate the evidence from multiple sources to guide professional practice.

# Learning Outcomes

After this course, participants will be able to:

- 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.

# What is motion sickness?

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

**Symptoms**

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



Takov & Tadi 2023; Hromatka et al 2015

# What is the most common motion sickness trigger?

# Motion Sickness

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

# Motion Sickness

**Seasickness**

- 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%

Carsickness

Airsickness

Centrifugal motion sickness

Dizziness due to spinning

Virtual reality

Space motion sickness

Koch et al 2018

# Motion Sickness

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

- Up to 46%
- Increased prevalence in high velocity situations (e.g., rally cars), sitting in the back seat, when reading

Koch et al 2018; Golding & Gresty 2015; Schmidt et al 2020

# 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

# Motion Sickness

Seasickness

Carsickness

Airsickness

Centrifugal motion sickness

Dizziness due to spinning

**Virtual reality**

Space motion sickness

- Cybersickness – up to 73% experience increased motion sickness
- Video games
- Automated cars

Curry et al 2020; Laessoe et al 2023; Salter et al 2019

# How common is motion sickness?

- *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 2023; Herron 2010

# How common is motion sickness?

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



Besnard et al 2021; Peddareddygari et al 2019; Takov & Tadi 2023; Zhang & Sun 2021; 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

# 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%
  - 35 single-nucleotide polymorphisms associated with motion sickness
  - Genes mapped to chromosome 4



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

# 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



Zhang & Sun 2020; 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

# How common is motion sickness?

- 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



Wurthmann et al 2021; Takov & Tadi 2023; 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

# 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

# MOTION SICKNESS

Takov & Tadi 2023; Brainard & Gresham 2014

# MOTION SICKNESS

Onset

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

Takov & Tadi 2023; Brainard & Gresham 2014

# MOTION SICKNESS

## Onset

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

## Early

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

Takov & Tadi 2023; Brainard & Gresham 2014

# 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 2023; Brainard & Gresham 2014

...

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

Takov & Tadi 2023; Brainard & Gresham 2014

...

- 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

Takov & Tadi 2023; Brainard & Gresham 2014

...

- 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

And then …

- Retching / vomiting

Symptoms will not resolve until offending stimulus stops

- Symptoms resolve completely within 24 hours

# Post-Motion

***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 & Mead 1998

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Golding 2016; Shankar Kikkeri & Siddiqui 2018

# Motion sickness = unpleasant

Why does this mechanism exist?

# 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**

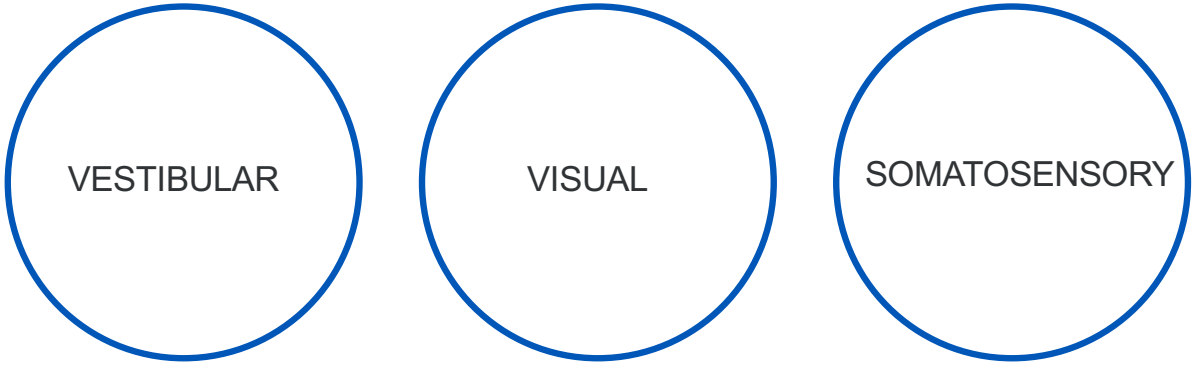
Vestibular stimulation of extra-ocular muscles leads to stimulation of vagus nerve



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

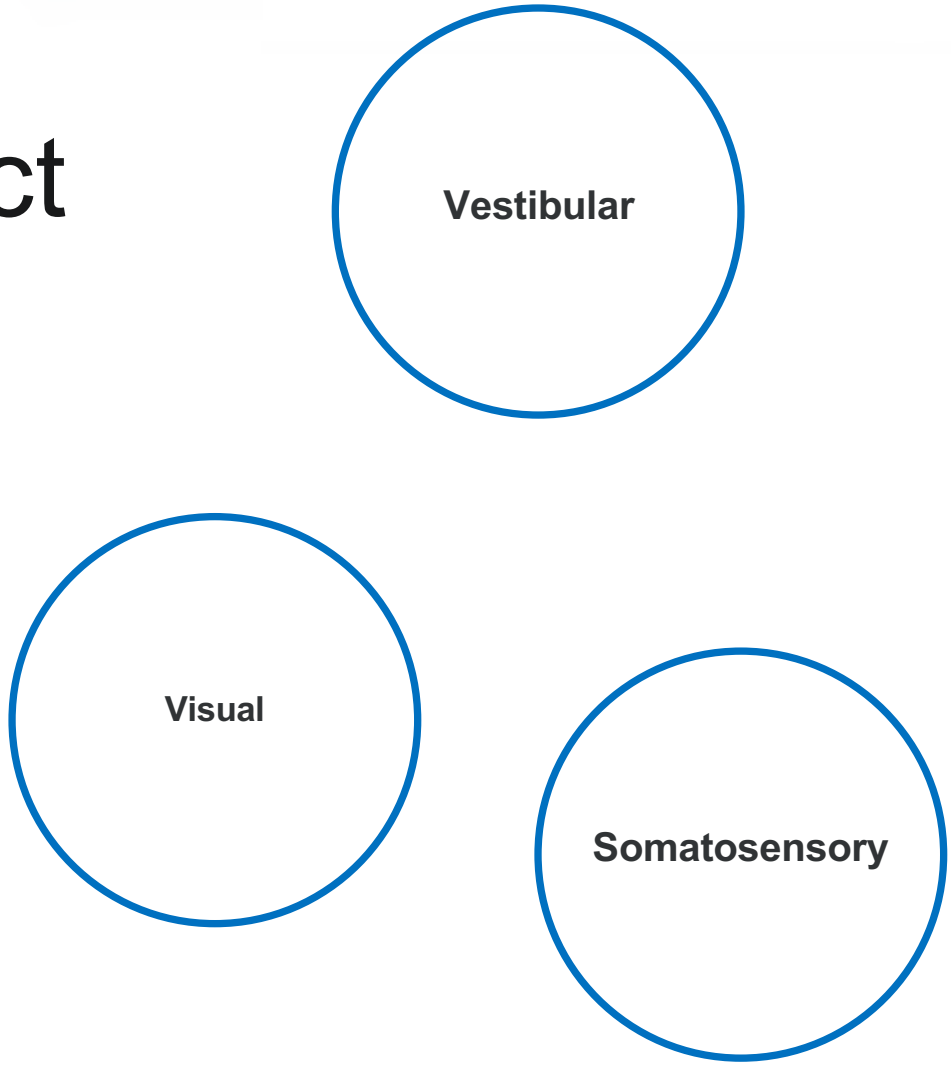
# Sensory Conflict

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



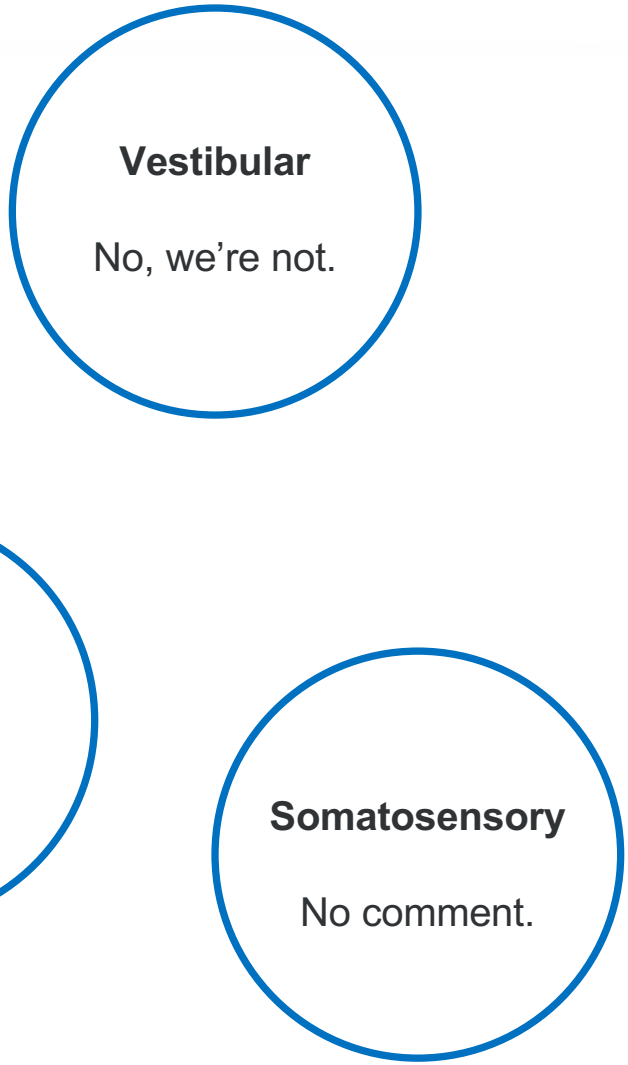
# Sensory Conflict

- Why does this happen?



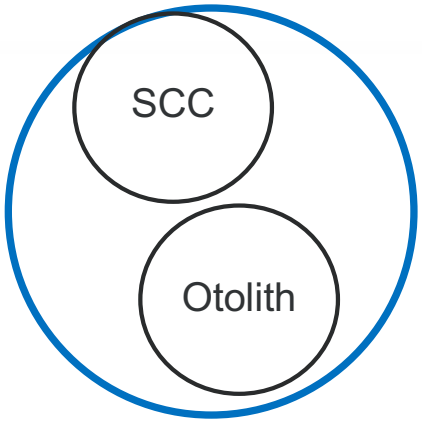
# Sensory Conflict

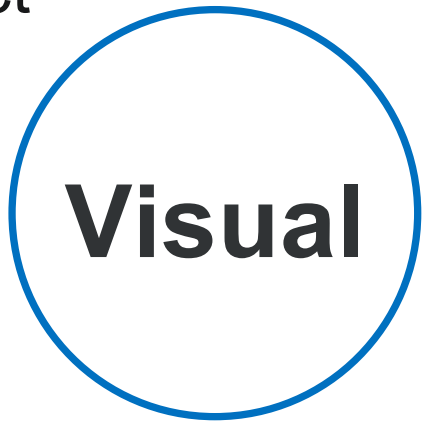
- Why does this happen?
- Sensory information is different than expected based on past experience
- Inter-modality conflict – typically vision versus vestibular

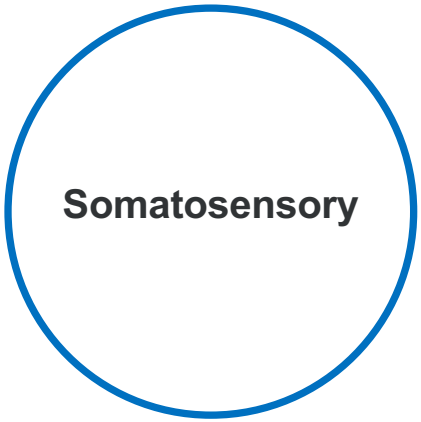


# Sensory Conflict

- Why does this happen?
- Intra-labyrinthine conflict

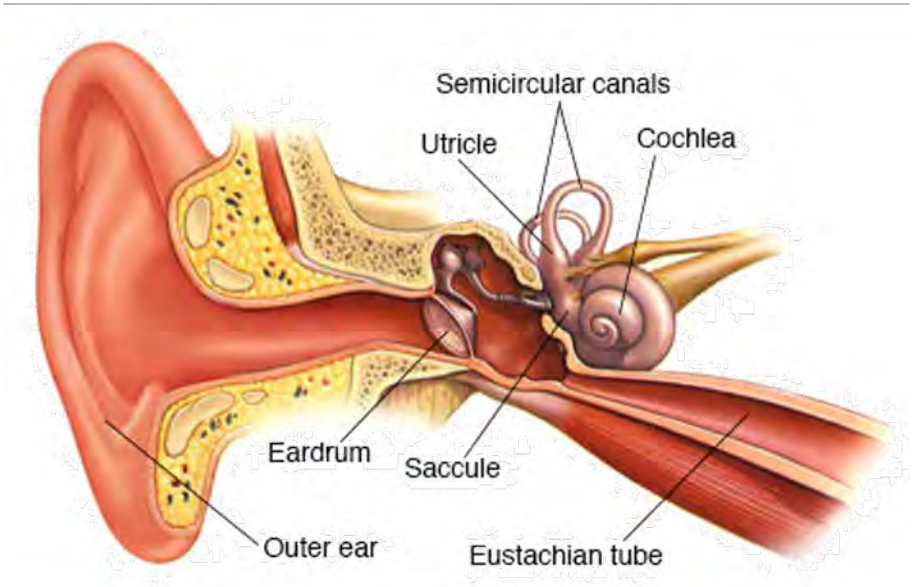


# Intra-labyrinthine Conflict

- **Hypothesis:** *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.



# Intra-labyrinthine Conflict

- Control / migraine subjects demonstrate evidence of canal-otolith resolution
- Vestibular migraine subjects did not.
  - Unable to accurately identify earth vertical axis
  - Unable to suppress velocity storage

- **Results suggest that increased motion sickness may relate to inappropriate SCC / otolith integration**

Wang & Lewis 2016

# 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 2016; Angelaki & Hess 1994; Angelaki & Hess 1995; Kitama et al 2014; Cohen et al 1992

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

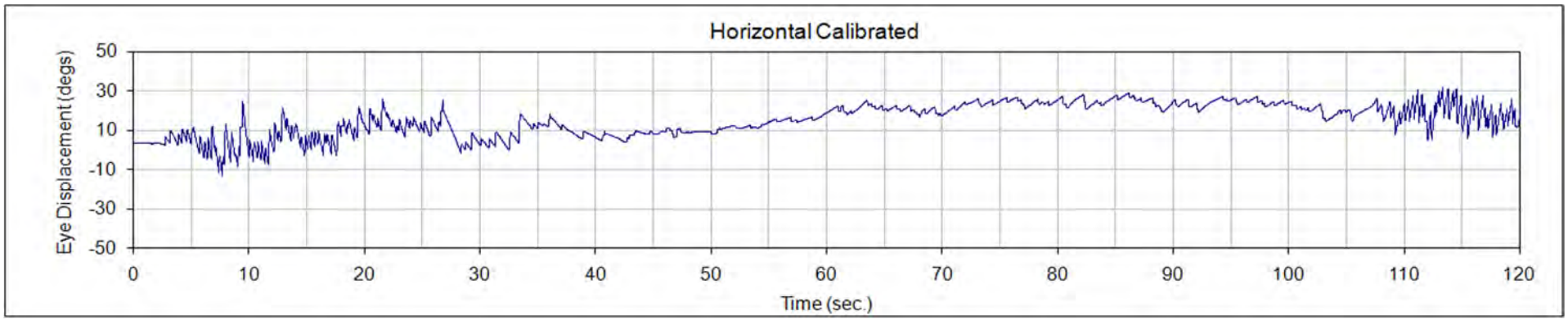
Bles 1998; Cohen 2004; Bertolini & Straumann 2016; Cohen 2011; Donohew & Griffin 2004; Joseph & Griffin 2008; Dai et al 2010

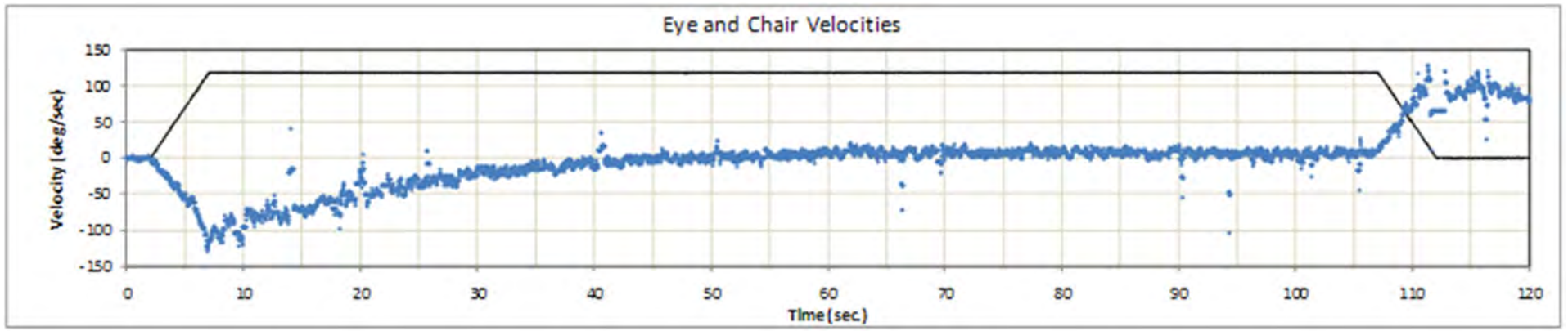
# Vestibular System Role

- Peripheral vestibular system is **essential** to producing motion sickness.
- Vestibulo-autonomic symptoms triggered from the **velocity storage integrator** in the vestibular nuclei and nodulus (vestibulo-cerebellum).
- Velocity storage
  - Nystagmus / perception after rotation ends (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.

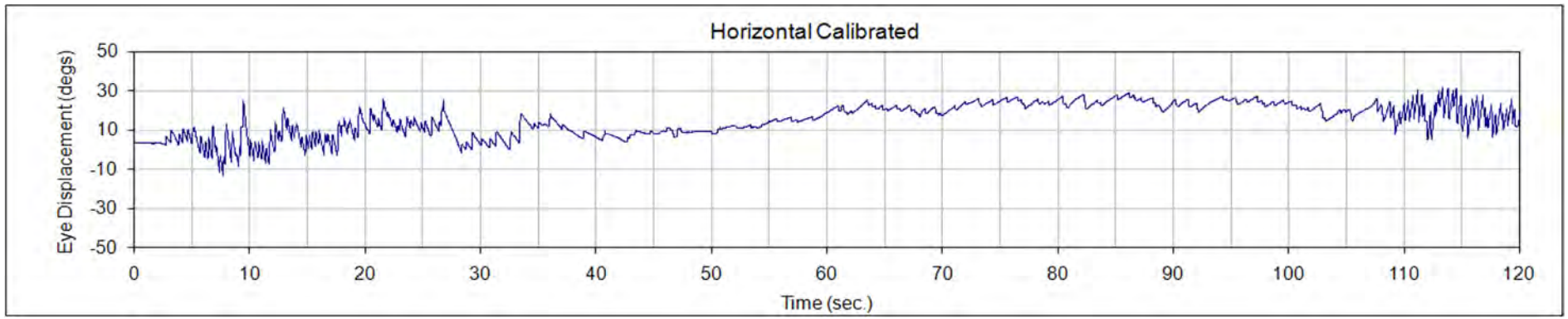
Tyler & Bard 1949; Bard & Woolsey 1947; Mach 1875; Robinson 1977; Raphan et al 1981; Raphan & Cohen 1985; Raphan & Cohen 2002; Dai et al 2003

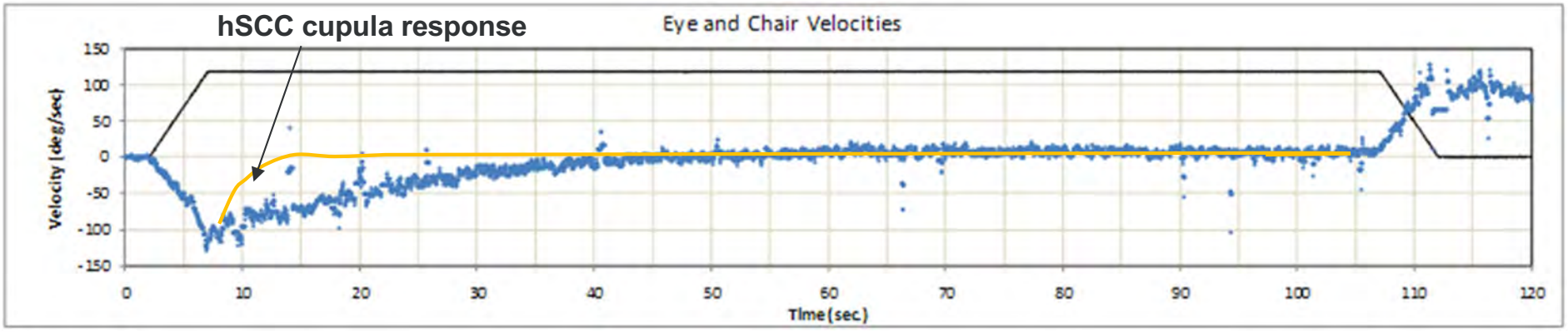
# Velocity Storage



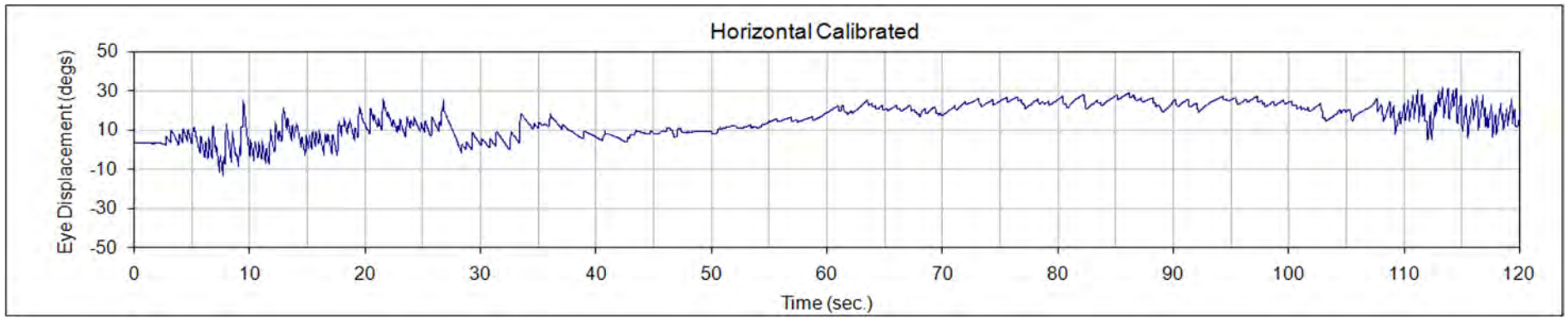



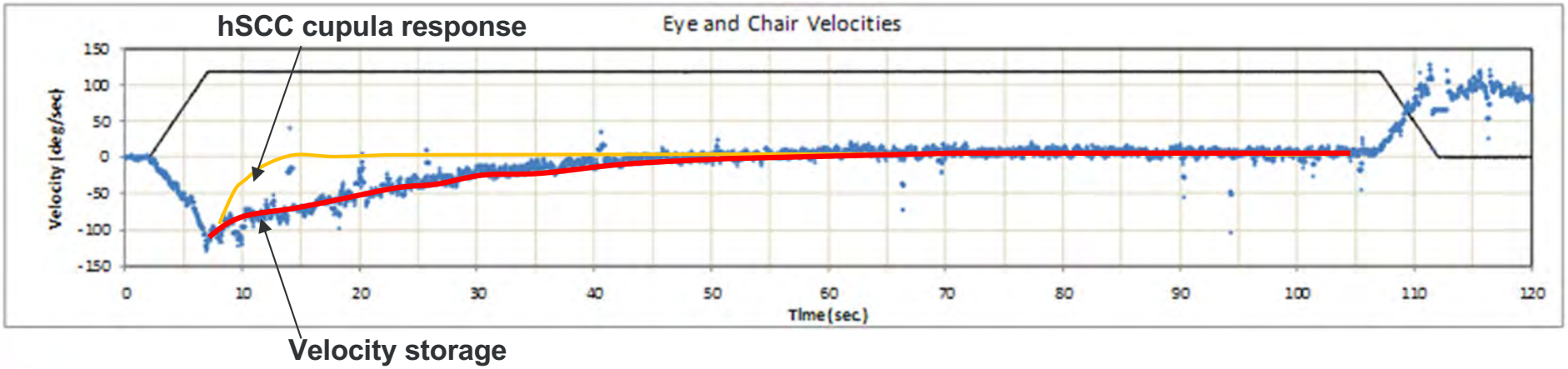
# Velocity Storage



# Velocity Storage



# 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 and other systems to sustain vestibular activity beyond the primary afferent signal

# Clinical Presentation

- ***Prolonged velocity storage*** noted in severe motion sickness (Chua & Kek 2020; Clément & Reschke 2018)
- No relationship between VOR gain and motion sickness severity (Clément & Reschke 2018)
- Parabolic flight
- Pattern noted between ***motion sickness and velocity storage***, suggesting relationship to otolith offloading (DiZio & Lackner 1991)

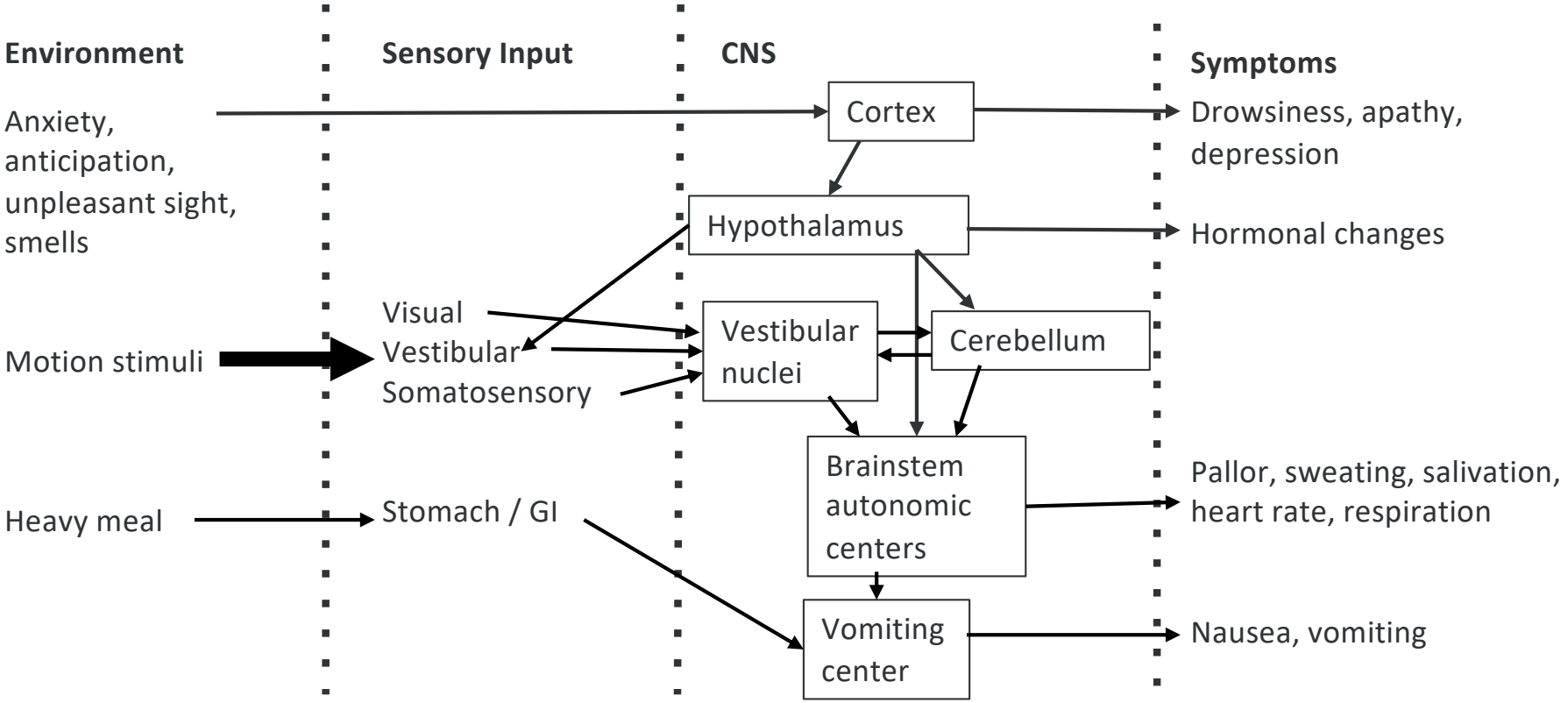
Should we expect prolonged time constants with trapezoidal/step testing?

# Motion Sickness Schematic

Can we predict motion sickness from clinical findings?

Probably not.

# Motion Sickness Schematic



# 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

| | Not applicable – never travelled (0) | Never felt sick (0) | Rarely felt sick (1) | Sometimes felt sick (2) | Frequently felt sick (3) |
|---|---|---|---|---|---|
| Your CHILDHOOD Experience Only (before 12 years of age), for each of the following types of transport or entertainment please indicate: | | | | | |
| 1. As a CHILD (before age 12), how often you **Felt Sick or Nauseated** (tick boxes): | | | | | |
| Cars | | | | | |
| Buses or coaches | | | | | |
| Trains | | | | | |
| Aircraft | | | | | |
| Small boats | | | | | |
| Ships, e.g. channel ferries | | | | | |
| Swings in playgrounds | | | | | |
| Roundabouts in playgrounds | | | | | |
| Big Dippers, Funfair rides | | | | | |
| | | | | | |
| Your experience over the LAST 10 YEARS (approximately), for each of the following types of transport or entertainment please indic | | | | | |
| 2. Over the LAST 10 YEARS, how often you **Felt Sick or Nauseated** (tick boxes): | | | | | |
| Cars | | | | | |
| Buses or coaches | | | | | |
| Trains | | | | | |
| Aircraft | | | | | |
| Small boats | | | | | |
| Ships, e.g. channel ferries | | | | | |
| Swings in playgrounds | | | | | |
| Roundabouts in playgrounds | | | | | |
| Big Dippers, Funfair rides | | | | | |
| | | | | | |

Or– Do you / did you get motion sick?

“Can you read while travelling in a car without getting motion sick?”

# Clinical Presentation

- No significant differences noted in oculomotor performance → perception may be considered
  - Smooth pursuit, optokinetic stimuli
  - Dynamic visual acuity may/may not be abnormal
- Caloric responses *may* demonstrate higher slow phase velocity, but within the range of expected values
  - Overall, 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

# Associated Conditions

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

# 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

# Meniere Disease

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

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

# 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

# Counseling for Motion Sickness

- High prevalence of motion sickness in patients with vestibular symptoms

- How can we prevent / reduce the effects?

# 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

# 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 a particular stimulus

# Additional Recommendations

- Most effective prophylactically and when combined with other behavioral modifications
- **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)

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

# Additional Recommendations

- 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

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

# 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

# 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.**

# Thanks!

bogle.jamie@mayo.edu

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