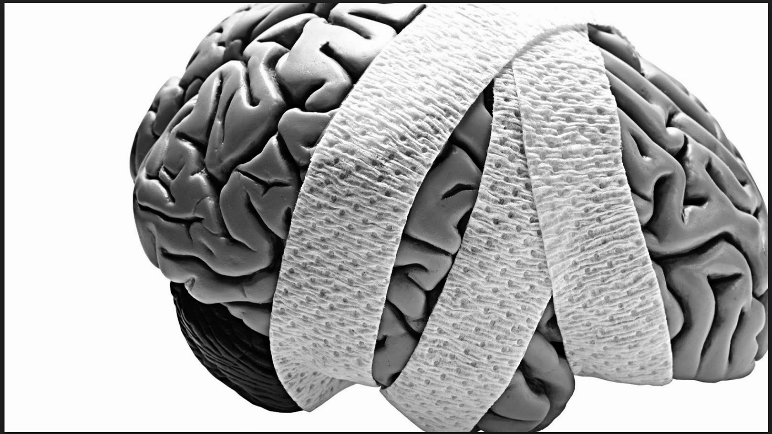

Traumatic Brain Injury:
Vestibular Testing, Findings, and Cognition

Liz Fuemmeler, AuD and Daniel Romero, AuD, PhD

A little about us…

Liz Fuemmeler, AuD

Clinical Product Manager at Interacoustics

Disclosures:

- Employee at Interacoustics
- AAA Volunteer
- Missouri Academy of Audiology President-Elect
- Co-Host of a Dose of Dizzy

a dose of dizzy

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Assistant Professor at Vanderbilt University Medical Center

Disclosures:

- Employee at Vanderbilt University Medical Center
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Today's Timeline and Agenda

1. **What is a TBI?**
   Definitions, impacts, and audiologic considerations

2. **Acute Phase**
   The early stages of TBI - symptoms and test findings

3. **Chronic Phase**
   Long term impacts, including effects on cognition

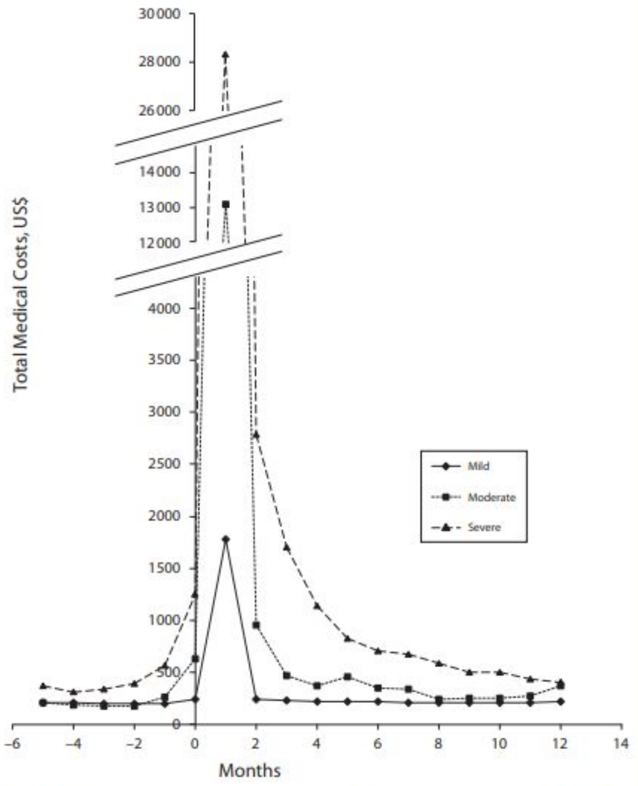
4. **Case Studies**

Traumatic Brain Injury

“A **concussion** is a type of traumatic brain injury—or TBI—caused by a bump, blow, or jolt to the head or by a hit to the body that causes the head and brain to move rapidly back and forth.” CDC, 2019.

Prevalence of TBIs

- 42 million people are diagnosed with concussion worldwide every year (NIH, 2020)
- 223,135 hospitalizations related to TBIs in 2019 (CDC)
    - Out of 330 million Americans
- People aged 75 years and older have the highest rates of TBI-related hospitalizations (32%) and deaths (28%)



Note. Line graph shows mean costs for pediatric patients with mild, moderate, and severe traumatic brain injury (TBI) for 6 months before and 12 months after injury. The sample size was n = 319 103.

FIGURE 1—Unadjusted monthly total health care costs for patients diagnosed with traumatic brain injury: Sample of Patients, United States, January 1, 2007–December 31, 2010.

Graves et al, 2015 (AJPH)

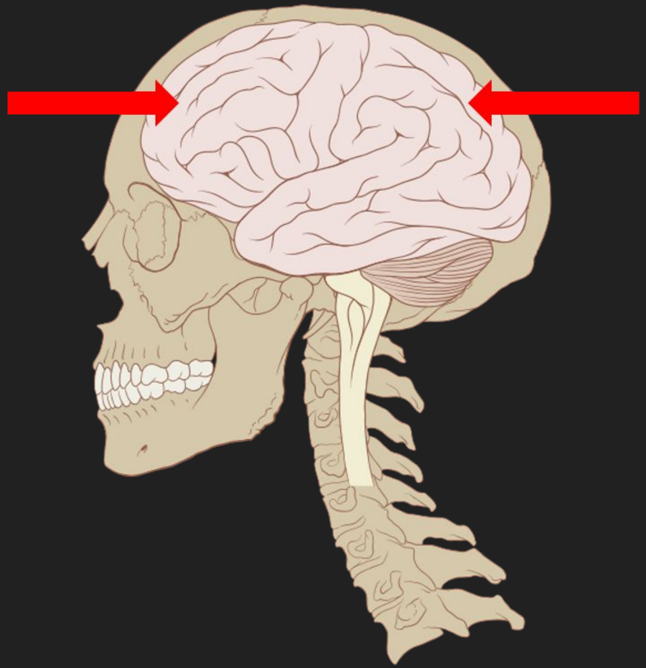
Common Causes of TBIs

- Motor vehicle accidents
- Sports
- Falls
- Military/combat
- Assault

pic : Huffpost

Types of Injuries

Deceleration Injury

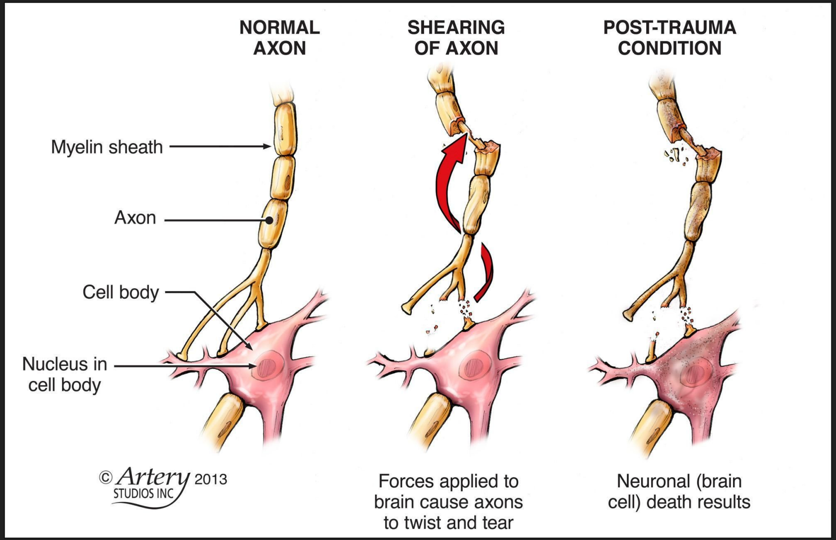


Rotational Injury

pic : sites.nd.edu

Pathophysiology of Concussion

- Biomechanically induced brain lesion in the absence of gross anatomic lesions
- Functional dysfunction, not structural abnormalities
- Neurometabolic dysfunction: Metabolic chemical changes that take place in the brain



True or false?

A direct head impact is necessary for diagnosis of concussion.

A direct head impact is necessary for diagnosis of concussion.

FALSE

It is the acceleration, deceleration or rotation of the brain inside the skull that causes injury to the brain's function. This movement can happen secondary to direct or indirect head impact.

A concussion is technically classified as a mild traumatic brain injury.

A concussion is technically classified as a mild traumatic brain injury.

TRUE

A concussion is a type of injury to the brain, usually mild in nature due to symptom presentation and recovery prognosis.

Loss of consciousness is required for a true diagnosis of concussion.

Loss of consciousness is required for a true diagnosis of concussion.

FALSE

More than 90% of concussions do not involve loss of consciousness.

CDC 2006

Common Concussion Symptoms

- Dizziness
- Imbalance
- Disorientation
- Blurred vision
- Tinnitus
- Noise sensitivity

TABLE 2

Select Signs and Symptoms of Concussion

Affective/emotional	Sleep
Anxiety/nervousness*†	Decreased sleep†
Clinginess	Difficuty initiating sleep†
Depression†	Drowsiness*†
Emotional lability	Increased sleep*†
Irritability*†	**Somatic/physical**
Personality changes	Blurred vision*
Sadness	Convulsions
Cognitive	Dizziness/poor balance*‡
Amnesia	Fatigue*†
Confusion‡	Headache*‡
Delayed physical reactions	Light-headedness†
Delayed verbal responses	Light sensitivity*†
Difficulty concentrating*†	Nausea*‡
Difficulty remembering*†	Noise sensitivity*
Disorientation*†	Numbness/tingling
Feeling in a fog*	Tinnitus†
Feeling slowed down*	Vomiting‡
Feeling stunned	
Inability to focus	
Loss of consciousness	
Slurred speech	
Vacant stare	

*—Common to most self-report symptom checklists.
†—Defined by the American Academy of Neurology as late symptoms lasting days to weeks.
‡—Defined by the American Academy of Neurology as early symptoms lasting minutes to hours.

Adapted with permission from Scorza KA, Raleigh MF, O'Connor FG. Current concepts in concussion: evaluation and management. Am Fam Physician. *2012;85(2):125.*

Challenges in Concussion Diagnosis

TABLE 1

Definition of Concussion from the Fifth International Conference on Concussion in Sport

Sports-related concussion is a traumatic brain injury induced by biomechanical forces.

Several common features help to clinically define the nature of a sports-related concussion:

- Direct blow to the head, face, neck, or elsewhere on the body with an impulsive force transmitted to the head
- Rapid onset of short-lived impairment of neurologic function that resolves spontaneously; however, signs and symptoms sometimes evolve over minutes to hours
- Neuropathologic changes, but the acute clinical symptoms largely reflect a functional disturbance rather than a structural injury; no abnormality is visible on standard structural neuroimaging studies
- Range of clinical signs and symptoms that may involve loss of consciousness; resolution of the clinical and cognitive features typically follows a sequential course; however, in some cases, symptoms may be prolonged

The clinical signs and symptoms cannot be explained by substance use (i.e., alcohol, illicit drugs, medications), other injuries (e.g., cervical injuries, peripheral vestibular dysfunction), or other comorbidities (e.g., psychological factors, coexisting medical conditions)

Information from reference 6.

There is no **one** objective test to determine whether a concussion has occurred.

1. Multi-disciplinary approach
2. Utilizing multiple types of tools and evaluations

No two concussions are the same.

1. Injury mechanism (cause, direct vs. indirect, etc.)
2. Symptom profile / clinical presentation
3. Pre-existing medical conditions
4. Prolonged recovery risk factors (previous concussions, hx of migraines, etc.)

Team-Based Evaluation of a Concussion

- Neurocognitive evaluation
  - ImPACT, King Devick, SCAT5, etc.

- Neurologic evaluation
  - Symptom pathology, reflexes, balance, oculomotor, etc.

- Imaging
  - MRI/CT scan

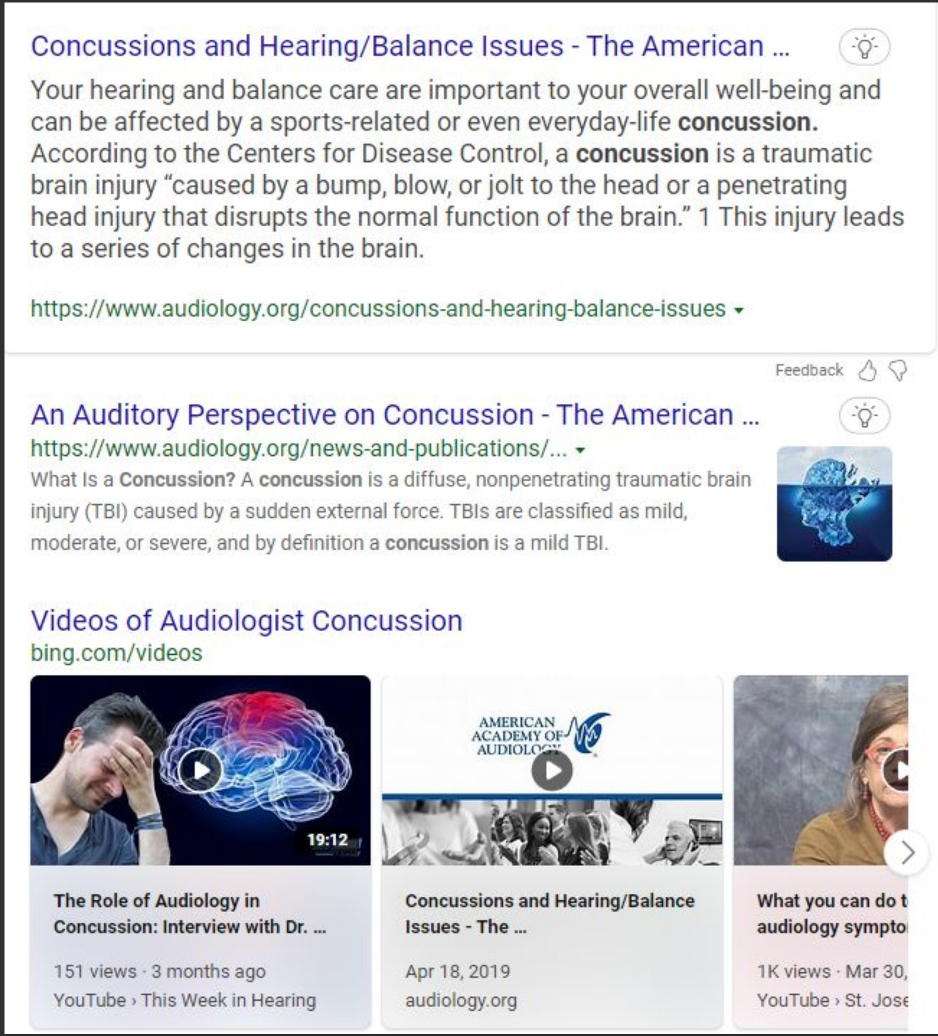
- Symptom Evaluation
  - Written, computerized, or verbal

Challenges with a Symptoms-Based Diagnosis

- **Diagnosis**
  - Patient may have motivation to over- or under-exaggerate symptoms
    - Athlete wanting to/avoiding return to play
      - Baugh, Kierman, Kroshus et al., 2014; Delaney et al., 2002; Delaney et al., 2015; Kaut et al., 2003; Kroshus, Kubzansky, Goldman & Austin, 2014; Llewellyn et al., 2014; McCrea et al., 2004; Meehan et al., 2013; Register-Mihalik et al., 2012; Torres et al., 2014; Williamson et al., 2005
    - Student wanting to/avoiding return to school
    - Employee wanting to/avoiding return to work
- **Recovery Process**
  - Are they making adequate progress? Is the intervention type appropriate? Are there other factors that need to be address to speed up recovery?
- **Discharge**
  - Patient may be returned back to field/work too early
  - Risk for second impact
  - Patient may prolong return back to field or work
  - Misuse of medical system, insurance, work comp, etc.

Acute Phase

An audiologist's role in vestibular evaluation post head injury



Concussions and Hearing/Balance Issues - The American ...

Your hearing and balance care are important to your overall well-being and can be affected by a sports-related or even everyday-life **concussion.** According to the Centers for Disease Control, a **concussion** is a traumatic brain injury "caused by a bump, blow, or jolt to the head or a penetrating head injury that disrupts the normal function of the brain." 1 This injury leads to a series of changes in the brain.

https://www.audiology.org/concussions-and-hearing-balance-issues

Feedback

An Auditory Perspective on Concussion - The American ...

https://www.audiology.org/news-and-publications/...

What Is a **Concussion?** A **concussion** is a diffuse, nonpenetrating traumatic brain injury (TBI) caused by a sudden external force. TBIs are classified as mild, moderate, or severe, and by definition a **concussion** is a mild TBI.

Videos of Audiologist Concussion

bing.com/videos

19:12

The Role of Audiology in Concussion: Interview with Dr. ...

151 views · 3 months ago

YouTube › This Week in Hearing

AMERICAN ACADEMY OF AUDIOLOGY

Concussions and Hearing/Balance Issues - The ...

Apr 18, 2019

audiology.org

What you can do t audiology sympto

1K views · Mar 30,

YouTube › St. Jose

Vestibular Assessment Goals Post-Concussion

Initial Appointment:

1) Rule in / out peripheral auditory conditions
2) Schedule for central auditory disorders, if applicable
3) Rule in / out peripheral vestibular conditions
4) Evaluate vestibular and ocular reflex pathways for disruption

Follow up:

5) Schedule follow-up assessment
6) Refer to other providers (neurology, optometry, psychiatry, physical or speech therapy), as needed

Modifications for “Typical Vestibular” Assessment

THINK, LISTEN, and BE CREATIVE

- Questionnaires and Case History
- Auditory Assessment:
  - Bone at 250 Hz
  - Tympanometry
  - Acoustic reflexes
  - Tinnitus evaluation
- Vestibular Assessment
  - Rotational studies
  - Optokinetics
  - Calorics
  - VEMPs

Concussion-Specific Vestibular Assessment

Goals:

- Identify abnormalities to guide recommendations for therapy/intervention types
- Establish baseline to measure and track recovery
- Rule in and out otologic involvement

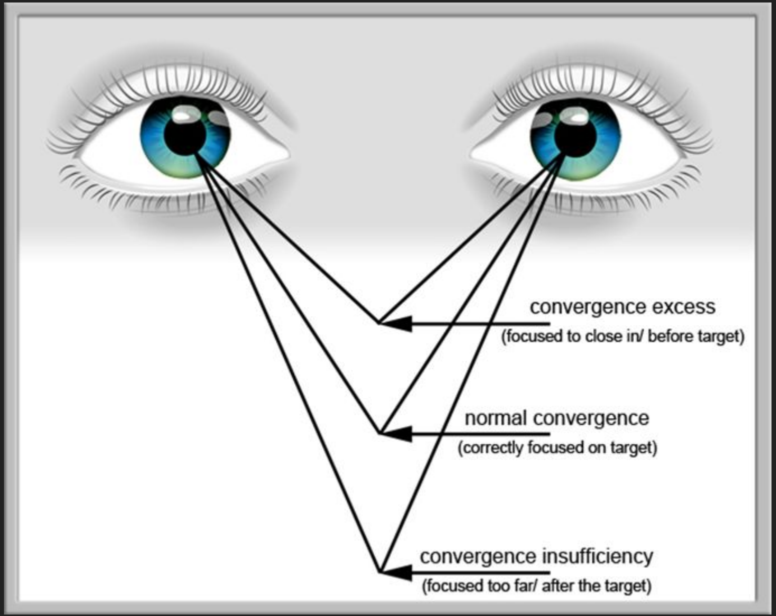
Oculomotor Evaluation

Standardized stimulus, video goggles, compare to age-based norms

- **Convergence**
- Gaze
- Pursuit
  - Horizontal
  - **Vertical**
  - **Smooth Pursuit Neck Torsion**
- Saccades
- **Anti-saccades**
- Optokinetics

Near Point Convergence

- Ability of the eyes to focus on an object close to the face without doubling of the target
- Can be low tech
- ~5% of individuals have congenital CI
  - (2.25-8.3% - Letourneau et al, 1988; Porcar et al, 1997; Rouse et al, 1999)



Neuro-vision development center

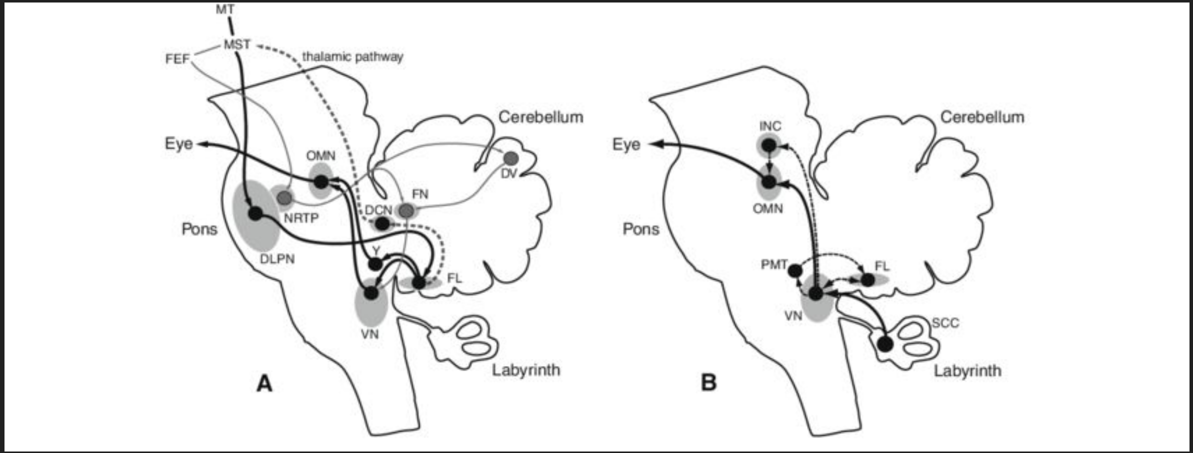
Near Point Convergence

Post Head Injury:

- Military: 28-42%
  - Goodrich et al, 2013; Brahm et al, 2009; Stelmack et al, 2009; Capo-Aponte et al, 2012
- Sport: 42-45%
  - Mucha et al, 2014; Pearce et al, 2015
- Adult: 23-42%
  - Suchoff et al, 1999; Ciuffreda et al, 2007; Alvarez et al, 2012
- Children: 49%
  - Master et al, 2016

Vertical Pursuit

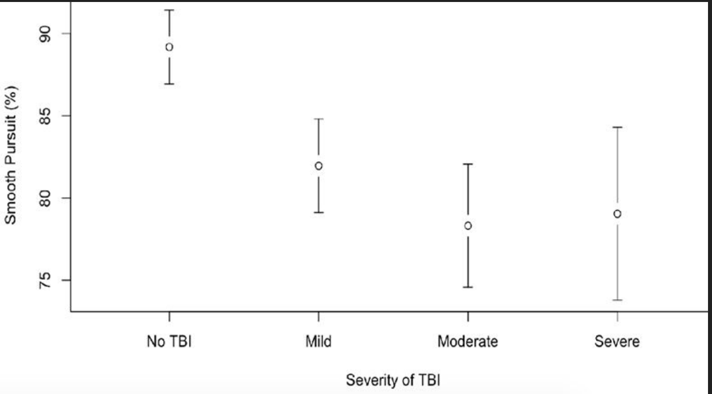
- Vertical smooth pursuit requires the patient to smoothly follow the target up and down at different frequencies.
- Not frequently tested as a part of the VNG test battery:
  - Increased artifact with eyelid closure
  - No normative data



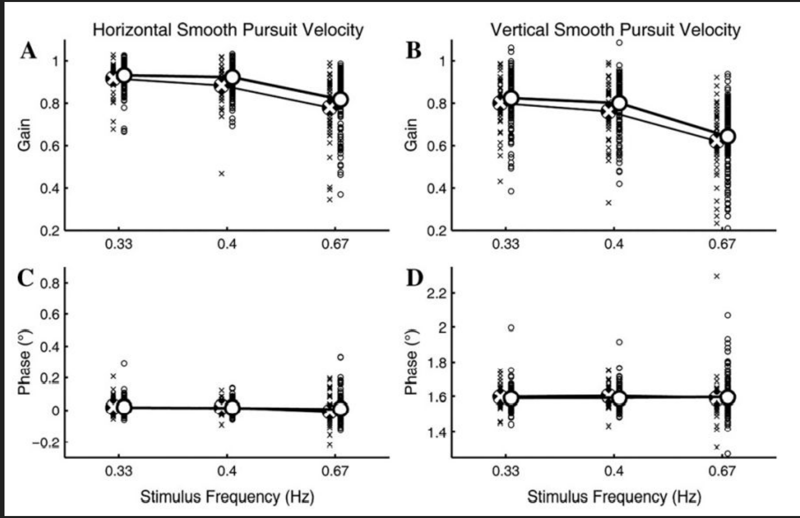
Marti et al, 2008

Vertical Pursuit

Post-Head Injury



Hunfalvay et al, 2020



Maruta et al, 2017

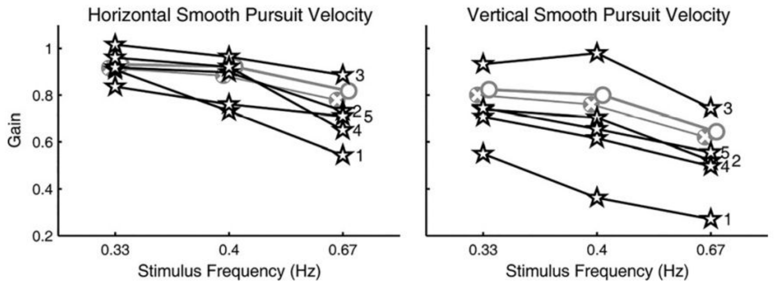


FIGURE 4. Smooth pursuit velocity frequency response of accurately concussed patients. Stars connected with lines indicate individual patients. The means of control subjects (circles) and patients (crosses) with persistent postconcussion symptoms are shown in gray.

Smooth Pursuit Neck Torsion Test

- Comparison of smooth pursuit gain in neck neutral versus neck torsion positions
  - Gain
  - $SPNT_{diff}$

Gain neutral − (Gain torsion L + Gain torsion R)/2

- SPNT test has been shown to be reliable tool to evaluate cervical and whiplash disorders

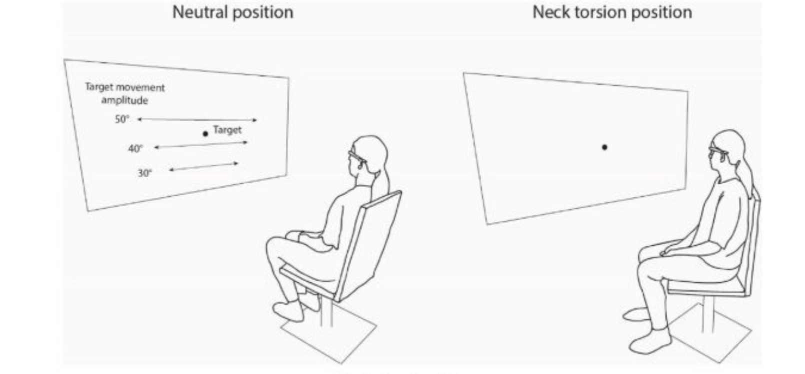


Fig. 1. Experimental setup.

Rosker et al, 2022

Smooth Pursuit Neck Torsion

Post-Head Injury

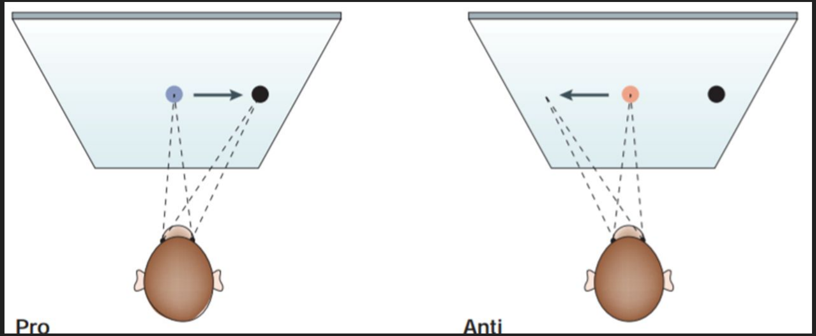
- Increase in saccadic instructions
- Reduced gain in neck torsion positions (measured by the $SPNT_{diff}$)
  - Tjell et al, 2002

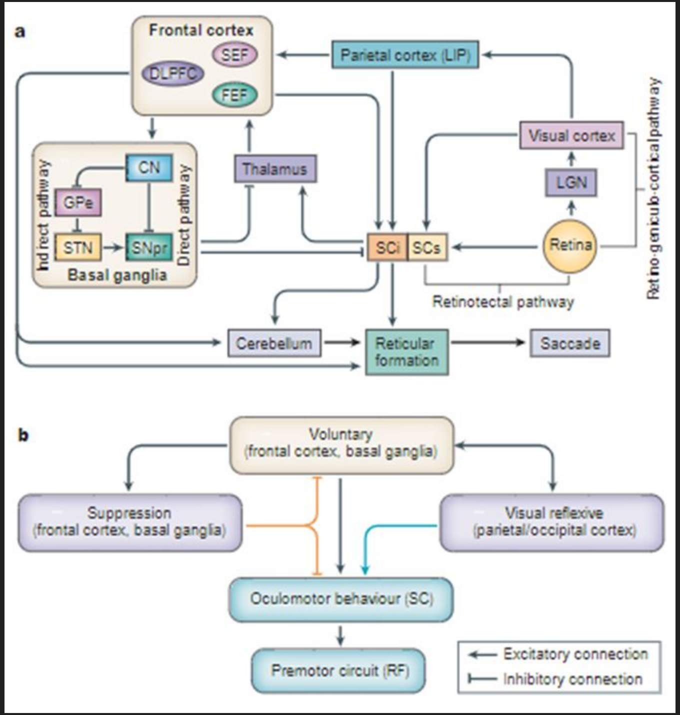
Clinical Studies using $SPNT_{diff}$

- Whiplash disorder patients - 92% specificity and 72% sensitivity
  - Tjell et al, 2002
- Idiopathic neck pain patients - 70.8% specificity and 50% sensitivity
  - Rosker et al, 2022
- No significant difference between chronic neck patients and healthy controls
  - Janssen et al, 2015

Saccadometry Testing

- Advanced oculomotor test composed of pro-saccades and anti-saccade tasks
- Measurement parameters: latency, velocity, accuracy, directional error rate



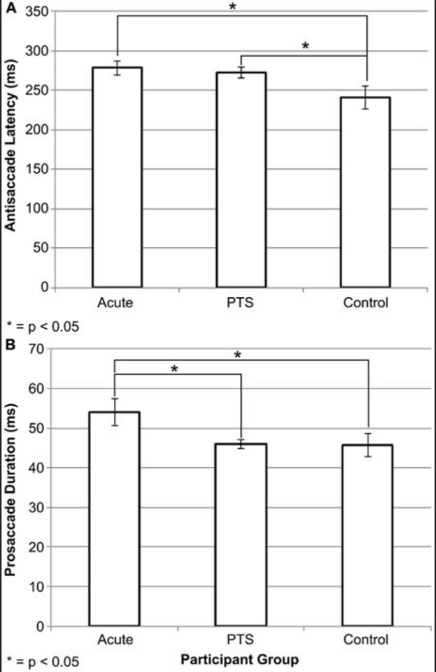


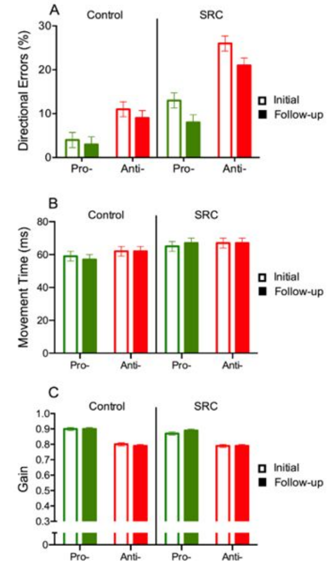
Munoz and Everling, 2004

Saccadometry Testing

Post-Head Injury

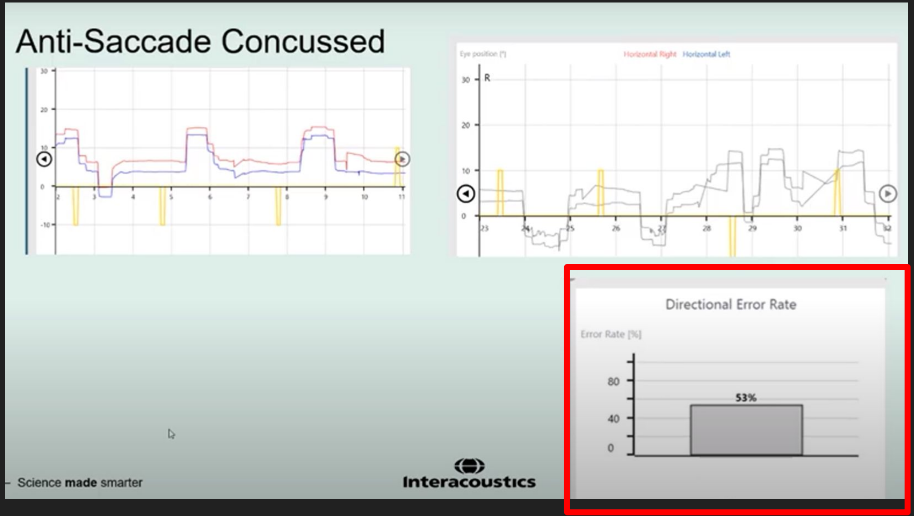
- Increased directional errors and latency in acute post-head injury
    - Balaban et al 2016; Ting et al, 2016; Ayala and Heath 2020; Johnson et al 2015a and 2015b





5. Control and sport-related concussion (SRC) group mean pro- and antisaccade directional errors (A), movement time (B), and gains (C) at initial and follow-up assessments. Error bars represent 95% within participant confidence intervals.

Two Cases of Saccadometry in Concussion



Balance Evaluation

Challenge the system!

- Tasking
- Headshake condition
- Visual background

Balance Evaluation

Post-Head Injury

- COBALT - 100% of uninjured athletes could complete the test whereas only 55% of concussed athletes could complete the test
    - Massingale et al, 2018

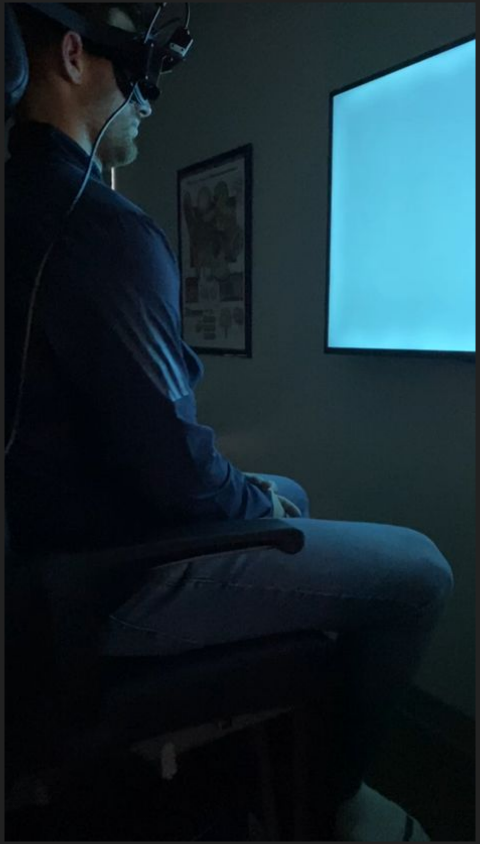
Table 1. - Percentage of Uninjured and Concussed Athletes Who Committed Errors on All COBALT Conditions (Uninjured, n = 132; Concussed, n = 58)

	Errors					
	0		1		2+	
Condition	Uninjured	Concussed	Uninjured	Concussed	Uninjured	Concussed
C1	NA	100%	NA	0%	NA	0%
C2	NA	100%	NA	0%	NA	0%
C3	100%	98.3%	0%	1.7%	0%	0%
C4	100%	98.3%	0%	1.7%	0%	0%
C5	NA	100%	NA	0%	NA	0%
C6	NA	94.3%	NA	5.7%	NA	0%
C7	85.6%	39.7%	12.1%	32.8%	2.3%	27.5%
C8	93.2%	51.7%	6.8%	31.0%	0%	17.3%

Abbreviation: NA, not applicable.

Rotational Studies

- Evaluation of the VOR at multiple frequencies
- Consider additional rotational evaluation tests:
  - WHY?
- RC Battery:
  - VOR enhancement
  - VOR Suppression
  - SHA
  - Step Testing



Rotational Studies

Post-Head Injury

- SHA testing:
    - **Athletes:** no difference (Christy et al, 2019)
    - **Military:** 45.5% abnormality of at least one SHA measure (King et al, 2018)
    - **Pediatrics:**
        - Reduced VOR gain (Zhou and Brodsky, 2015)
        - Normal in 95% of patients (Karl et al, 2021)
- VOR suppression:
    - **Athletes:** significant difference in gain in concussed athletes (Christy et al, 2019)

Dynamic Visual Acuity / Video Head Impulse Test

- Evaluation of high frequency VOR
  - DVA testing is looking at the patient's ability to keep clear vision with continuous head movement
  - vHIT looks at the predictable eye movement in response to a quick head movement/impulse

Post-Head Injury

- High frequency VOR - 57% abnormal
  - Zhou and Brodsky, 2015

Review of 100 Concussion Patients

- **Oculomotor:**
  - 49 of patients had convergence insufficiency
  - 6 had spontaneous nystagmus
  - 18 abnormal horizontal pursuit, 53 abnormal vertical pursuit
  - 46 abnormal antisaccades or couldn’t complete task
- **Balance:**
  - 49 abnormal results on dynamic surface (8 CNT)
  - 66 abnormal in headshake (25 CNT)
  - 50 abnormal in complex visual
- **VOR**:
  - 45 abnormal SHA
  - 28 abnormal VOR suppression
  - 52 abnormal DVA (14 CNT)

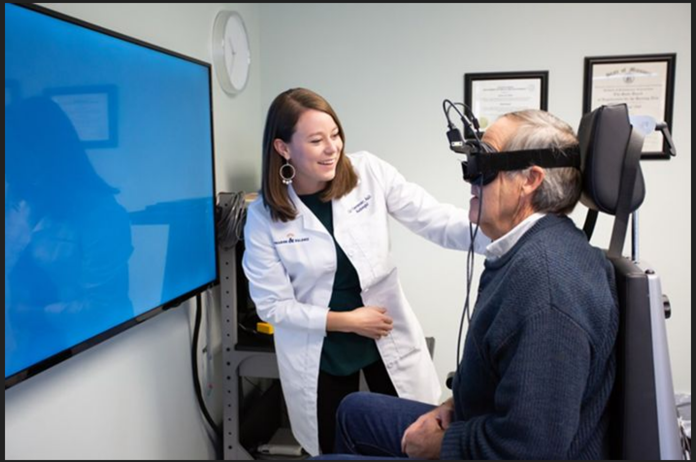
Review of 100 Acute Concussion Patients

- **VEMP:**
  - 11 abnormal
    - 2 patients had SSCD
- **BPPV:**
  - 7 patients had RPC BPPV
  - 4 had subjective BPPV
- **Auditory:**
  - 2 had air-bone gaps at 250 Hz
  - 7 had an asymmetric hearing loss
  - 31 had a symmetric hearing loss

Auditory Processing Disorder!!

Clinical Recommendations

- Case history and questionnaires
- Balance with headshake
- Oculomotor:
  - Anti-saccades
  - Near point convergence
- VOR suppression (RC)
- Dynamic Visual Acuity
- **Auditory Processing Disorder**



Questions

Chronic Phase

What is the chronic phase?

“No universally agreed upon definition” Liz, 2022.

Most generally defined as around 3 months post-head injury

Type of TBI

Blast

- Most often associated with direct damage to the end organ due to positive and negative pressure caused by a blast (Akin et al., 2017)
  - BPPV most common (~57%) followed by otolith dysfunction (~30%)
- Dizziness can start almost immediately and can last for six months or longer (Cohen et al., 2002; Dikmen et al., 2010)

Non-Blast

- Chronic effects are far less understood from an audiological perspective
- Handful of studies discuss the frequency, nature and progression of peripheral vestibular impairment in TBI

Direct effects of TBI on the vestibular system

- Ruptured otoliths and membranes (Kerr and Burne, 1975)
- Degenerative changes in otoliths (Schuknecht et al, 1956)
- Postural instability/imbalance attributed to otolith damage (Basta et al, 2005; Scherer et al, 2007)

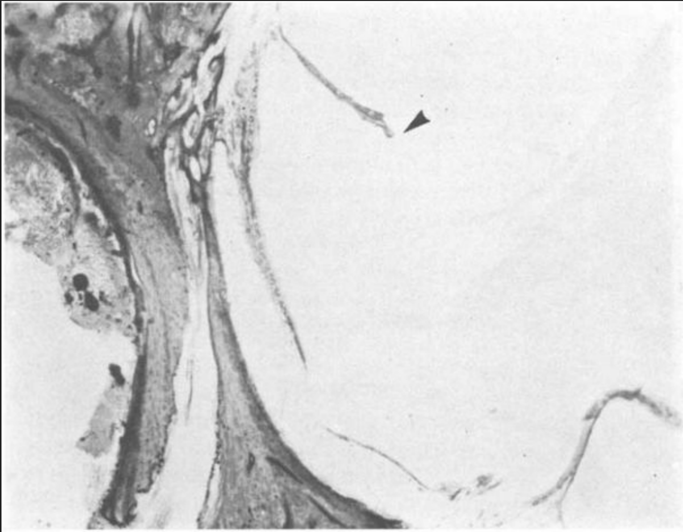


FIG. 16.
Rupture of the saccule.

Figure: Kerr and Byrne, 1975

Indirect effects of TBI on the vestibular system

- TBI is associated with distortion, shearing, impact to the cell membranes, compression, rotation, translation, laceration, and vascular injury affecting certain brain structures (Pinto et al., 2012; Laplaca, 2016; McGinn et al., 2015)
- This may lead to <u>secondary</u> factors that are non-mechanical
  - Vestibular systems particularly vulnerable due to complexity of subcortical and cortical structures involved.

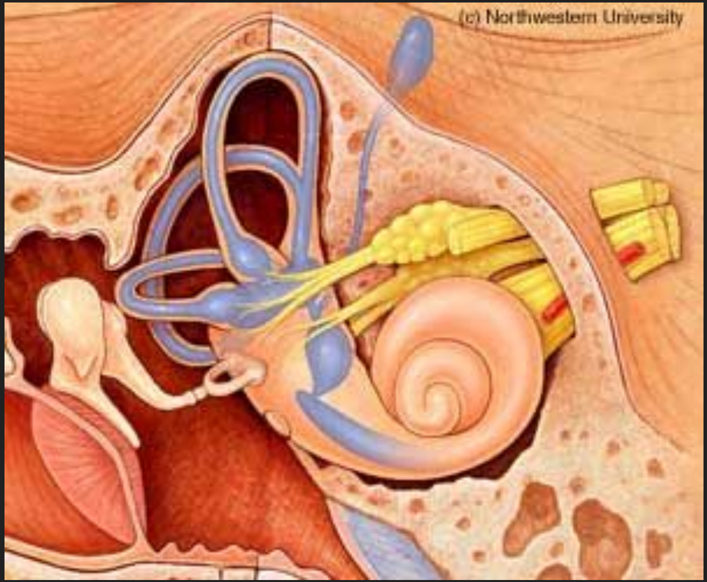
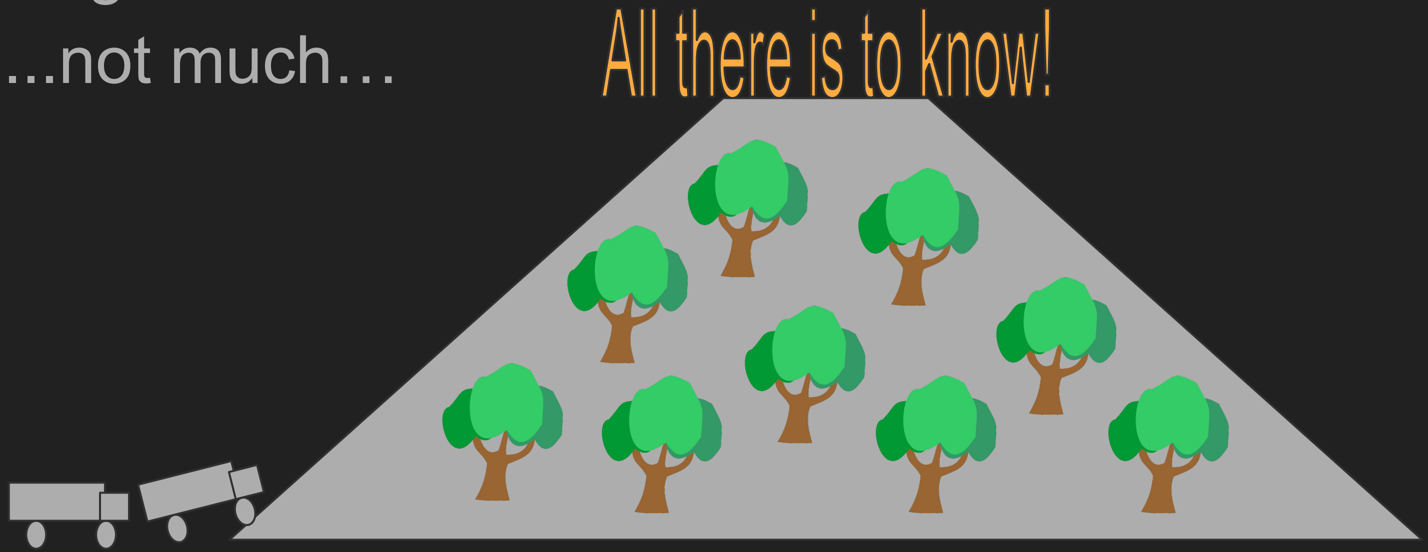


Figure: Northwestern University

Prevalence of peripheral vestibular impairment

From an audiological perspective? ...not much…



REVIEW ARTICLE

Check for updates

Frequency of peripheral vestibular pathology following traumatic brain injury: a systematic review of literature

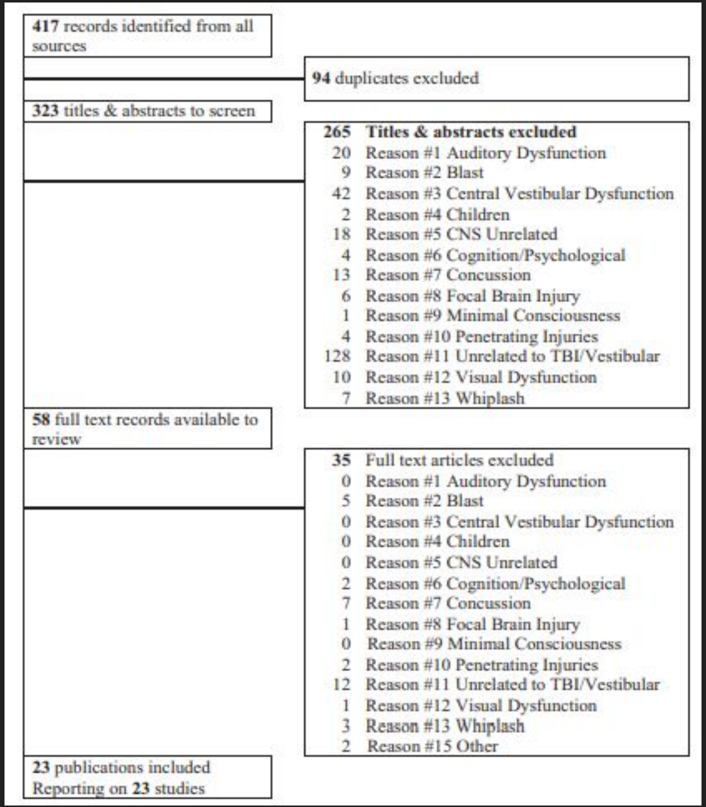
Bojana Šarkić[a], Jacinta M. Douglas[b,c], Andrea Simpson[b], Alexandra Vasconcelos[b], Bethany R. Scott[b], Lauren M. Melitsis[b] and Stephanie M. Spehar[b]

- Literature later than 1990 were included
- Inclusion criteria
  - January 1990 - May 2019
  - Adults >18 years
  - Studies were published in English and peer-reviewed
  - Full text available

- Exclusion criteria
  - Focused exclusively on auditory dysfunction
  - Blast-related TBI
  - Main focus on CNS impairment/lesions
  - Participants less than 18 years old
  - Studies focused on cognitive or psychological aspects of injury, focal brain injury described as root cause of symptoms
  - No loss of consciousness
  - Visual impairments
  - Neck injuries

Systematic Review (Sarkic et al., 2021)

- Most studies were retrospective analyses
- Multiple studies yielded inconclusive and/or unclear results. Inconclusive reported on vestibular deficits
- Four authors reviewed 323 studies



Reported tests (Sarkic et al., 2021)

Videonystagmography (VNG) - 12 of 22 studies (*n = 253*)

- Positioning Testing (Dix-Hallpike)
- Positional Testing
- Caloric Testing

Cervical and Ocular Vestibular Evoked Myogenic Potentials - 2 studies (*n = 61*)

Rotational Chair Testing - 3 studies (*n = 41*)

Video Head Impulse Testing (vHIT) - 3 studies (*n = 25*)

Davies and Luxon 1995; Basford et al. 2003; Herdman 1990; Kleffelgaard et al. 2016; Marzo et al. 2004; Shea, Ge, and Orchik 1995; Balatsouras et al. 2017; Basta et al. 2005; Bertholon et al. 2005; Fitzgerald 1995; Guyot et al. 2001; Skora et al. 2018; Basta et al. (2005) and Lee et al. (2011); Herdman 1990; Marzo et al. 2004; Kleffelgaard et al. 2016

After studies were excluded

1. PC-BPPV = 40% of participants (51/129)
2. Otolith impairment = 38 - 65% across two studies
    a. oVEMP
    b. cVEMP
3. Lateral SCC impairment = 37% of participants (59/155)
4. Abnormal vHIT = 6 - 52% of participants

(Sarkic et al., 2021)

Gaps in the literature

1. Substantial gap in well controlled studies investigating peripheral vestibular involvement in TBI
2. Great variability in between-subject study designs
3. Retrospective studies help identify the gaps
4. Findings are likely influenced by inconsistencies in nature of deficits reported, type of vestibular test chosen, differences in diagnostic criteria, small sample sizes, TBI severity and time of assessment post-TBI
5. Lack of standardization in reporting results (mention ABS discussion about this)

Need for prospective study designs!

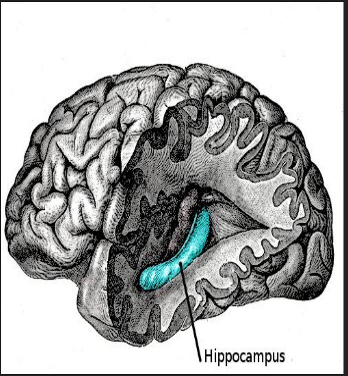
Rationale: Dizziness is among the top symptoms reported post-head injury, yet what is currently understood about the prevalence of peripheral vestibular impairment in TBI patients is largely based on retrospective studies and clinical experience

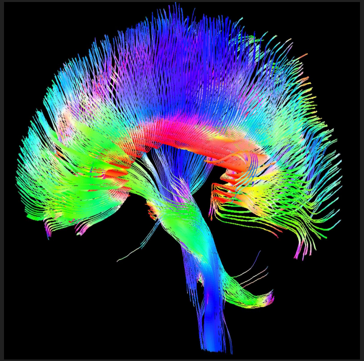
Collaboration with Memory and Communication Lab

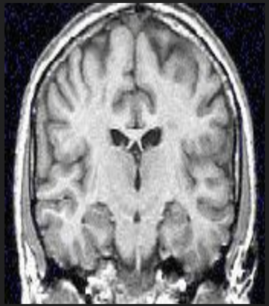
Melissa Duff, PhD

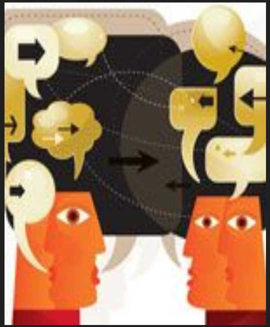
Professor at Vanderbilt University
Medical Center

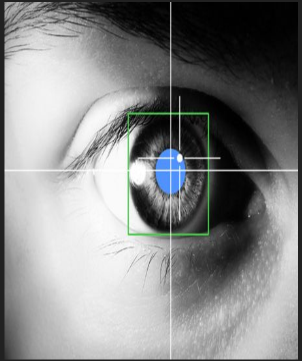
- Internationally recognized leader and expert in cognitive neuroscience and hippocampal memory in individuals with TBI.
- Research combining neuropsychology, and cognitive neuroscience of memory approaches, and rehabilitation..
- Director of the Brain Injury Registry at VUMC











Purpose

1. Primary purpose: is to characterize the prevalence of peripheral vestibular system impairments in chronic TBI through a well controlled and prospective study design

Design

TBI Group:

- Age 18-55 years
- Moderate-severe (>3 months post-head injury)
- No history of vision, hearing, or neurological disease (other than TBI)

NC Group:

- Age-matched (18-55) years
- Educated-matched
- No history of vision, balance, hearing, or neurological disease

Aim 1A: To characterize otolith function in patients with TBI

TBI is associated with:

- Ruptured otoliths and membranes (Kerr and Burne, 1975)
- Degenerative changes in otoliths (Schuknecht et al, 1956)
- Postural instability/imbalance attributed to otolith damage (Basta et al, 2005; Scherer et al, 2007)
- **Abnormal cVEMPs and oVEMPs** (Basta et al, 2005; Ernst et al, 2005)

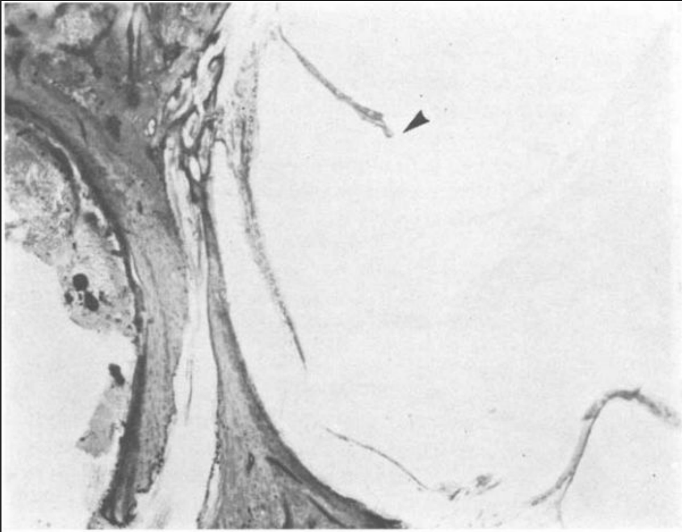


FIG. 16.
Rupture of the saccule.

Figure: Kerr and Byrne, 1975

Outline

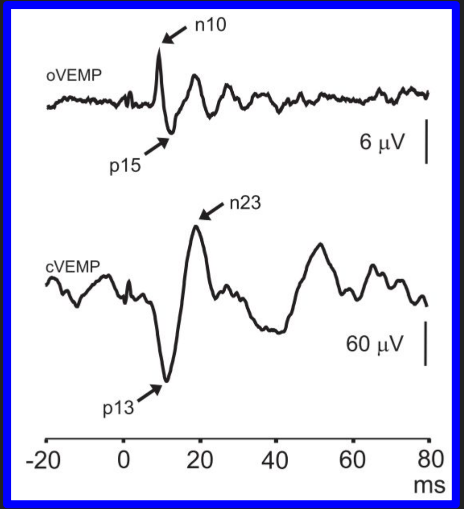
Design

Aim 1
Hypothesis 1a
Hypothesis 1b

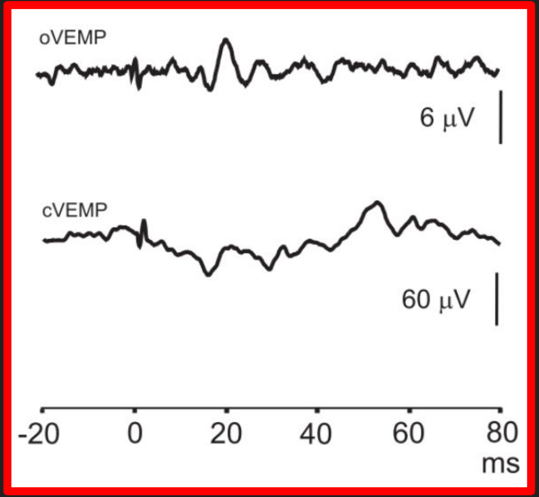
Aim 1A: To characterize otolith function in patients with TBI

Hypothesis: TBI group will have significantly reduced and/or absent VEMP responses compared to normal controls.

Exemplar Normal Response



Exemplar Abnormal Response



Figures adapted from: Rosengren et al, 2019

Outline

Design

Aim 1
Hypothesis 1a
Hypothesis 1b

Aim 1B: To characterize vestibular function in patients with TBI

TBI is associated with:

- Semicircular canal dysfunction, even for non-blast related TBI (for review, see Sarkic et al., 2021)

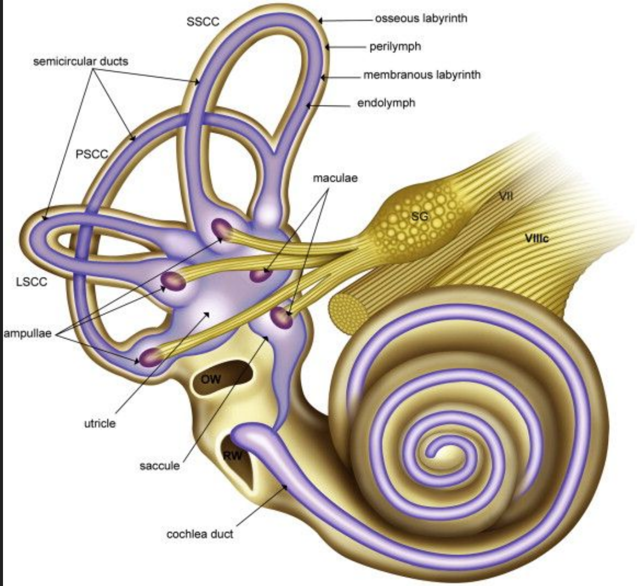


Figure: Connor and Sriskandan, 2014

Outline

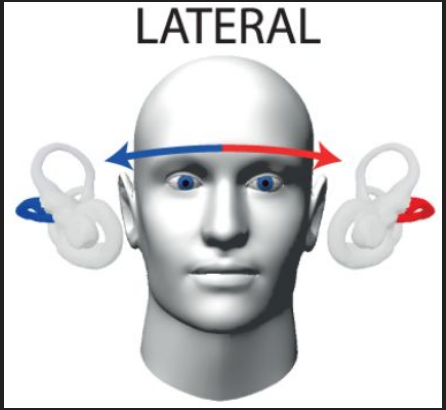
Design

Aim 1
Hypothesis 1a
Hypothesis 1b

Aim 1B: To characterize vestibular function in patients with TBI

Hypothesis: TBI group will have significantly reduced vHIT gain values and/or demonstrate an increased number of corrective saccades compared to normal controls.

Exemplar Normal Response **Exemplar Abnormal Response**



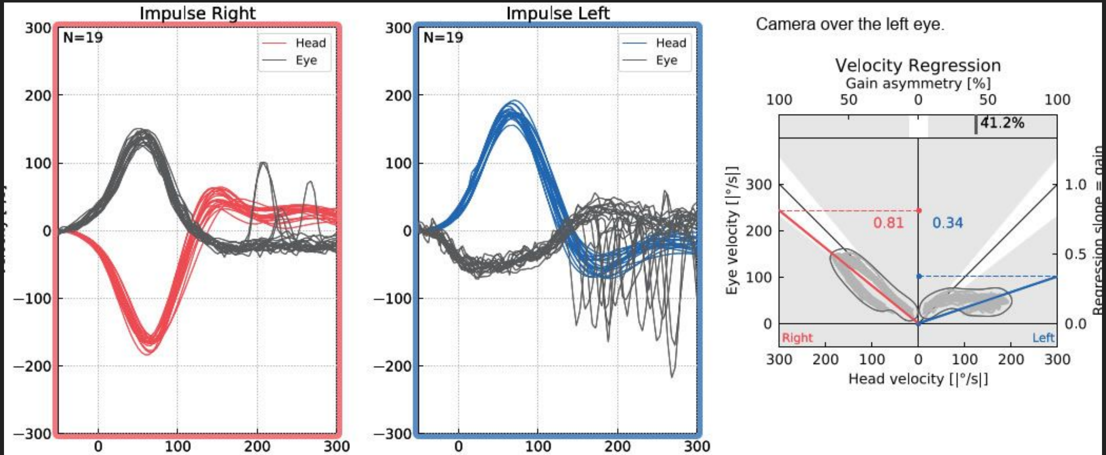


Figure adapted from MacDougall et al., 2013

Figure: Hain, 2021

Outline

Design

Aim 1

Hypothesis 1a

Hypothesis 1b

Spatial Memory Deficits in TBI

Secondary purpose

<u>TBI is associated with:</u>

- Impairments in spatial memory, navigation, and spatial reconstruction. (Lehnung et al, 2003; Skelton et al, 2006; Rigon et al, 2020)
- Impairments in temporal processing, such as verbal estimation and visual temporal order (Mioni et al, 2014; Dulas et al, 2022)

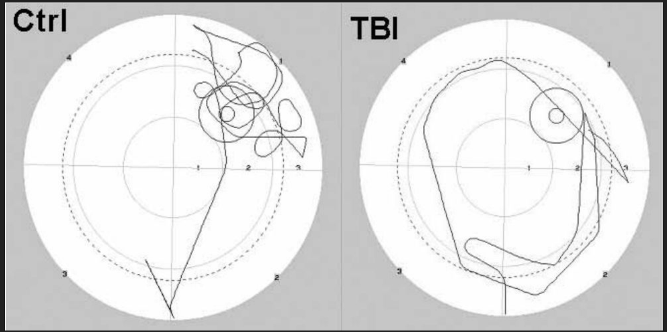


Figure (top): Skelton et al, 2006

The relationship between vestibular loss and spatial memory

Vestibular loss causes hippocampal atrophy and impaired sp

Thomas Brandt,[1] Franz
Roger Kalla,[1] Cynthia

Paul F. Smith[a,*], Yiwen Zheng[a], Arata H
[a]Department of Pharmacology and Toxicolog
Zealand
[b]Department of Otolaryngology, Osaka Univ

Journal of Vestibular Research 25 (2015) 73–89
DOI 10.3233/VES-150544
IOS Press

Vestibular involvement in cognition: Visuospatial ability, attention, executive function, and memory

Robin T. Bigelow* and Yuri Agrawal
Department of Otolaryngology, Head & Neck Surgery, Johns Hopkins University School of Medicine, Baltimore, MD, USA

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Kevin Pineault[1], Deryck Pearson[1], Eric Wei[1], Rebecca Kamil[1], Brooke Klatt[1], Yuri Agrawal[1]
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f

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From ear to uncertainty:
contributions to cognitiv

Paul F. Smith* and Yiwen Zheng
Department Pharmacology and Toxicology, School of Medical Scien
University of Otago, Dunedin, New Zealand

Study sample	Year	Number of subjects	Findings
Baltimore Longitudinal Study of Aging	2013 – 2014	183	Reduced saccular function associated with significantly poorer scores on neurocognitive tests of visuospatial memory
National Health and Nutrition Examination Survey	1999 – 2002	1303	Vestibular impairment (based on Romberg test) associated with significantly poorer Digit Symbol Substitution Score
National Health Interview Survey	2008	20,950	Symptoms of vertigo significantly associated with 4-fold increase in difficulty remembering and confusion

Adapted from Agrawal et al., 2020

Weighing the evidence

Animal studies

- Animals use vestibular information to navigate their environment suggesting a direct connection to the hippocampus (Beritoff, 1965; Potegal et al., 1977; Etienne, 1980; Mittelstaedt and Mittelstaedt, 1980; Horn et al., 1981; Potegal, 1982; Miller et al., 1983; Etienne and Jeffery, 2004)
- Vestibular stimulation modulates 'place cells' in the hippocampus and theta rhythm oscillation (Gavrilov et al., 1995; Wiener et al., 1995)
- Vestibular damage led to deficits in spatial memory even after compensation occurred (Zheng et al., 2006, 2007, 2008; Baek et al., 2010)
- Bilateral vestibular loss typically leads to more severe deficits (Baek et al., 2010)

Human studies

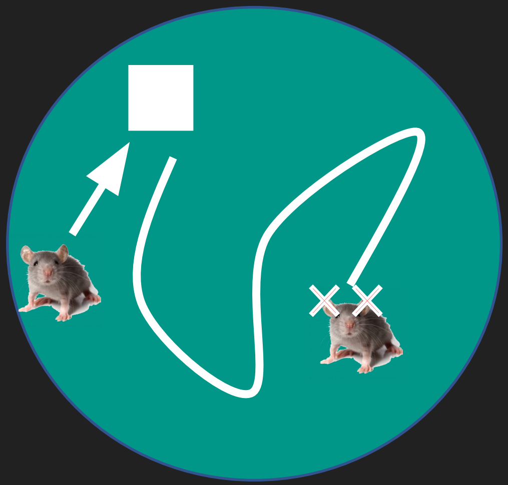
- Vestibular loss and cognitive deficits in humans is less understood
- However, many of the general themes are supported by evidence to suggest a connection between the vestibular system and hippocampus (Brandt et al., 2005; Schautzer et al., 2006)
- Theories suggesting vestibular damage is associated with direct and indirect effects on cognition (Hanes and McCollum, 2006)
    - **Direct:** chronic stress, absent vestibular input leading to cell death, etc.
    - **Indirect:** limited attentional resources, concentration, etc.

Vestibular loss causes hippocampal atrophy and impaired spatial memory in humans

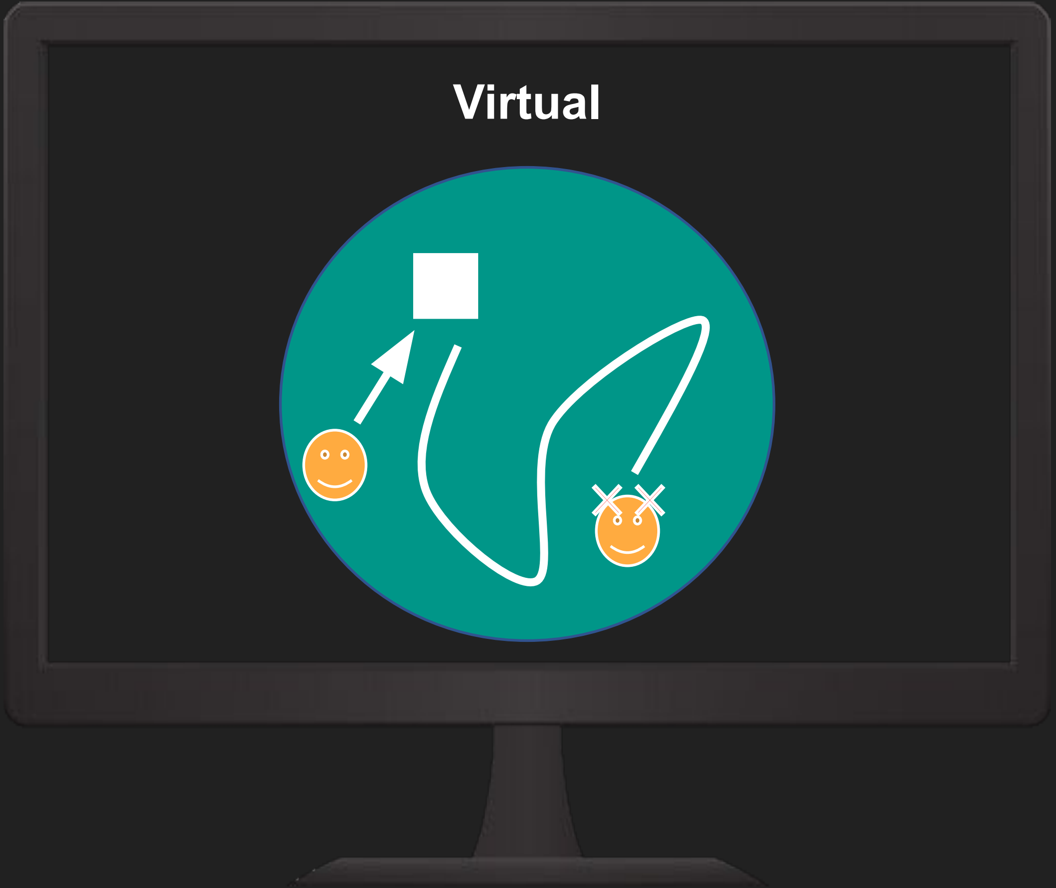
Thomas Brandt,[1] Franz Schautzer,[2] Derek A. Hamilton,[3] Roland Brüning,[4] Hans J. Markowitsch,[5] Roger Kalla,[1] Cynthia Darlington,[6] Paul Smith[6] and Michael Strupp[1]

- One of the first studies in humans to show that vestibular loss was associated with poor <u>spatial memory and navigation (even when stationary)</u>

Animal studies



Virtual



Vestibular loss causes hippocampal atrophy and impaired spatial memory in humans

Thomas Brandt,[1] Franz Schautzer,[2] Derek A. Hamilton,[3] Roland Brüning,[4] Hans J. Markowitsch,[5] Roger Kalla,[1] Cynthia Darlington,[6] Paul Smith[6] and Michael Strupp[1]

- One of the first studies in humans to show that vestibular loss was associated with poor <u>spatial memory and navigation (even when stationary)</u>
    - In patients with bilateral vestibular loss (BVL) compared to age- and education-matched controls:
        - Virtual water maze task: patients with BVL took longer paths and more time to reach hidden platform

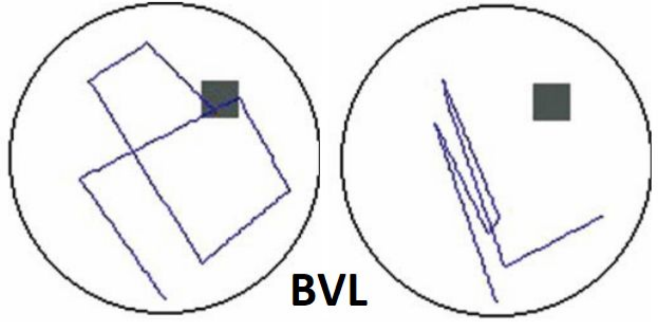
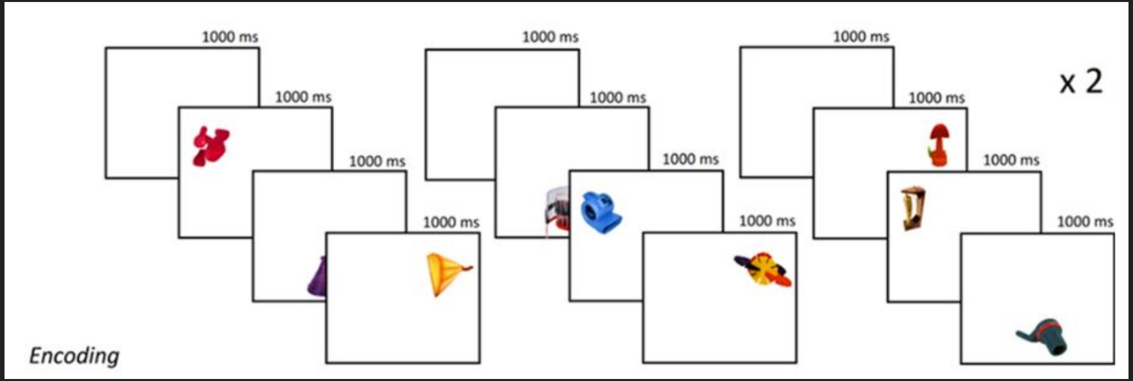


Figure (bottom): Brandt et al., 2005

Aim 2a: Determine the relationship between vestibular loss and spatial memory in patients with TBI

Hypothesis 2a: Participants with reduced vestibular function will demonstrate significantly poorer spatial cognitive performance.



Figures adapted from: Lee et al., 2020

Outline

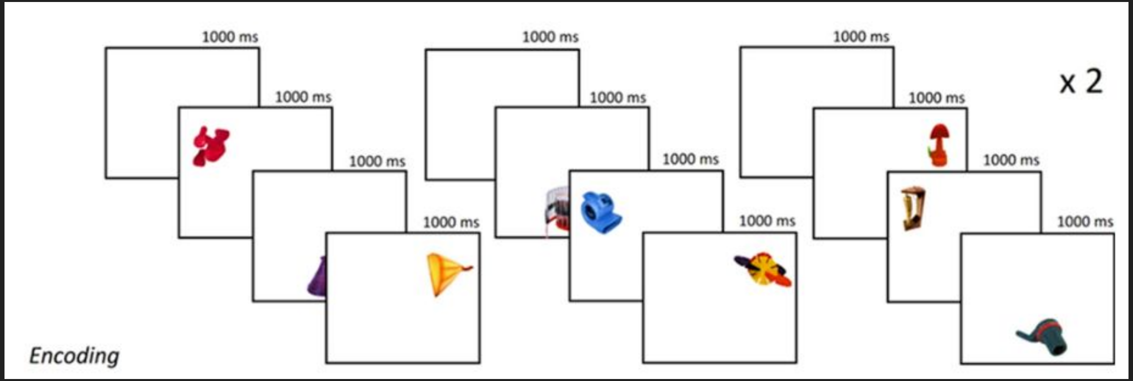
Design

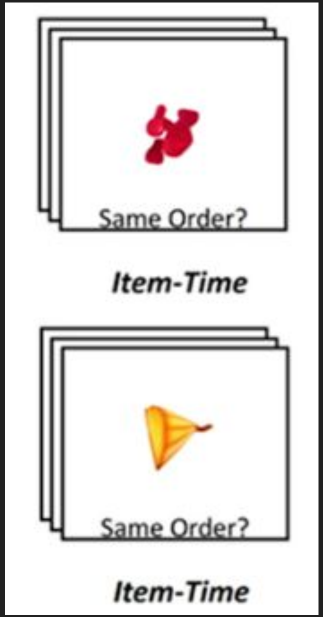
Aim 1
Hypothesis 1a
Hypothesis 1b

Aim 2
Hypothesis 2a
Hypothesis 2b

Aim 2b: Determine the relationship between vestibular loss and spatial memory in patients with TBI

Hypothesis 2b: Participants with vestibular impairment will not significantly differ in temporal cognitive performance.





Figures adapted from: Lee et al., 2020

Outline

Design

Aim 1
Hypothesis 1a
Hypothesis 1b

Aim 2
Hypothesis 2a
Hypothesis 2b

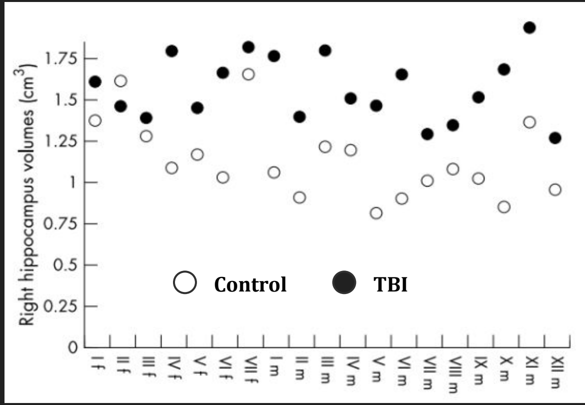
Future Aims: To characterize hippocampal volumes in patients with and without vestibular loss in TBI

TBI is associated with:

- A mean 27% decrease in hippocampal volume compared to controls {Tomaiuolo et al., 2004)
- A mean 10% decrease in hippocampal volume between acute and chronic stages {Warner et al., 2010)

Vestibular loss is associated with:

- A mean 17% decrease in hippocampal volume compared to controls {Brandt et al., 2005)



Control BVL

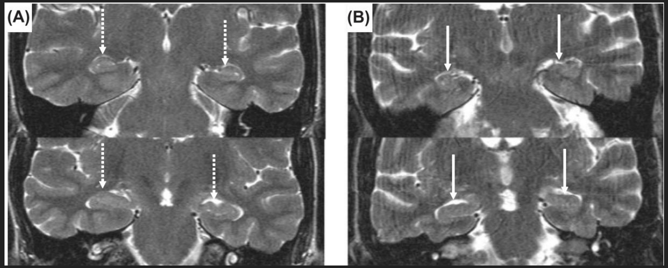


Figure (top): Tomaiuolo et al, 2004; Figure (bottom): Brandt et al., 2005

Outline

Design

Aim 1
Hypothesis 1a
Hypothesis 1b

Aim 2
Hypothesis 2a
Hypothesis 2b

Future Aims

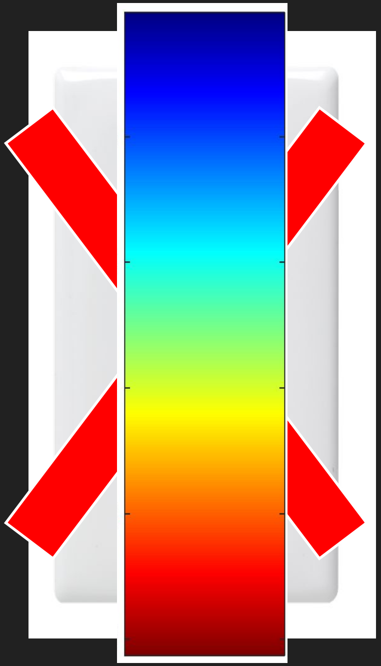
Other areas worth exploring:

- Prevalence of central vestibular system impairments
- Functional consequences of spatial memory deficits
- Ocular motility

Stay tuned!

Questions

- **What proportion of patients with TBI have an underlying vestibular impairment?**
- **Does vestibular loss contribute to the spatial memory deficits commonly seen in TBI?**
- What are the functional consequences of spatial memory impairment and how does it relate to balance?
- Does vestibular rehabilitation therapy improve spatial memory deficits?
- What degree of impairment is associated with cognitive deficits? (e.g., sensory end organs involved, frequency, etc.)
- Can we better characterize the type of spatial memory deficits following a vestibular impairment?
- Do TBI patients with and without a vestibular impairment show the same degree of cognitive impairment?
- Can we implement screening protocols to monitor cognitive function in dizzy patients? (e.g., questionnaires, etc.)
- What are the long term cognitive effects of TBI?



- Sensory end organ
- Peripheral vs. central
- Frequency of impairment
- Compensation status
- Duration

Research Team

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Yuri Agrawal, MD, PhD

Part of this study is funded by the Vanderbilt Institute of Clinical and Translational Research

General Conclusions

Acute and Chronic

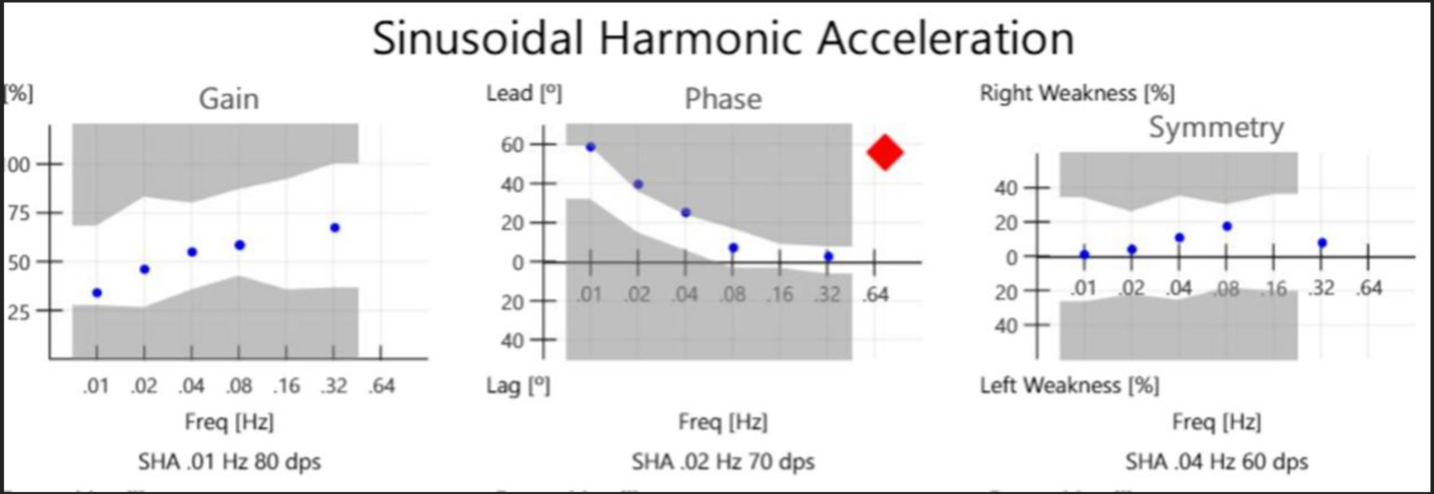
- Audiologists can be a play a vital role in assisting with the diagnosis and tracking the recovery following a head injury.
- Concussion evaluation can utilize existing vestibular tests, but your questions/expected outcome of the tests may be different.
- The vestibular system is particularly vulnerable to TBI given the complexity of subcortical and cortical system involved
- The vestibular system plays a role in acute and chronic recovery of a patient following head injury.
- There is evidence to support peripheral involvement in TBI, though the type, severity, and degree of vestibular impairment is not well defined.

Case Studies

Case Study 1

Using Vestibular Testing to Track Recovery

19 year old female - MVA (31 days post head injury)



- Abnormalities at First Eval:
  - Abnormal balance - eyes closed on foam
  - Phase lead in SHA testing
  - Shortened TCs and asymmetry in Step

Returned two weeks later - no intervention

- RC returned to normal limits
    - SHA testing
    - Step testing
- Balance performance normal
    - Could perform eyes closed head shaking on foam

Case Study 2

Using Objective Testing to Discover Origin of the Issue

Two Patients - Same Outcome

13 year old female
Direct head injury (volleyball)

- Primary complaint: headaches
- Dx with POTS
- Fam hx of headaches/migraines
- Taking magnesium

57 year old female
Fall on cement floor

- Daily headaches, imbalance, lightheadedness
- Left ear tinnitus
- Personal hx of anxiety, depression, sleep disorder

Two Patients - Same Outcome

13 year old female
Direct head injury (volleyball)

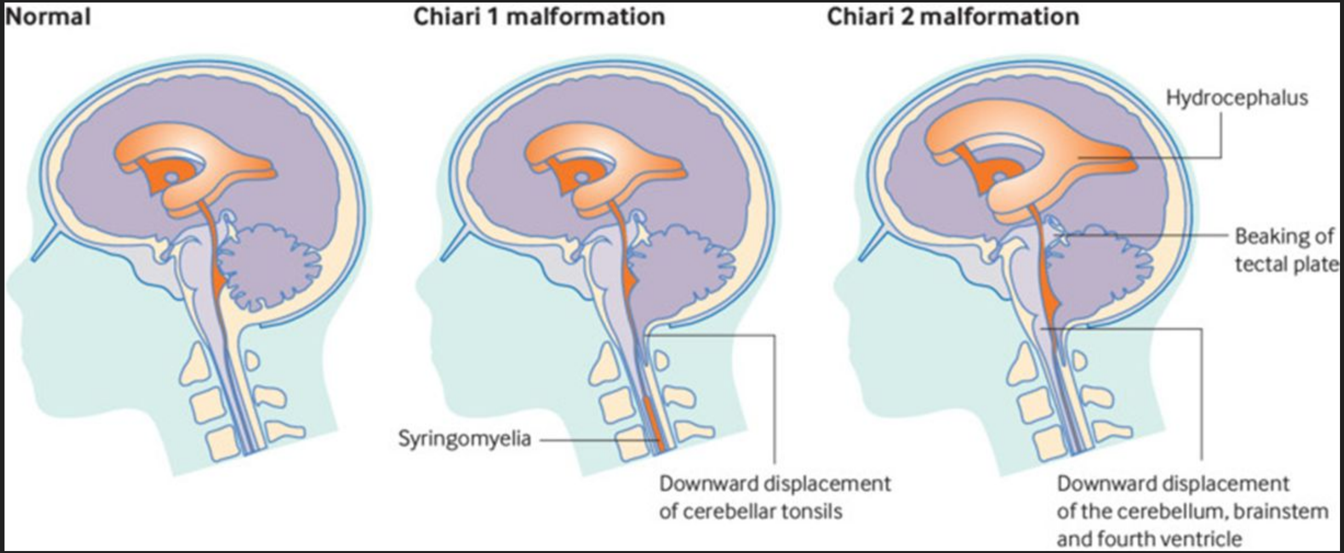
- Upbeating spontaneous nystagmus
- Upbeating nystagmus post-HS
- Upbeating nystagmus in supine, head and body right and left
- Abnormal reduction of VOR gain in rotational studies

57 year old female
Fall on cement floor

- Saccadic pursuit
- Abnormal OPK (low gain)
- Prolonged saccades
- Upbeating nystagmus in all positionals
- Abnormal reduction of VOR gain in rotational studies

Recommendations? Thoughts?

Referred to Neuro…



Chiari Type 1 Malformation

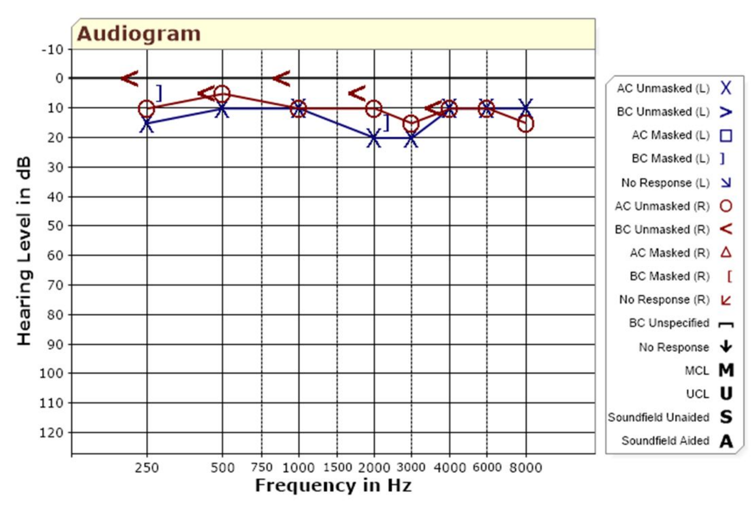
Is Chiari Caused by Concussion?

- May be incidental findings through imaging following head injury
  - Pediatric study of 421 concussed patients, 52 abnormalities and 8 were Chiari Malformations
- Patients with Chiari may have increased risk of concussion:
  - Spencer R, Leach P. Asymptomatic Chiari type I malformation: should patients be advised against participation in contact sports? *Br J Neurosurg*. 2017; 31 (4): 415– 421. doi:10.1080/02688697.2017.1297767
  - Tator CH, Davis HS, Dufort PA, et al Postconcussion syndrome: demographics and predictors in 221 patients. *J Neurosurg*. 2016; 125 (5): 1206– 1216. doi:10.3171/2015.6.JNS15664
- Patients who are asymptomatic may become symptomatic following head injury.
  - Freeman MD, Rosa S, Harshfield D, Smith F, Bennett R, Centeno CJ, Kornel E, Nystrom A, Heffez D, Kohles SS. A case-control study of cerebellar tonsillar ectopia (Chiari) and head/neck trauma (whiplash). Brain Inj. 2010;24(7-8):988-94. doi: 10.3109/02699052.2010.490512. PMID: 20545453.
  - Spencer R, Leach P. Asymptomatic Chiari Type I malformation: should patients be advised against participation in contact sports? Br J Neurosurg. 2017 Aug;31(4):415-421. doi: 10.1080/02688697.2017.1297767. Epub 2017 Mar 2. PMID: 28637118.
  - Wan et al, 2008. Conversion to symptomatic Chiari I malformation after minor head or neck trauma. November 2008 Neurosurgery 63(4):748-53; discussion 753

Case Study 3

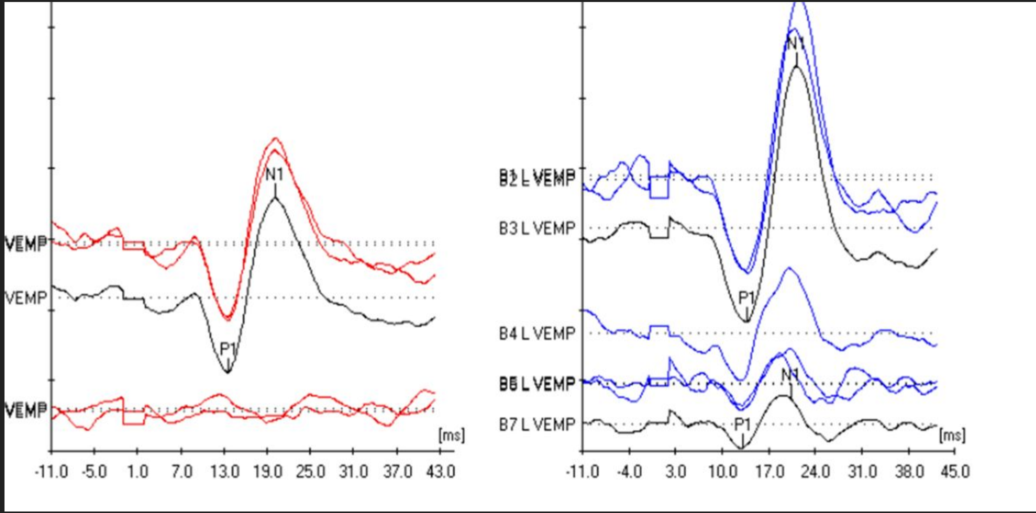
Why you shouldn’t ignore sound sensitivity…

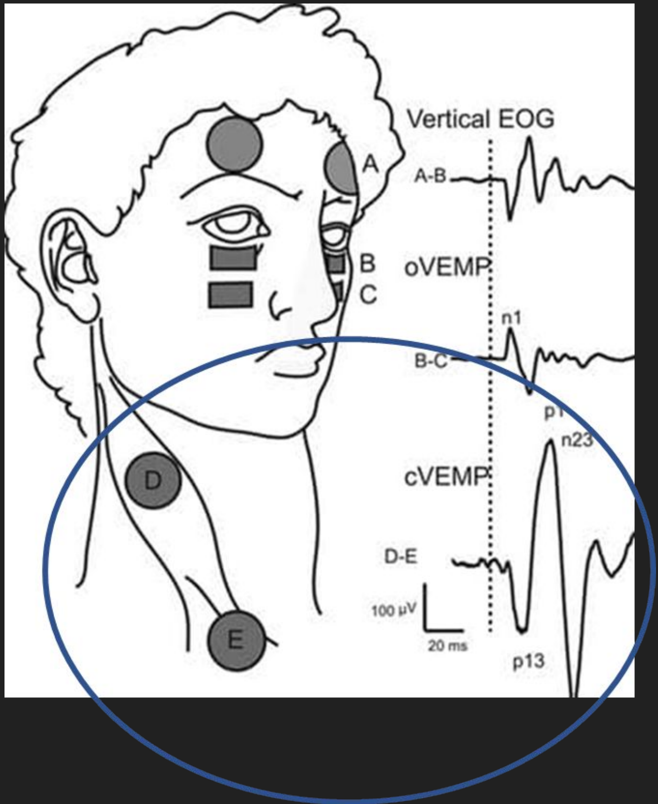
36 year old female hit by a garage door



- Increased sound sensitivity
- Aural pressure/fullness
- No vertigo
- Left ear tinnitus
  - Sounds like blood flowing
- Imbalance
- Difficulty reading
- Daily headaches

Cervical VEMP Results





Barany Society 2020 Diagnostic Criteria

The diagnosis of superior semicircular canal dehiscence syndrome requires all of the following criteria:

A. At least 1 of the following symptoms consistent with a third window lesion of the inner ear:
    1. Bone conduction hyperacusis[1]
    2. Sound-induced vertigo and/or oscillopsia time-locked to the stimulus[2]
    3. Pressure-induced vertigo and/or oscillopsia time-locked to the stimulus[3]
    4. Pulsatile tinnitus

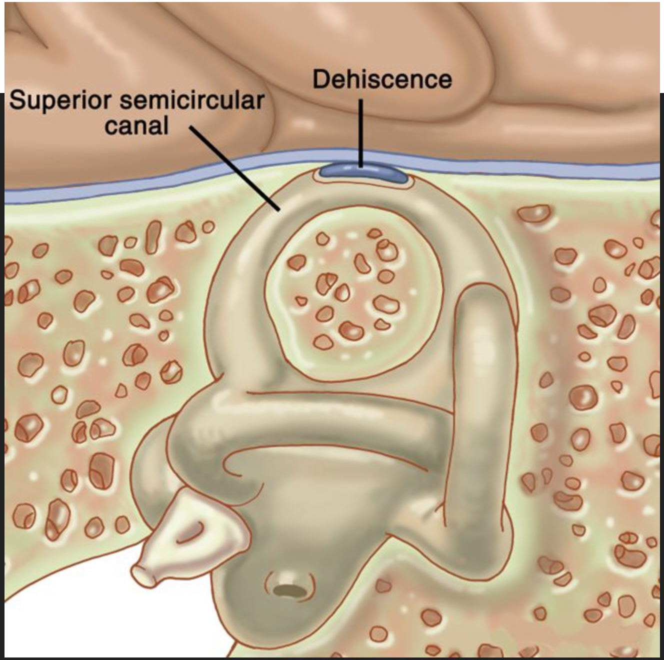
B. At least 1 of the following signs or diagnostic tests indicating a 'third mobile window' of the inner ear:
    1. Nystagmus characteristic of excitation or inhibition of the affected superior canal evoked by sound, or by changes in middle ear pressure or intracranial pressure[4]
    2. Low-frequency negative bone conduction thresholds on pure tone audiometry[5]
    3. Enhanced VEMP responses (low cervical VEMP thresholds or high ocular VEMP amplitudes)[6]

C. High resolution temporal bone CT imaging with multiplanar reconstruction demonstrating dehiscence of the superior canal[7]

D. Not better accounted for by another vestibular disease or disorder

She has left superior semicircular canal dehiscence by history, symptomatology, vestibular testing, and imaging. Options of observation and possible physical therapy versus pharmacologic intervention were discussed; plugging of the superior semicircular canal was also discussed through a possible middle fossa approach. Risks were discussed with my handout, hearing loss, facial weakness, dizziness, infection, bleeding within the skull, seizures, and cerebrospinal fluid leak, speech changes were discussed.

- Approximately 3% of my patients have been diagnosed with a SSCD post-concussion



Treatment & Follow Up

Assessment and Plan:

[redacted] returns about 2 weeks out from a left superior semicircular canal plugging with fascia and coverage with bone pate. She is not spinning spontaneously; but she does tend to veer to the left side. She is slowly improving her activity level. She is not up to where she would like to be.

Exam today reveals a left normal eardrum, cranial nerves are intact, Weber is to the left side.

I Minna send her to [redacted] for vestibular rehabilitation therapy after the plugging; I did explain to her that this will help work out her brain in response to the changes that occurred to her left ear. I expect her to have a full or near complete recovery with time. I will have her return in about 2 months.

Questions?

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Daniel - daniel.romero@vanderbilt.edu

22 episodes

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Join us this month for another guest episode with you guessed it, Dr. Richard Roberts! We discuss vestibular audiology, getting involved, research, and so much more!

▶ PLAY 1 hr 5 min

AUG 11, 2022

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How do we even begin to assess vestibular function in children? We cover all you need to know this month with pediatric vestibular testing! Follow us on Instagram @adoseofdizzypodcast Follow us on Twitter @adoseofdizzy

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