**TBI-BH ECHO**

Traumatic Brain Injury – Behavioral Health ECHO

UW Medicine | Psychiatry and Behavioral Sciences

# Pediatric Moderate to Severe TBI

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UW Medicine
UW SCHOOL OF MEDICINE

Project ECHO

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# Speaker disclosures

✓ No conflicts of interest to disclose

The following series planners have no conflicts of interest:

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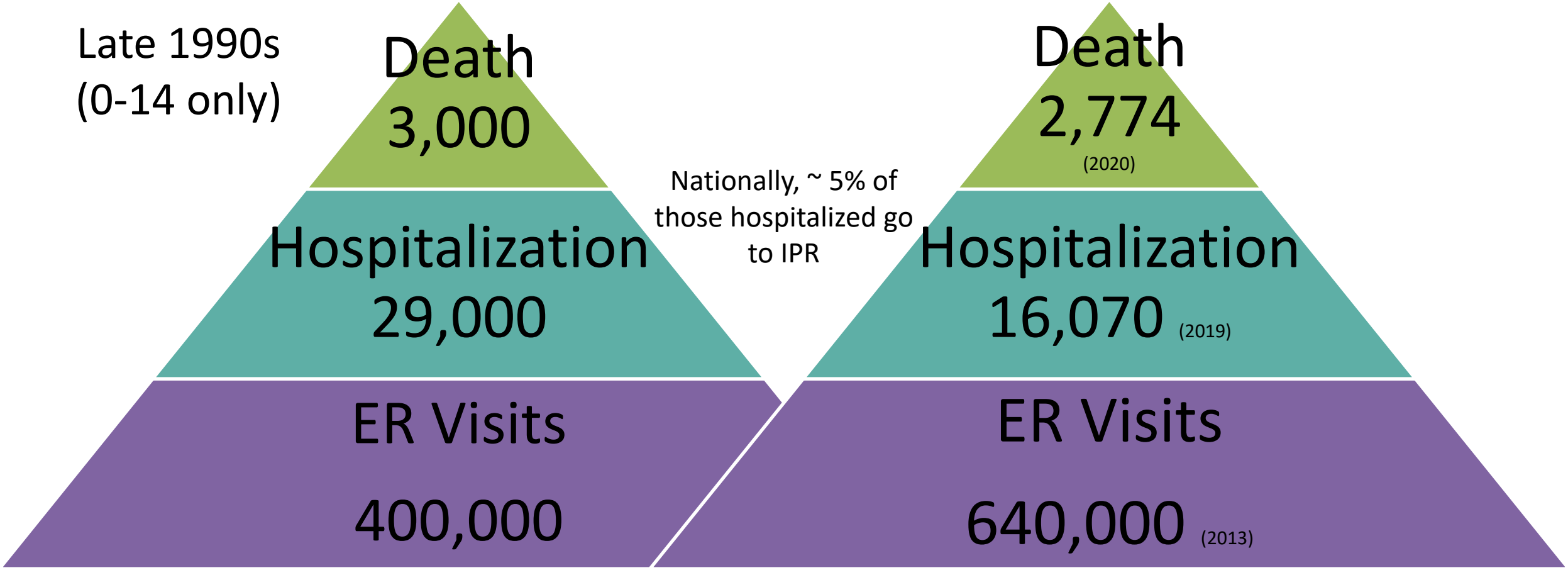
# Objectives

1. Understand TBI pathophysiology and impact on brain function
2. Identify medical complications after TBI (focus on post-acute care)
3. Review outcomes and prognostic factors
4. Review inequities related to TBI

# Pediatric (0-17) TBI – U.S. annual numbers




CDC TBI Data https://www.cdc.gov/traumaticbraininjury/data/index.html; https://www.cdc.gov/traumaticbraininjury/assessing_outcomes_in_children.html

# What is a traumatic brain injury?

**Definition:**

1. **External mechanical force to the brain**
2. **Results in diminished or altered state of consciousness**
3. **Impairment of cognitive, physical and/or psychosocial function**

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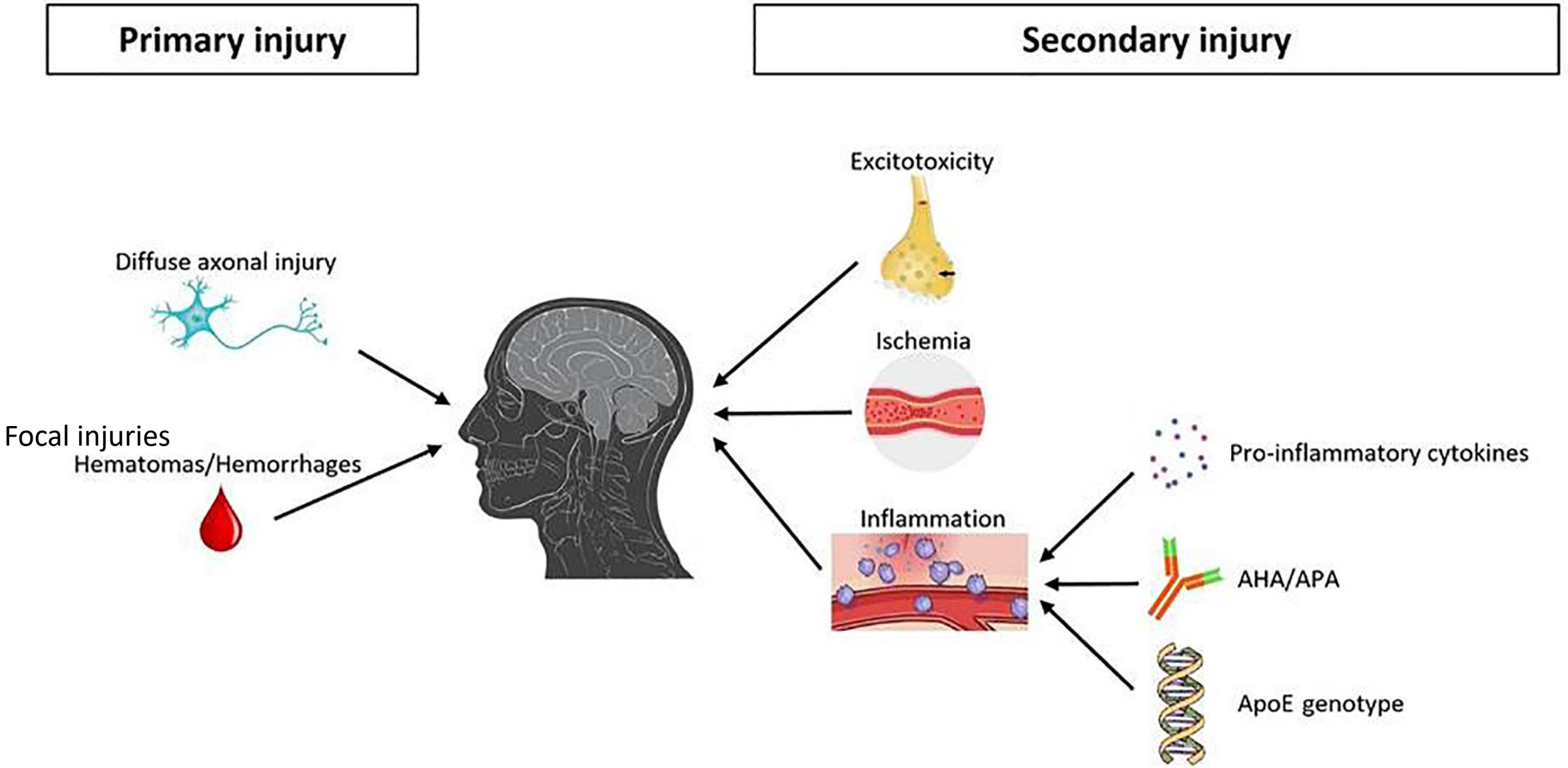


Gasco 2015

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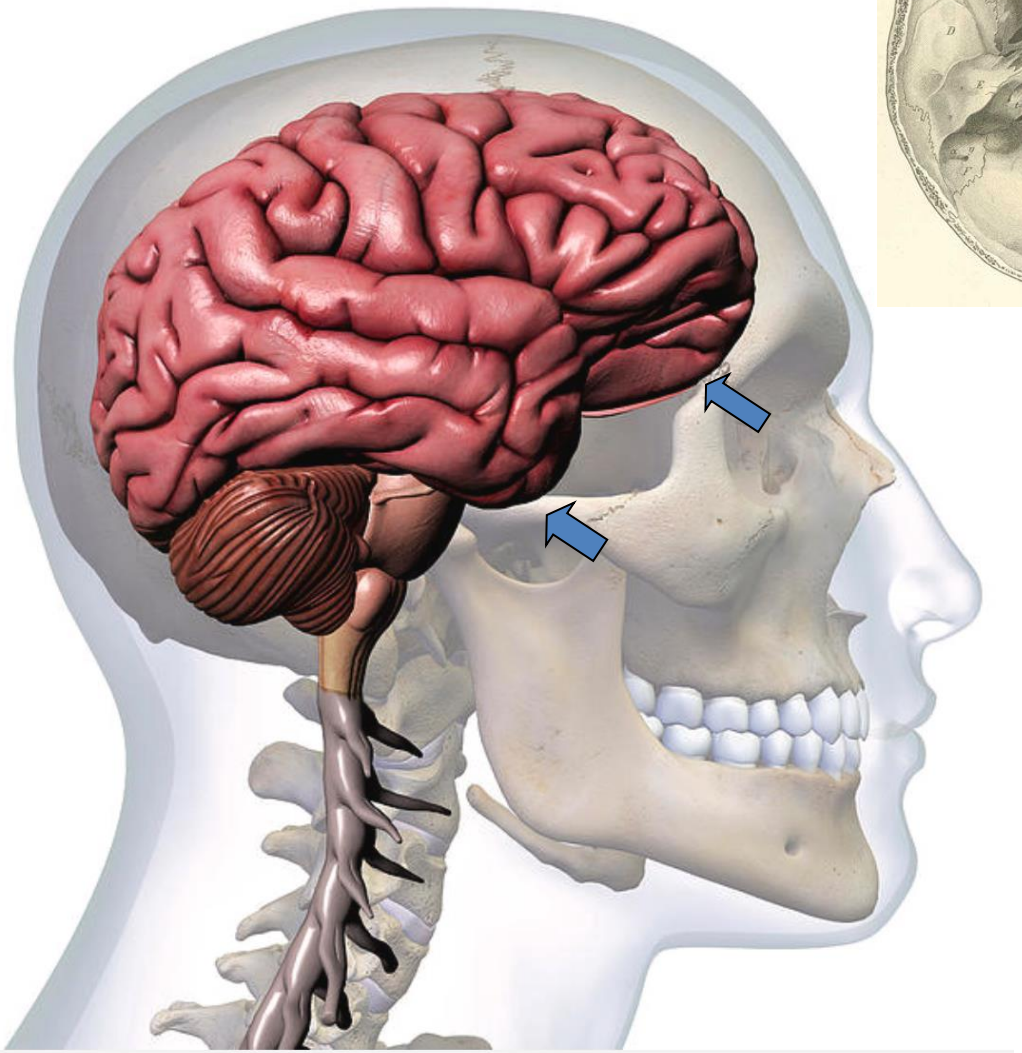
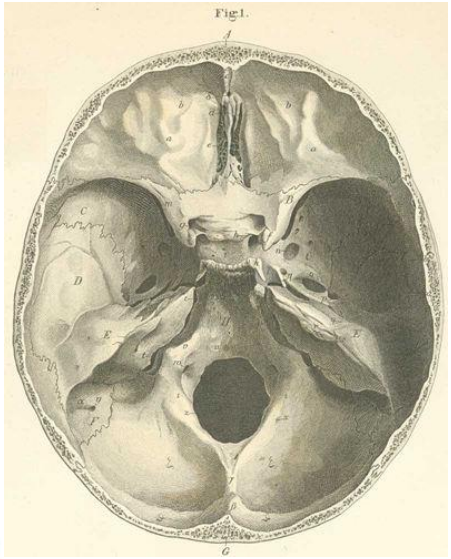
# Focal Injuries






- Contact to head
  - Coup, contracoup
- Linear movement of head
- Contusion, hematoma, SDH, SAH

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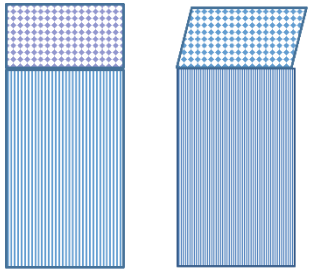
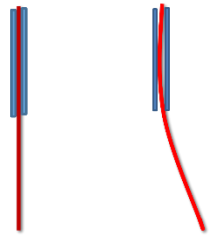
# Diffuse Axonal Injury

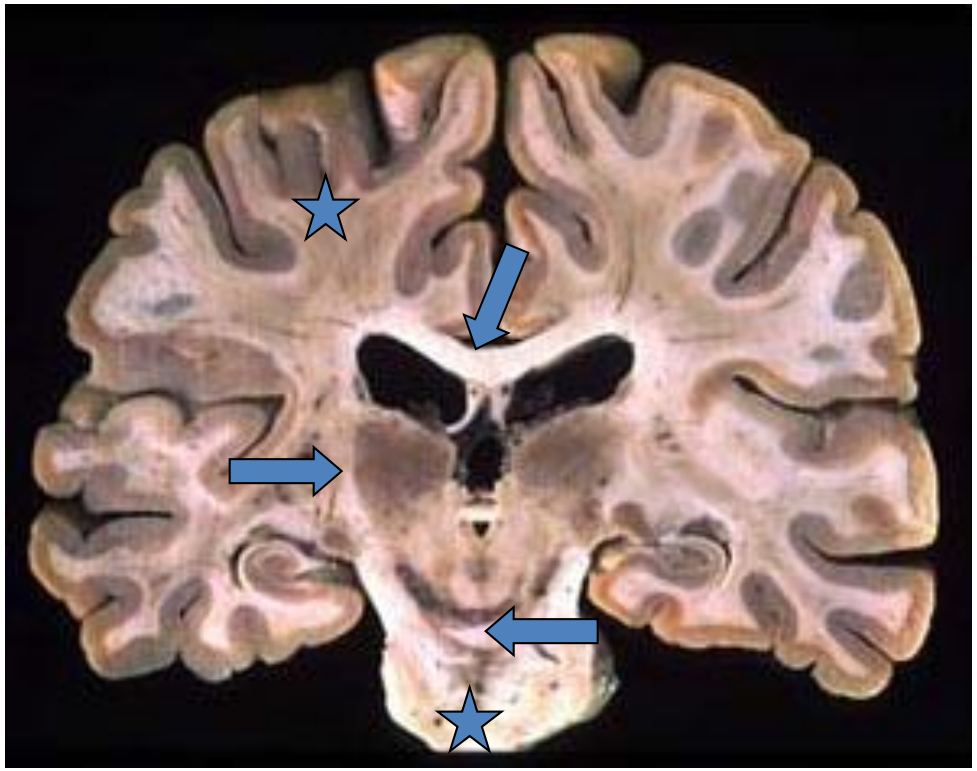
Rotational Forces → Axonal Stretch → Cytoskeletal injury → Influx of $Na^{+}$ and $Ca^{++}$ → Proteolytic activity/2° axotomy → Axotomy



Different densities



Tethered areas



Vulnerable Areas

- Grey-white junction
- Corpus callosum
- Internal capsule
- Superior cerebellar peduncle
- Brainstem

# Abusive head trauma

- Injury to the skull or intracranial injury from inflicted blunt impact or violent shaking
- Leading cause of head injuries <1 yr, 3rd leading cause <5 yr
- AHT is on differential for altered mental status, fussiness, vomiting
  - Comprehensive eval: skin, mouth, skeletal survey, head/spine imaging, ophthalmology, etc.
- Associated with delay in seeking medical care, more secondary brain injury (hypoxia, ischemia, inflammatory cascades)
- 70% have some degree of chronic neurologic impairment
  - If TBI (from any cause) occurs before age of 2, falls under CP umbrella

AAP "Abusive Head Trauma" https://www.aap.org/en/patient-care/child-abuse-and-neglect/abusive-head-trauma/?srsltid=AfmBOopPG7XUbRy3o1AepzgpZrwgu7Ohwcy16B0F-2eaTvY9yRw9Fu62

Narang et al "Abusive Head Trauma in Infants and Children" *Pediatrics* (2020) 145 (4): e20200203

# Disorders of Consciousness

- **Consciousness**: State of awareness of self and the environment.
- Requires adequate wakefulness + awareness of experience:
  - Sensory experience
  - Cognitive experience
  - Affective experience
- DOC refers to impaired awareness of self and the external environment

Plum & Posner, 1982

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# Disorders of Consciousness

## Coma

- Unresponsive, eyes closed, no arousal on stimulation

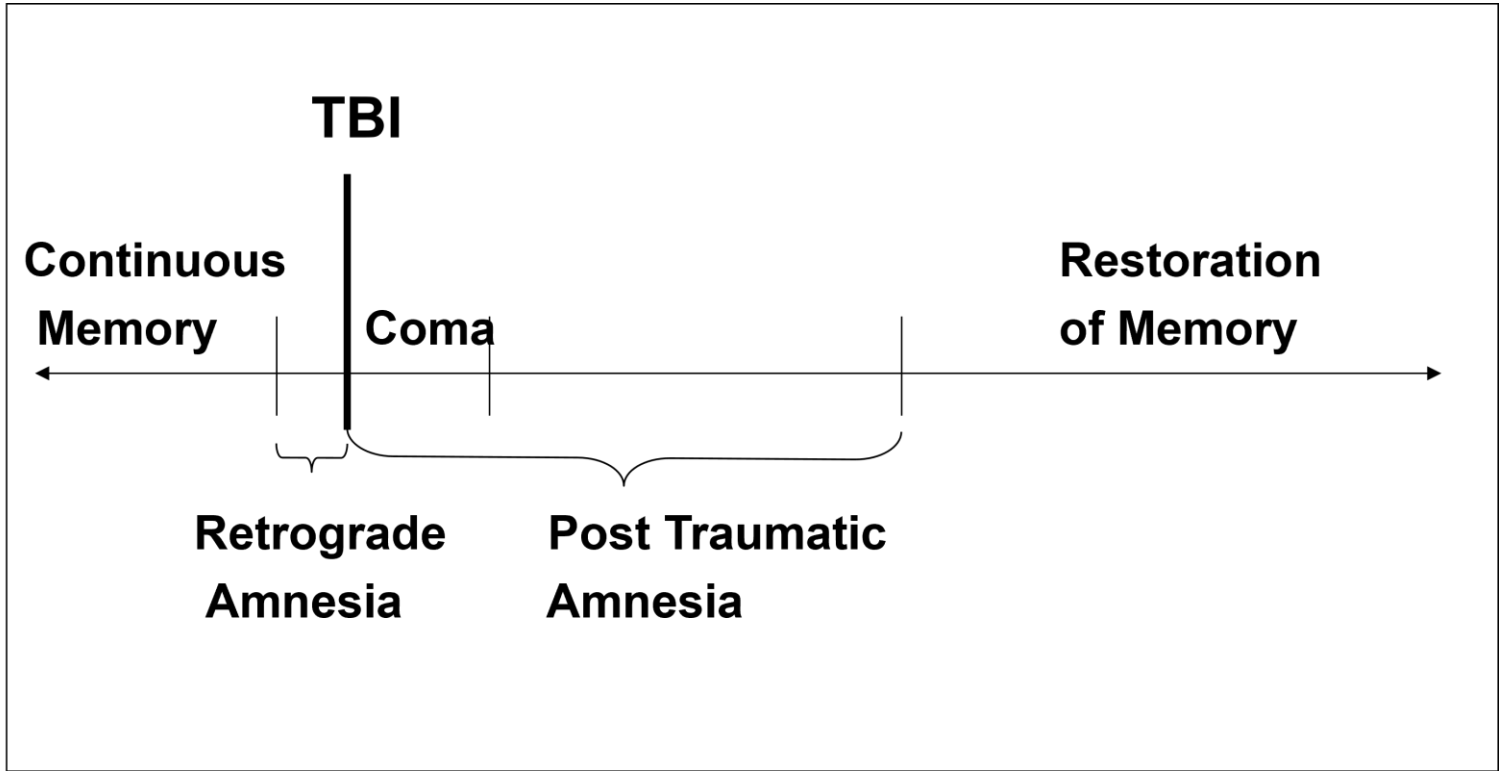
## Unresponsive Wakefulness Syndrome/VS

- Periods of waking without awareness of self and/or environment

## Minimally Conscious State (MCS-/MCS+)

- Minimal but reproducible behavioral signs of consciousness, with (MCS+) or without (MCS-) evidence of language function

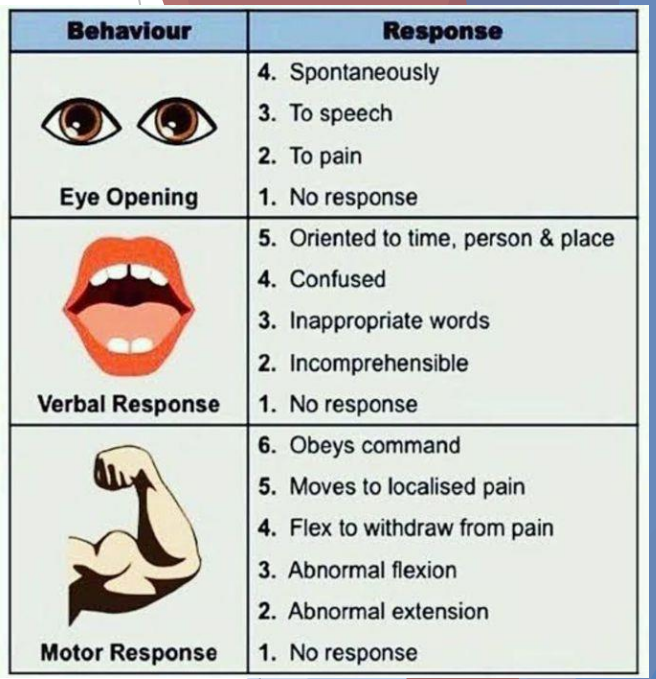
# Post Traumatic Amnesia




- Daily events not consistently recalled
- Objective measures (COAT, GOAT)
- Better predictor w DAI than focal injuries (Chadwick 1981)
- PTA >2 weeks: predict moderate to severe disability (Chadwick 1981)
- PTA < 24 hours: predict good outcome (Chadwick 1981)
- Similar predictive validity as length of time to follow commands and latter easier to measure (McDonald 1994)

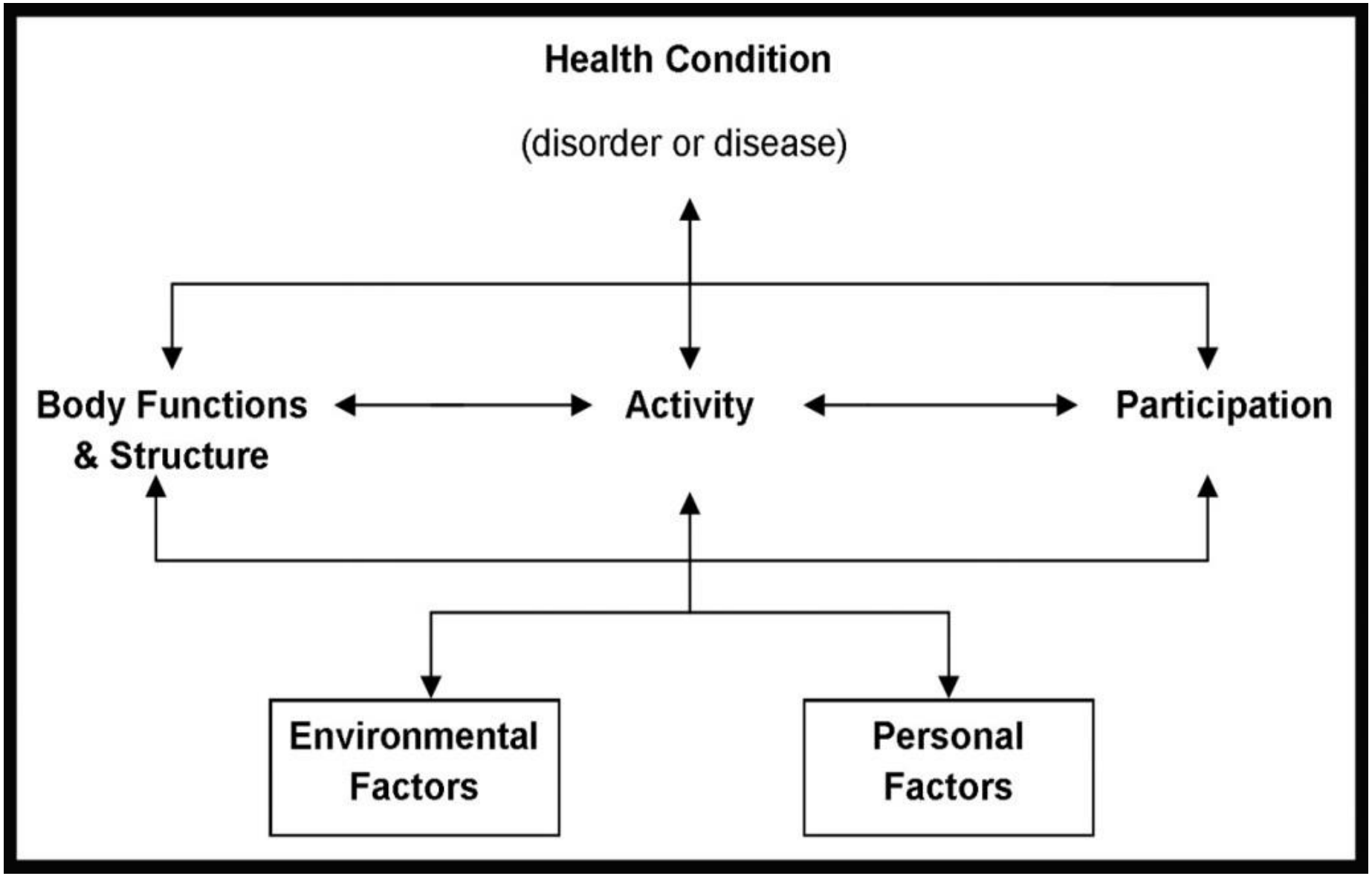
# Severity of TBI

| Criteria | Mild Complicated Mild | Moderate | Severe |
|---|---|---|---|
| **Structural Imaging** | Normal | Normal or abnormal | Normal or abnormal |
| **Loss of Consciousness** | < 30 minutes | 30 minutes to 24 hours | >24 hours |
| **Post-traumatic Amnesia** | 0–1 day | >1 and <7 days | >7 days |
| **Glasgow Coma Scale (best available score in 24 hours)** | 13–15 | 9–12 | 3–8 |




Orman JAL, Kraus JF, Zaloshnja E, et al. Epidemiology. In: Silver JM, McAllister TW, Yudofsky SC, editors. Textbook of traumatic brain injury. 2nd ed. Washington, DC: American Psychiatric Pub.; 2011. pp. 3–22

# Biopsychosocial Model of Disability: International Classification of Functioning, Disability and Health (WHO)



# WHO International Classification of Functioning, Disability, and Health

Body Functions/Structure
- Muscle Strength, coordination
- Spasticity/Dystonia
- Sensory changes
- Cognitive changes
- Headaches

TBI

**Health Condition**

(disorder or disease)

Activity
- Mobility
- Eating/Nutrition
- Communicating
- Dressing

**Body Functions & Structure** ↔ **Activity** ↔ **Participation**

- School or work
- Playing with friends
- Recreation
- Family roles

**Environmental Factors**

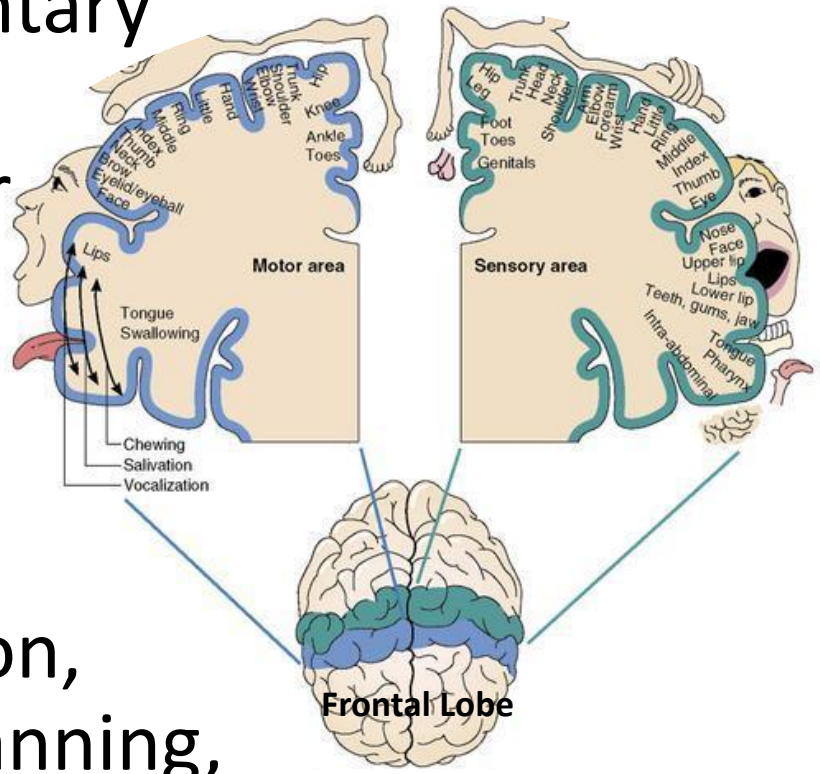
- Rural/urban
- Built environment
- Climate
- Social attitudes
- Policies and laws
- Access to services

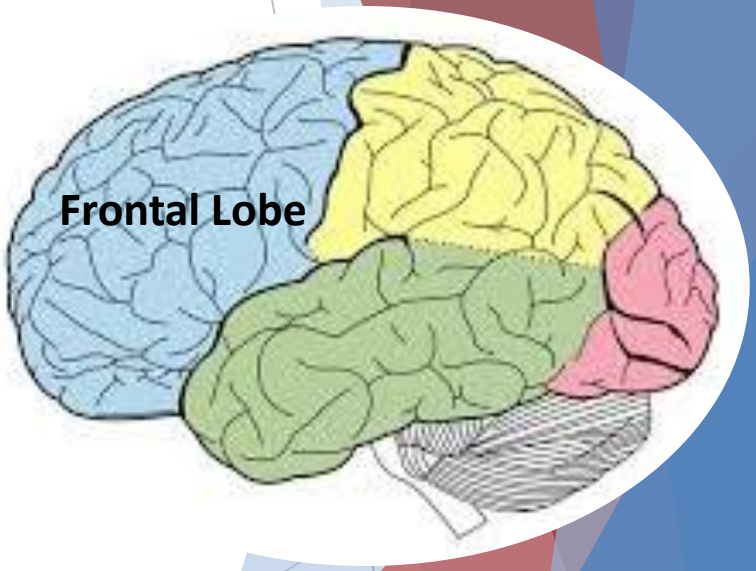
**Personal Factors**

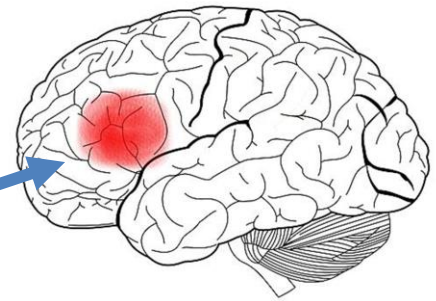
- Demographics
- Socioeconomic status
- Personal values

# Brain Structure and Function – Frontal Lobes

- Motor strip, supplementary cortex
- Basal: memory, bladder control
- Prefrontal: Executive functions
  - judgment, insight, reasoning, abstraction, mental flexibility, planning, sequencing, attention, concentration
- Language (left) – Broca's area



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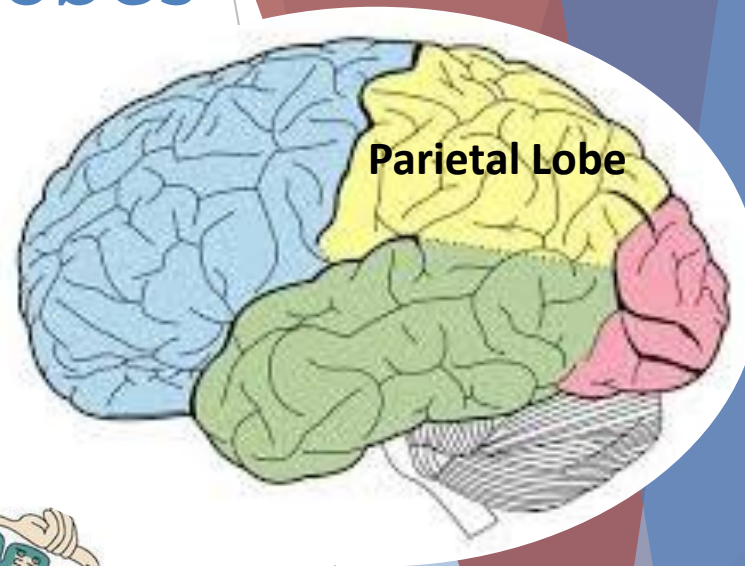
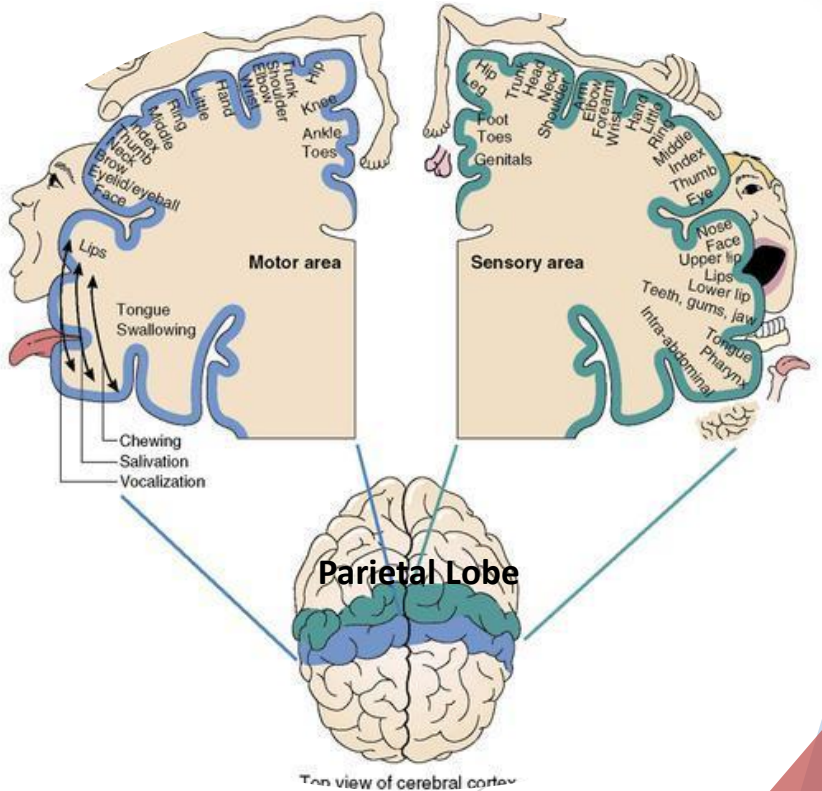
# Brain Structure and Function – Parietal Lobes

- Sensation, sensory processing
  - Two-point discrimination, proprioception, stereognosis, graphesthesia
- Left parietal: naming, reading, writing, calculation, L-R discrimination
- Right parietal: visuospatial perception, construct, neglect

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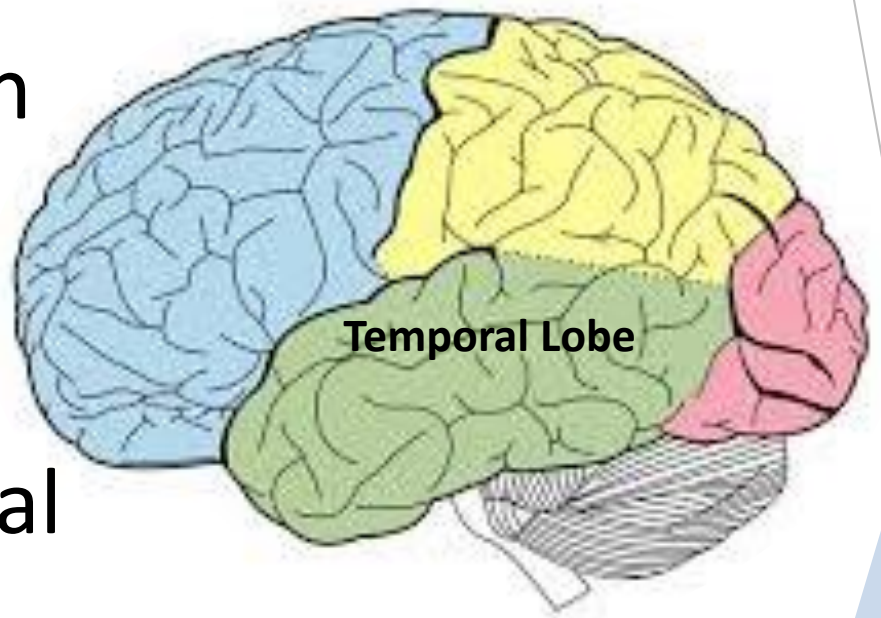
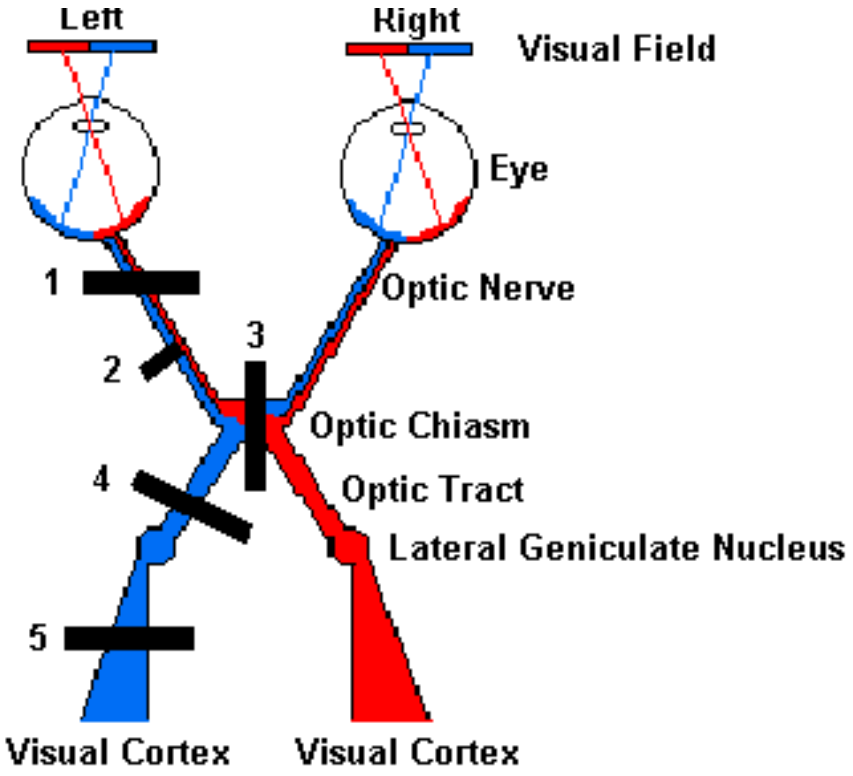
# Brain Structure and Function – Temporal Lobes

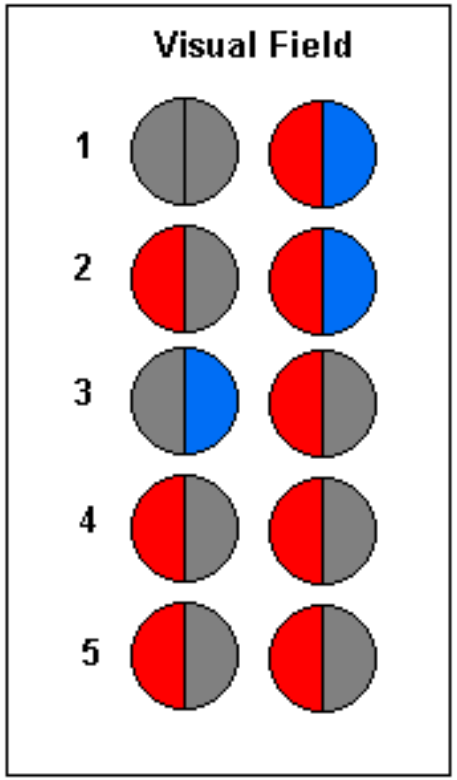
- Auditory cortex
- Language comprehension
- Prosody
- Left temporal: Verbal memory
- Right temporal: Nonverbal memory



# Brain Structure and Function – Occipital Lobes

- Vision
- Color





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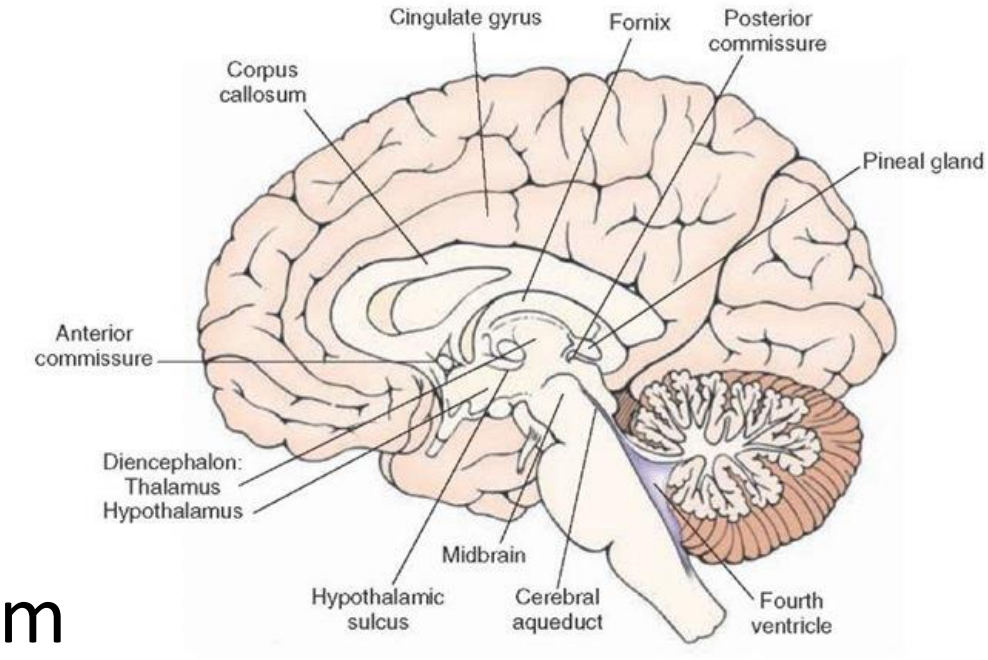
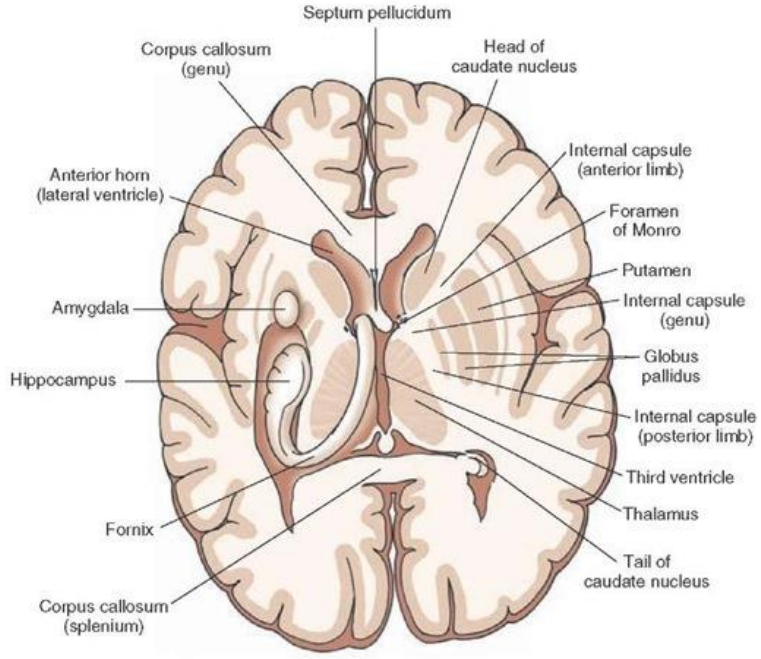
# Brain Structure and Function –Subcortex




Corpus Callosum
Basal ganglia, thalamus, internal capsule
Cerebellum
Brainstem
Cranial Nerves

# Impairments and limitations post TBI

Behavior:

- Acute: agitation, lability, confusion, ↓ tolerance to stimuli
- Late/chronic: fatigue, ↓ insight, impulsive, disinhibited, depression

Communication: aphasia, word finding, expressive organization, social/pragmatic communication

Cognition: attention, comprehension, speed of processing, memory, problem solving, abstraction

Motor: weakness, ↓ balance, ↓ coordination, hypertonicity

Social: social cues, tangential conversation, inappropriate acts, self-esteem, altered relationships

# Impairments and limitations post TBI

- May manifest immediately and persist
- May manifest immediately but improve or resolve (Jaffe 1995)
  - Improvement over first year, followed by chronicity
  - Children with deficits at 1 year unlikely to achieve parity with peers and showed little change in following 2 yrs
- **May be unapparent initially but become apparent as skills fail to emerge: "neurocognitive stall"**
  - **May not be recognized as associated with TBI**
- Continue to follow, monitor developmental transitions, update school plans (IEP/504)

# Objectives

1. Understand TBI pathophysiology and impact on brain function
2. Identify medical complications after TBI (focus on post-acute care)
3. Review outcomes and prognostic factors
4. Review inequities related to TBI

# Paroxysmal Sympathetic Hyperactivity

- "Storming"
- More common in hypoxic injuries (including secondary hypoxia)
- Episodic increased temperature, respiratory rate, heart rate, blood pressure; diaphoresis; agitation; posturing, hypertonia

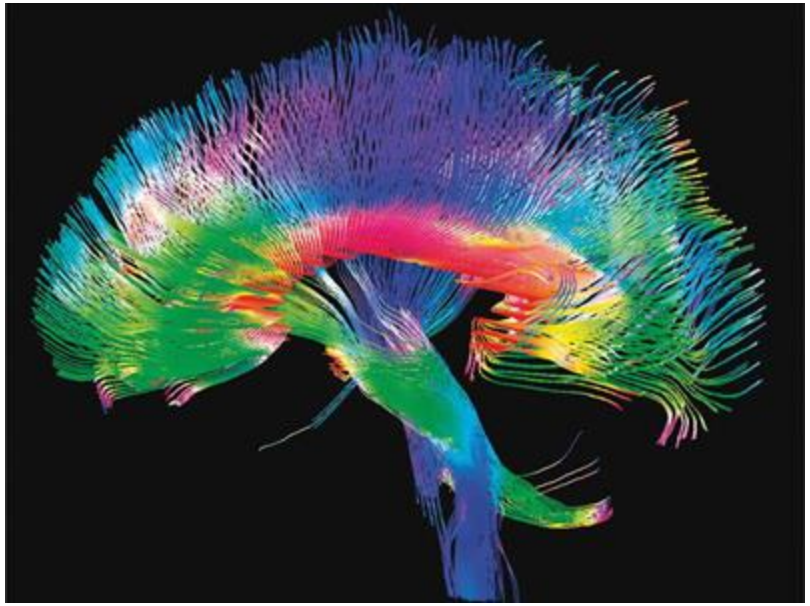


Table 1. Pediatric Paroxysmal Sympathetic Hyperactivity Scoring Reference

| | *0* | *1* | *2* | *3* |
|---|---|---|---|---|
| Heart rate | 1–4 years: <110<br>5–15 years: <100 | 1–4 years: 110–124<br>5–15 years: 100–119 | 1–4 years: 125–139<br>5–15 years: 120–139 | 1–4 years: ≥140<br>5–15 years: ≥140 |
| Respiratory rate | 1–4 years: <30<br>5–15 years: <25 | 1–4 years: 30–34<br>5–15 years: 25–29 | 1–4 years: 35–39<br>5–15 years: 30–34 | 1–4 years: ≥40<br>5–15 years: ≥35 |
| Systolic blood pressure | 1–4 years: <100<br>5–15 years: <120 | 1–4 years: 100–109<br>5–15 years: 120–129 | 1–4 years: 110–119<br>5–15 years: 130–139 | 1–4 years: ≥120<br>5–15 years: ≥140 |
| Diastolic blood pressure | 1–4 years: <65<br>5–15 years: <75 | 1–4 years: 65–72<br>5–15 years: 75–82 | 1–4 years: 73–79<br>5–15 years: 83–89 | 1–4 years: ≥80<br>5–15 years: ≥90 |
| Temperature | <37°C | 37–37.9°C | 38–38.9°C | ≥39°C |
| Sweating | Normal | Increased sweating | Localized diaphoresis | Generalized diaphoresis |
| Muscle tone increase | Absent | Mild increase | Neat increase | Generalized spasticity or opisthotonus |

- Rule out other etiologies
- Medication management directed at presenting symptoms; propranolol often used

Pozzi et al, 2014 J Neurotrauma

# Sodium abnormalities

- Posterior pituitary dysfunction:
  - SIADH - Syndrome of Inappropriate Antidiuretic Hormone – too much ADH, water retention, low Na
  - Diabetes insipidus – deficiency of ADH and too much water loss, high NA

| | SIADH | CSW | DI |
|---|---|---|---|
| Serum Na+ | < 135 mEq/L | < 135 mEq/L | > 145 mEq/L |
| Urine Na+ | > 25 mEq/L | > 40 mEq/L | < 25 mEq/L |
| Serum Osm | < 270 mOsm/kg | < 270 mOsm/kg | > 285 mOsm/kg |
| Urine Osm | > 300 mOsm/kg | > 300 mOsm/kg | < 300 mOsm/kg |
| Urine O/P | oliguria | polyuria | polyuria |
| Volume status | normal/high | low | normal/low |
| Plasma ADH | high | normal | low |
| Rx | Fluid restrict, give Na+, vaprisol, demeclocycline | Give volume, give Na+, fludrocortisone | Drink to thirst, DDAVP (central), HCTZ (nephrogenic) |

- Cerebral salt wasting; not part of HPA axis, neural effects on renal tubules
  - Lose sodium → lose volume

# Endocrine dysfunction after peds TBI

- Highly variable reported prevalence, 5% to 57% with endocrine dysfunction post-TBI in different peds TBI cohorts
  - Different injury severities, timing of assessment
  - No standardized screening

Cammisa et al. “Endocrine Dysfunctions After Pediatric Traumatic Brain Injury: Present Insights and Future Directions.” Children (Basel). 2025 Nov 3;12(11):1484. doi: 10.3390/children12111484. PMID: 41300601; PMCID: PMC12651762.

# Endocrine dysfunction after peds TBI

- Arizona Medicaid Records 2008-2014 queried for people ≤ 18 with TBI diagnosis followed by central endocrine diagnosis
  - Excluded those with endo dx prior to TBI onset
- Total of 107,458 diagnosed with TBI
- Overall prevalence of endo dx after TBI 10%
- Kids with TBI had 3x the risk of central endo dx compared to general population

Ortiz et al. “Epidemiology of Pediatric Traumatic Brain Injury and Hypothalamic-Pituitary Disorders in Arizona.” *Front Neurol* Jan 2020: Volume 10 - 2019 | https://doi.org/10.3389/fneur.2019.01410

# Endocrine dysfunction after peds TBI

- Growth Hormone deficiency → impaired linear growth velocity
  - TBI-related GHD symptom onset typically 6 to 24 months post-TBI
  - Small cohorts, 15 to 30% with GHD 1 year post-TBI

Cammisa et al. “Endocrine Dysfunctions After Pediatric Traumatic Brain Injury: Present Insights and Future Directions.” Children (Basel). 2025 Nov 3;12(11):1484

# Endocrine dysfunction after peds TBI

Gonadal axis dysfunction

- Hypogonadism
  - 4 to 9% prevalence after pediatric TBI (up to 29% in adult cohorts)
  - Usually transient in kids
- Central precocious puberty
  - Conflicting evidence if higher prevalence after TBI than general population (also associated w/ some non-traumatic acquired BI)
  - Inhibitory control of gonadotropin-releasing hormone activity may be compromised by TBI, leading to premature activation of HPG axis.

Cammisa et al. “Endocrine Dysfunctions After Pediatric Traumatic Brain Injury: Present Insights and Future Directions.” Children (Basel). 2025 Nov 3;12(11):1484.

# Endocrine dysfunction after peds TBI

- HPA dysfunction/Secondary adrenal insufficiency
  - Acute settings: hypotension, hypoglycemia, hyponatremia
  - Chronic (months after): fatigue, anorexia, recurrent abdominal pain, reduced stress tolerance
  - Small cohorts, 14-24% with low ACTH in acute phase
- Hypothyroidism
  - Small cohorts, 1-5% with central hypothyroidism 1 to 9 years after TBI

Cammisa et al. “Endocrine Dysfunctions After Pediatric Traumatic Brain Injury: Present Insights and Future Directions.” Children (Basel). 2025 Nov 3;12(11):1484

# Hypertonicity

- Spasticity

- Velocity dependent increase in tone
- Descending inhibitory pathways (brain/spinal cord level) not working fully
- Part of upper motor neuron syndrome
- Has to be felt, hands-on exam

- Dystonia

- Sustained or intermittent muscle contractions → abnormal movements, postures, or both
- Multiple mechanisms contribute to pathophysiology of dystonia, including injury to basal ganglia, thalamus
- Can observe without touching, can also elicit on exam, with movement

Becomes more pronounced as CNS myelinates

Can be exacerbated in times of illness, noxious stimuli

# When to treat hypertonicity

- Impacting function or ease of personal care
- Losing joint ROM or skin integrity impacting function/personal care
- Uncomfortable, prevent pain

….but some hypertonicity can support function (e.g., knee extensor hypertonicity and standing)

# Global versus focal tone management

**Global (enteral meds)**

- Many muscles involved
- Systemic, so side effects
- Can’t direct to specific muscle groups

**Focal (botulinumtoxin, phenol)**

- Limited muscles
- Injections, uncomfortable
- Training to administer
- Localization (ultrasound, EMG, anatomical)
- Sedation

# Global tone management options

| Medication | Mechanism | Side Effects |
|---|---|---|
| Baclofen* | GABA B agonist, pre- and post-synaptic, spinal cord level | Drowsiness (when↑ dose), constipation, unmasking weakness, renal excretion, ↓ seizure threshold, withdrawal |
| Benzodiazepines | GABA A agonist, pre-synaptic | Somnolence, memory impairment, also withdrawal, tolerance |
| Clonidine* | Alpha 2 presynaptic agonist | Dry mouth, hypotension, syncope, sedation, rebound effects |
| Tizanidine | Alpha 2 presynaptic agonist | Orthostatic hypotension, constipation, dry mouth, hepatotoxicity, sedation |
| Dantrolene | Inhibits calcium release from the sarcoplasmic reticulum | Hepatotoxicity, weakness, drowsiness |
| Gabapentin* | Selective inhibitor of voltage-gated calcium channels | Dizziness, somnolence |

*Recommended in AACPDM Dystonia Care Pathway

# Post-traumatic Epilepsy

- Seizure types
  - Immediate → within 24 hours
  - Early → 24 hours – 7 days
    - Incidence 20 – 39%
  - Late → anything after 1 week
    - Incidence 7-12%
- No evidence in pediatrics that preventing early seizures prevents late seizures
- AAPMR does not recommend use of prophylactic anti-epileptics for > 7 days

Alexander&Matthews, 2015

# Post-traumatic Epilepsy

- Diagnosis for post-traumatic epilepsy
  - 2 or more seizures in the late period (>7 days) after TBI
- Risk factors
  - Age <2 at time of injury
  - Lower GCS/more severe injury

- Choice of antiepileptic is determined by Neurology
  - Levetiracetam: can increase agitation, Vit B6 may help
  - Lamotrigine: can help with mood stabilization

Alexander&Matthews, 2015

# Objectives

1. Understand TBI pathophysiology and impact on brain function
2. Identify medical complications after TBI (focus on post-acute care)
3. Review outcomes and prognostic factors
4. Review inequities related to TBI

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# Factors associated w/ worse outcome after TBI

CT: Brain lesions (edema, SDH, ICH)

MRI: Deep brain lesions, volume loss, encephalomalacia

Initial CPP: <50 mm Hg

PTA: > 2 weeks

Initial GCS: <5

Time to follow commands: >12 days

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# Functional outcomes after pediatric TBI

## Disability persists among children hospitalized for TBI (Rivara et al, 2011)

- Reduction in quality of life, participation, self-care
- Improvements over time, but still impaired at 2-year follow-up
- QoL post-TBI higher in households with higher reported incomes

## Inpatient rehabilitation for pediatric TBI improves functional independence (Jimenez et al, 2015)

- Medicaid insurance associated with lower function at discharge
- Inpatient rehab at children's hospital (rather than general IPR that accepts kids) associated with better cognitive function at discharge

# Functional outcomes after pediatric TBI

- TBI during early childhood and effect on daily functioning (Wade et al, 2016)
  - Children with TBI sustained between ages 3 and 7 had impairments in daily activities 7 years after TBI
  - More severe TBI associated with poorer function
  - Parenting practices and home environment moderated relationship between injury severity and function
    - Permissive and authoritative parenting, less advantaged home environments associated with amplified adverse effect of TBI
- In a cohort of children hospitalized for TBI: (Slomine et al. 2006)
  - Majority have physical (56%) and cognitive (70%) impairments 1 year after injury
  - Only 39% received injury-related outpatient services
    - A separate cohort extended to 2yr follow-up; mod-severe TBI 58% physical dysfunction, 55% cognitive dysfunction (Fuentes et al. 2018)

# Health Disparities/Inequities in pediatric TBI

## Who gets TBIs?

Pediatric TBI rates clustered disproportionately in socioeconomically disadvantaged areas

- Kazemi et al “Variation of demographic and socioeconomic factors associated with pediatric traumatic brain injury: a geospatial analysis.” J Neurosurg Pediatr. 2025 Aug 8;36(5):630-640

Rural children more likely to have higher trauma/TBI severity

- Yue et al. Brain Sci. 2020 Mar; 10(3):135

Children with ADHD – rate of pre-TBI ADHD 16% in kids with TBI, only 10.8% in gen population

- Asarnow et al. JAMA Peds 2021 Oct 1;175(10):1009-1016

Overall hard to really know, and many studies aren’t describing different pediatric populations in detail

## Health Disparities/Inequities in pediatric TBI

# Do children with TBI have equitable outcomes?

Mortality for children hospitalized for TBI: 22.8% for Black children v 15.6% among White children

- Data source: Trauma Quality Improvement Program registry 2014-2017. (Piatt J. J Neurosurg. Published online 31 July 2020.)

Mortality for children hospitalized for TBI: Uninsured children 45% higher mortality than those with private insurance

- Data source: Healthcare Cost and Utilization Project State Inpatient Database (Greene et al. Arch Phys Med Rehab. 2014 Jun; 95(6):1148-1155)

Black children more likely to have functional impairment, more likely to discharge to inpatient rehabilitation after TBI than white children

- Data source: National Trauma Databank (Haider et al. J Trauma. 2007 May; 62(5):1259-62)

# Health Disparities/Inequities in pediatric TBI

- Do children with TBI have equitable access to rehabilitation care?

Uninsured children 68% less likely than those with private insurance to discharge to inpatient rehab after TBI hospitalization

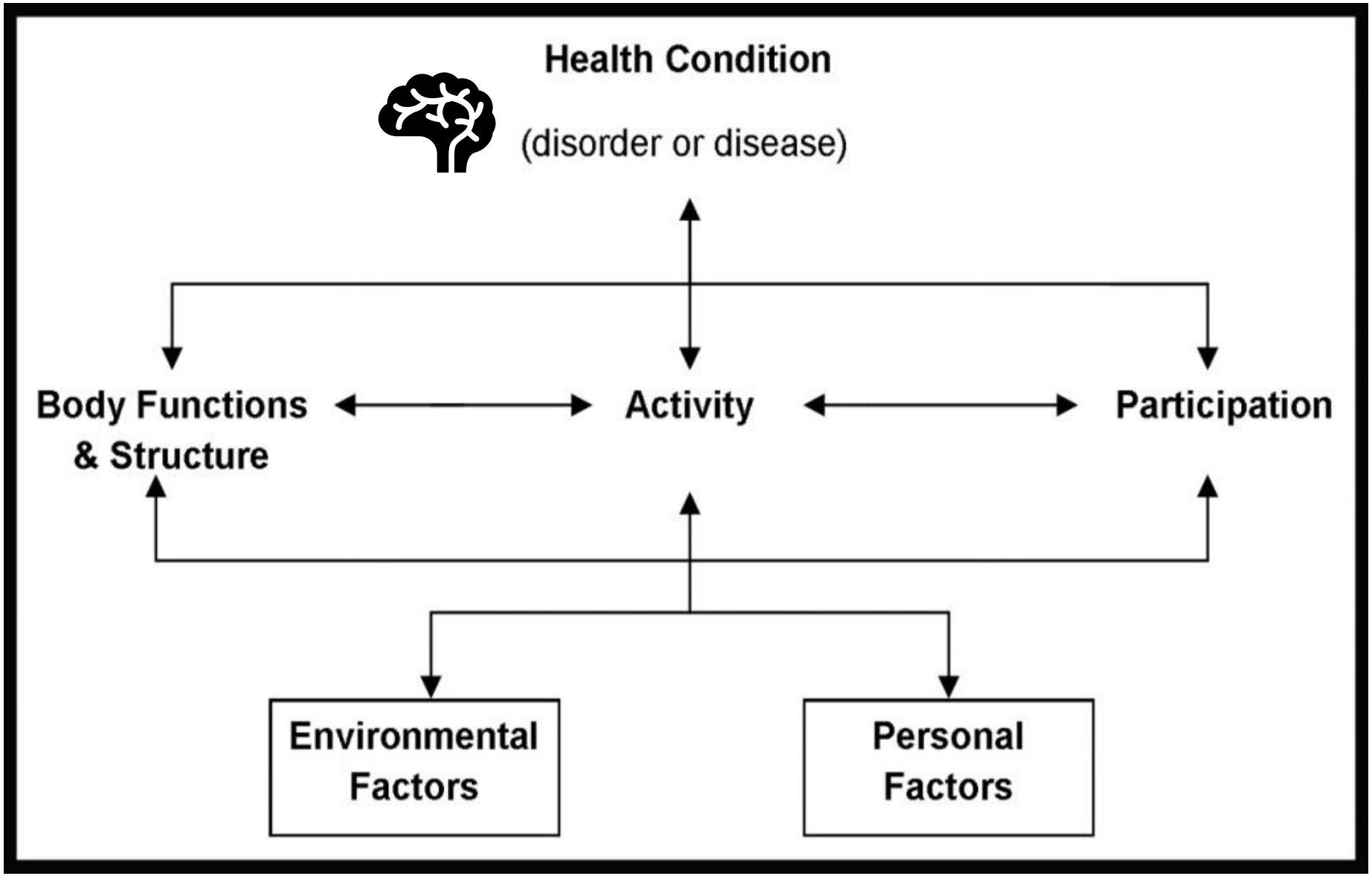
- Data source: Healthcare  Cost and Utilization Project State Inpatient Database (Greene et al. Arch Phys Med Rehab. 2014 Jun; 95(6):1148-1155)

Outpatient rehab therapy clinics in WA 33 to 42% more likely to accept private than public insurance

Wait to first appointment was longer at clinics accepting public insurance

- (Fuentes et al. Arch Phys Med Rehab. 2017 Sep; 98(9):1763-1770)

# Biopsychosocial Model of Disability: International Classification of Functioning, Disability and Health (WHO)



# Progression of care after TBI

**Brain Injury**

- Acute trauma hospital care
- +/- surgery

**Inpatient Rehabilitation**

- Intensive therapies to improve function, safety
- Medications if appropriate
- School reintegration team (hospital teacher, SLP, Neuropsych)

**Home**

- Adjusting to being at home with impairments
- Outpatient rehab therapies

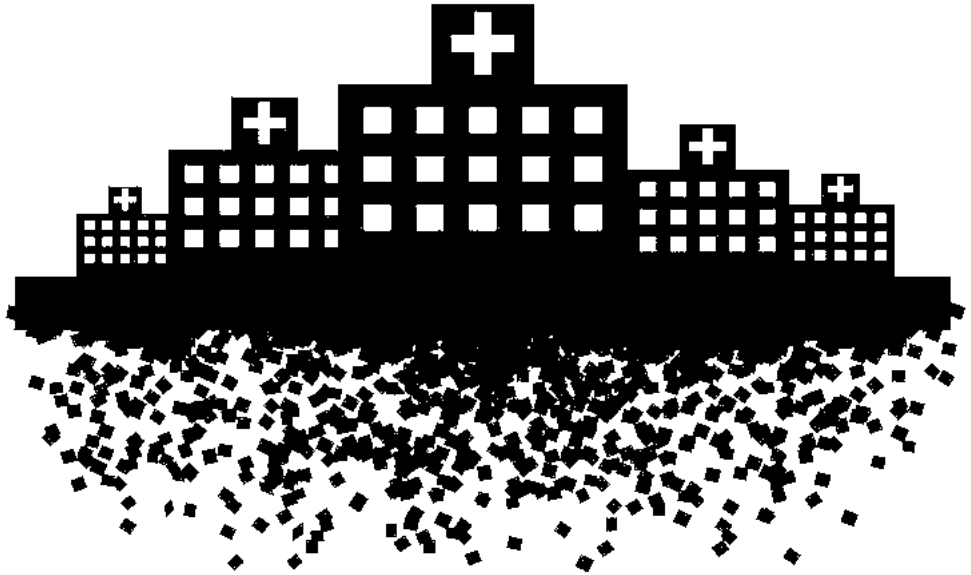
**Community Reintegration**

- Return to school; new IEP or 504 plan?
- Leisure and recreation
- Social relationships

# Peer interactions

- Disability type influences peer behavior
- Disability types more associated with peer rejection: invisible disabilities, impacting participation, affecting social problem-solving and emotional regulation
- Children with communication, behavioral, social/emotional problems, motor limitations found to have fewer friends
  - Race and age also impacted peer acceptance

Woodgate et al. “How do peers promote social inclusion of children with disabilities? A mixed-methods systematic review” Disabil Rehabil, 2020; 42(187):2553-2579




INDIVIDUALS WITH DISABILITIES EDUCATION ACT
50
YEARS
1975-2025

# School Reintegration

- Medical/Rehab recommendations can impact full school reintegration
  - Limit time in school/partial day attendance to reduce exacerbating TBI symptoms
  - Ongoing rehabilitation therapy and medical appointments = missed school
    - But if not in the environment, how can you belong?
  - "Two feet on the ground" or other activity recommendations to reduce risk of repeat head injury
    - Can lead to child not participating in recess, staying inside
    - Being treated differently can lead to "other-ing"
  - Medical providers not likely to update recommendations without repeat evaluation of child = delays
- Communication between school and medical teams often occurs through family; additional burden on family

# Belonging and influence of institutions

- How might schools and health care institutions promote belonging?
  - Social-emotional learning curricula
  - Health care professionals giving anticipatory guidance on how to approach questions from peers
  - Group-based cognitive therapy that also functions as pragmatic/social communication skills
  - Include representation of children with multiple types of disabilities in literature, classroom art, etc.
  - Support inclusive recreation programs

# Questions?

TBI-BH ECHO