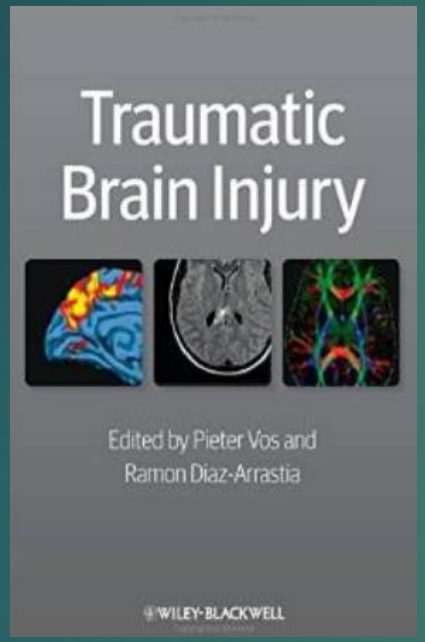
Neuropsychology of Traumatic Brain Injury

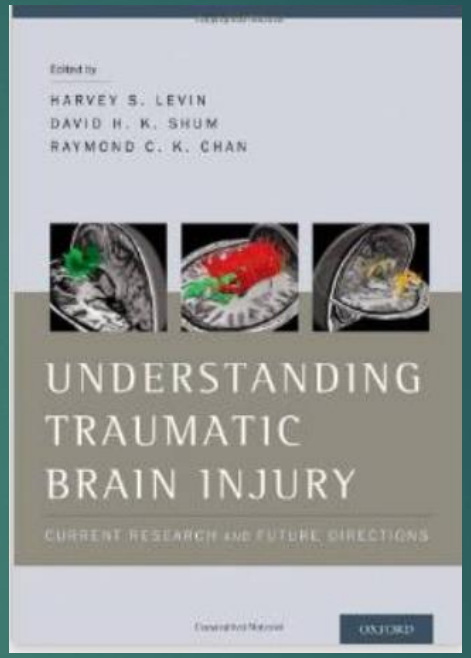
Charles J. Vella, PhD

2018

Reference Library



Pieter Vos (Editor),
Ramon Diaz-Arrastia
2015

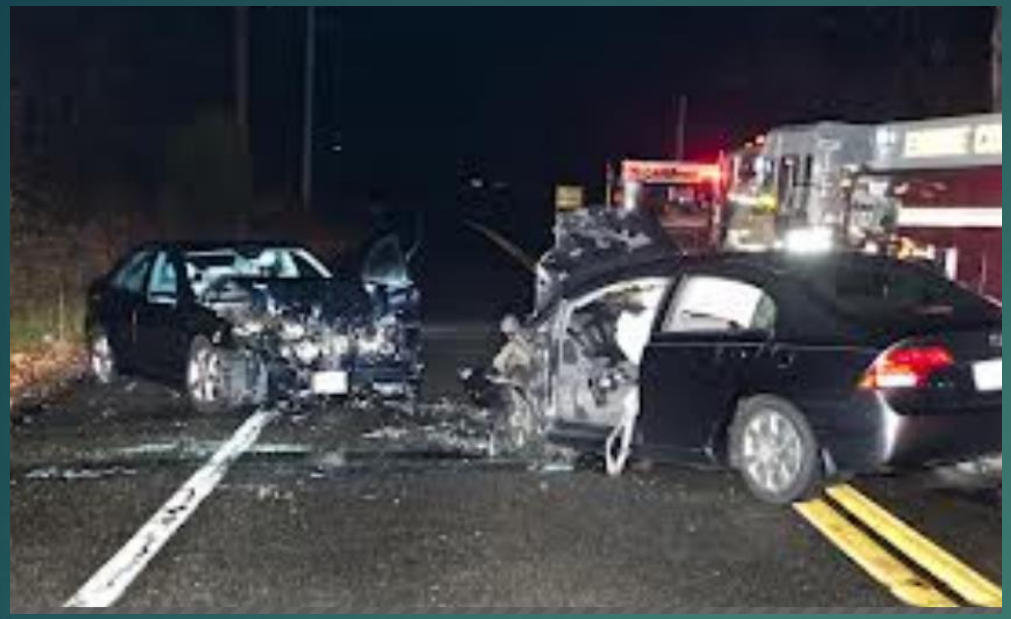


Harvey Levin (Editor),
David Shum,
Raymond Chan, 2014

- Buckle up!
- Wear your helmet!


BIGBMXMAGAZINE


UFC
UFC




RESPECT


26

The Unknown Disorder

- Until recently, TBI has received very little publicity and has been largely unknown as a neurological disorder.
- Recent news about TBI injuries as a consequence of military service (30%) and the professional football legacy of chronic traumatic encephalopathy has begun to change this.
- Research articles 1960-1999 = 4548; between 2000-2012 = 19,308

Traumatic Brain Injury

- A TBI is caused by a bump, blow or jolt to the head or a penetrating head injury that disrupts the normal function of the brain.
- The severity of a TBI may range from “mild” (a brief change in mental status or consciousness) to “severe” (an extended period of unconsciousness or amnesia after the injury, or death).
- Not a one time event, but a chronic disease process

- Traumatic Brain Injury is the most common cause of neurological injury.

TBI

- The most common mechanism of TBI in civilians is closed head trauma due to sudden acceleration & deceleration of the head & blunt impact by an external mechanical force.

- CHI results in focal & diffuse injuries in the brain due to rotational acceleration imparted to the brain & more localized impacts from blunt trauma.

- Blast-related brain injuries in US military is one of the most frequent injuries

Definition of Traumatic Brain Injury

- Closed head injury (CHI) – Skull intact, brain not exposed. Most common TBI

- Penetrating head injury (PHI) – Open head injury where skull and dura are penetrated by an object.

- Vascular insults (due to stroke, anoxia, etc.)

CDC TBI Definition

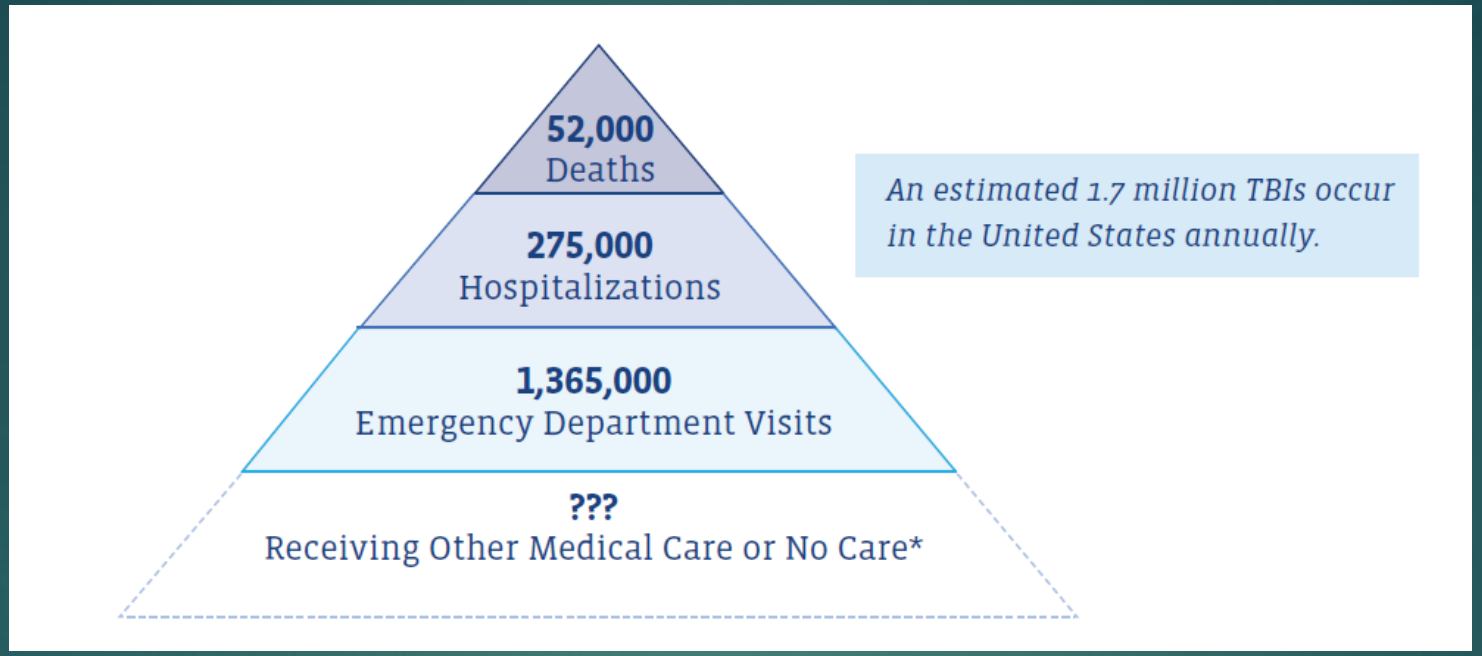
- Craniocerebral trauma, specifically, an occurrence of injury to the head (arising from blunt or penetrating trauma or from acceleration & deceleration forces) that is associated with any of these symptoms attributable to injury: decreased level of consciousness, amnesia, other neurologic or neuropsychological abnormalities, skull fracture, diagnosed intracranial lesions, or death.

- Thurman DJ, Sniezek JE, Johnson D, et al., Guidelines for Surveillance of Central Nervous System Injury. Atlanta, GA: National Center for Injury Prevention and Control, Centers for Disease Control and Prevention, US Department of Health and Human Services, 1995.

DSM-5: Major or Mild NCD due to TBI

- A. Criteria met for major or mild NCD
- B. Evidence of a TBI - an impact to the head or other mechanisms of rapid movement or displacement of the brain within the skull, with 1 or more of the following:
  - 1. LOC
  - 2. PT amnesia
  - 3. Disorientation & confusion
  - 4. Neurological signs (e.g., neuroimaging demonstrating injury; new onset seizures; marked worsening of a preexisting seizure disorder; visual field cuts; anosmia; hemiparesis)
- C. Neurocognitive disorder presents immediately after the occurrence of the TBI or immediately after recovery of consciousness and persists past the acute post-injury period

TBI Epidemiology per year: 1.7 M per year



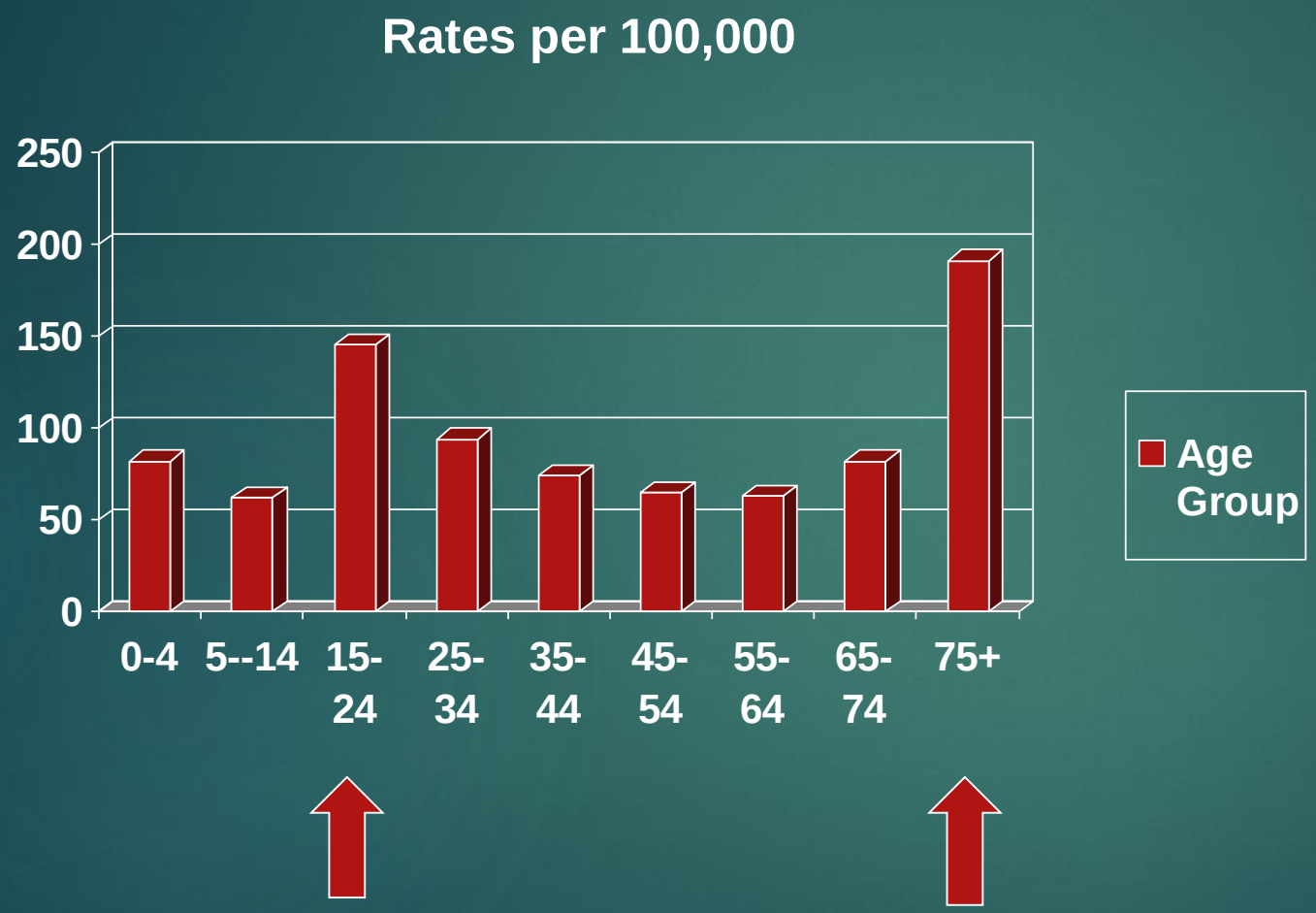
10 million worldwide hospitalized or killed

Disabled: 80,000 – 90,000: 6% Long-term Disability

1.4% of all ER visits

Of all the injury related deaths in the United State: 4.8% of all injuries seen in ED visits and 15.1% of all hospitalizations; 31% of deaths

Rates of TBI hospitalization and death by age group



Incidence

- #1 cause of mortality and disability in the under 45 of age
- #1 cause of seizure disorders
- Risk highest in infants, 15-24, and 75+
- Males 1.5x greater
- 50% of all injury deaths;
- leading cause of death & disability in young adults

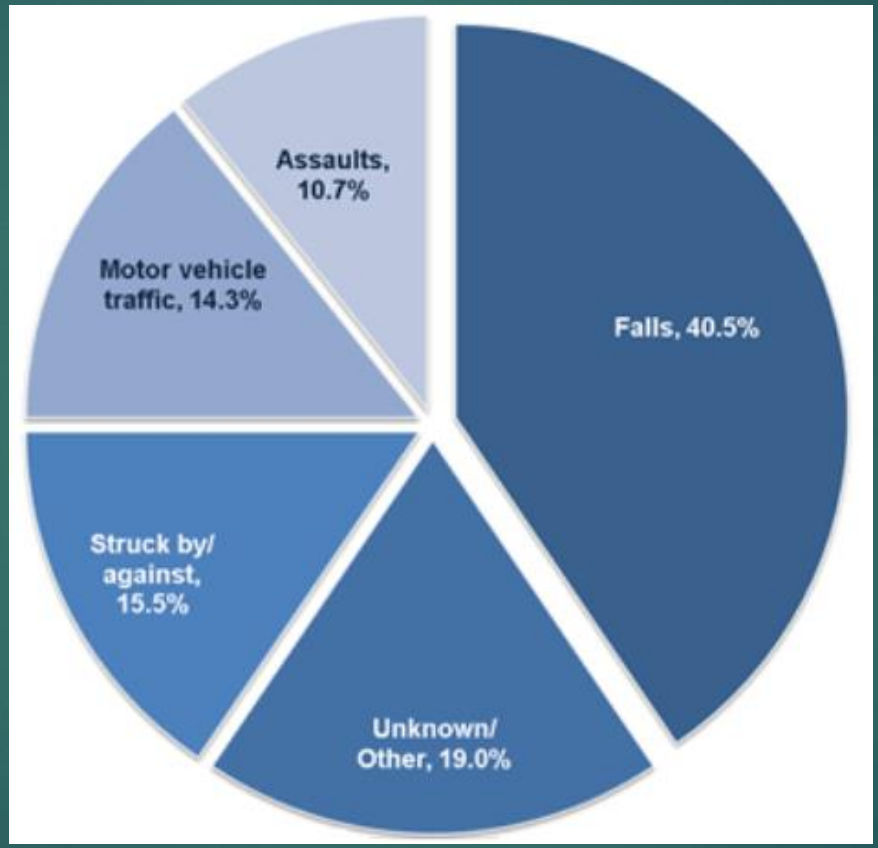
Epidemiology

- Mean age in years = 36
- Gender = 75% male
- Race = 57% white; 31% African-American
- Marital status = 71% unmarried
- Education = 12; 36% without HS degree
- Average GCS score upon admission = 7
- 80% of injuries are mTBI

Epidemiology

- 5.3 million Americans currently living with TBI related disability (1.5%)
- 22% of TBI result in death
- 60% employed at time of injury
  - Only 25% employed 1-year post injury

CDC: Leading causes of TBI



TBIs due to falls, MVAs, & assaults = 84% of all TBI related hospital visits;
TBI mortality declined 11% from 1989-1998 due to seat belt laws

Leading Causes of TBI

- MVA (14%; CA 80% of pedi);
  - Pedal-driven vehicle accidents (3%)
- Assault/Violence (11%); 57% CHI, 23% penetration
- Falls (41 %; NY, 55% pedi);
- Collisions: Sports (300,000 per year)
  - 5-10% of ski accidents

Violence & TBI

- Firearms (6%); Suicide (1%);
- 2/3rds of firearm related TBI are suicidal
- 44% of fatal TBI are due to gunshot
- 9% of non-fatal TBI due to violence
- Who suffers TBI due to violence:
  - Older than other types of TBI (modal age = 40)
  - 3 times more likely to be a minority
  - More likely single/divorced & living alone
  - Lower education levels & more unemployment

Violence Induced TBI: Recovery

- Higher ETOH at admission than nonviolent
- Less severe injuries (CGS = 9.6 vs. 8.3)
- Shorter lengths of stay both acute & rehab
- Payor is Medicaid in 58% of cases, which is 3X that of the non-violently injured
- Higher rates of unemployment & ETOH use post injury than non-violently injured.

41 year Swedish Study: Psych & TBI

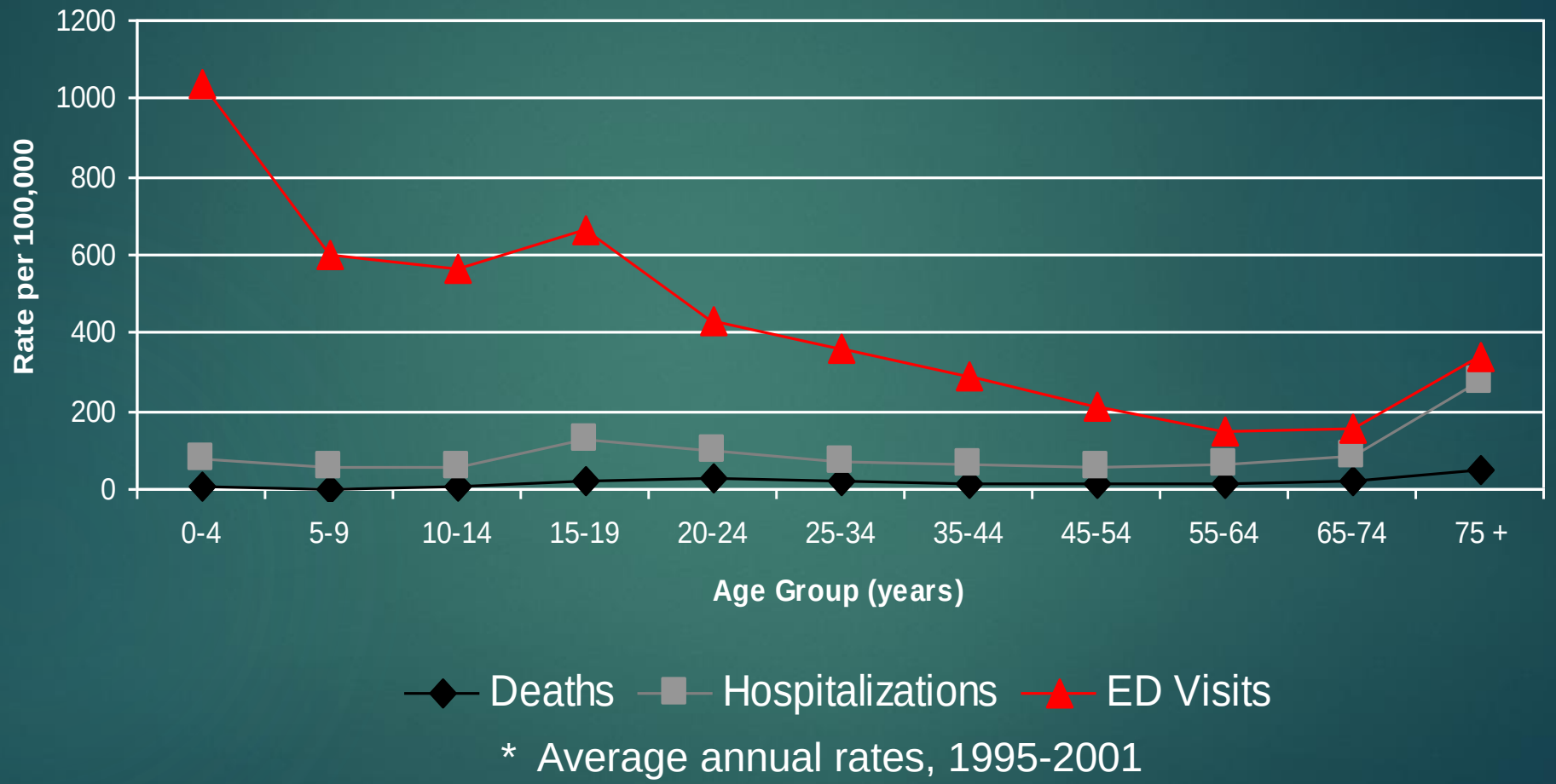
- N = 218T
- All-cause mortality and suicide were 3-fold higher than in the general population.
- TBI is an independent risk factor for premature mortality, particularly for suicide, injuries, and assaults
- Strong associations reported between premature deaths and both psychiatric disorder and substance abuse, with 61% of premature deaths in TBI patients having a lifetime psychiatric or substance abuse diagnosis.

Causes by age

- Falls in age 1-5 and 65+
- 140,000 bicycle TBI
- 136,000 concussions occur per academic year in high schools
- Alcohol: 49% of TBIs; 41% of fatal TBI; 7% of all crashes; 7% of falls; 60% of MVA; >70% of MVA comas; 1 person injured every 2 min where alcohol present

TBI in the United States

TBIs by Age Group* 1995-2001

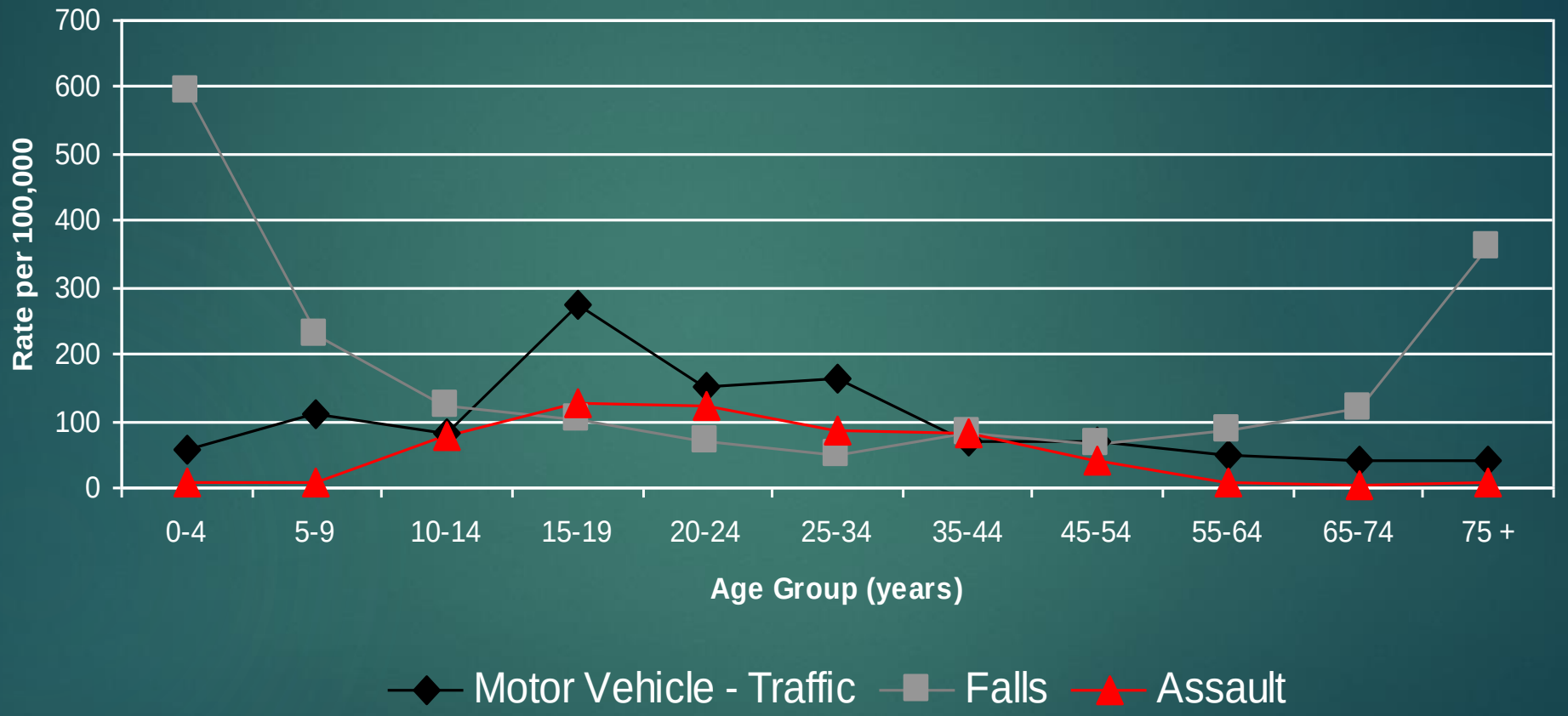


* Average annual rates, 1995-2001

Bimodal distribution with very young and people more than 64 y/o at risk;
w/ 75 y/o have highest risk of hospitalization and death

TBI in the United States

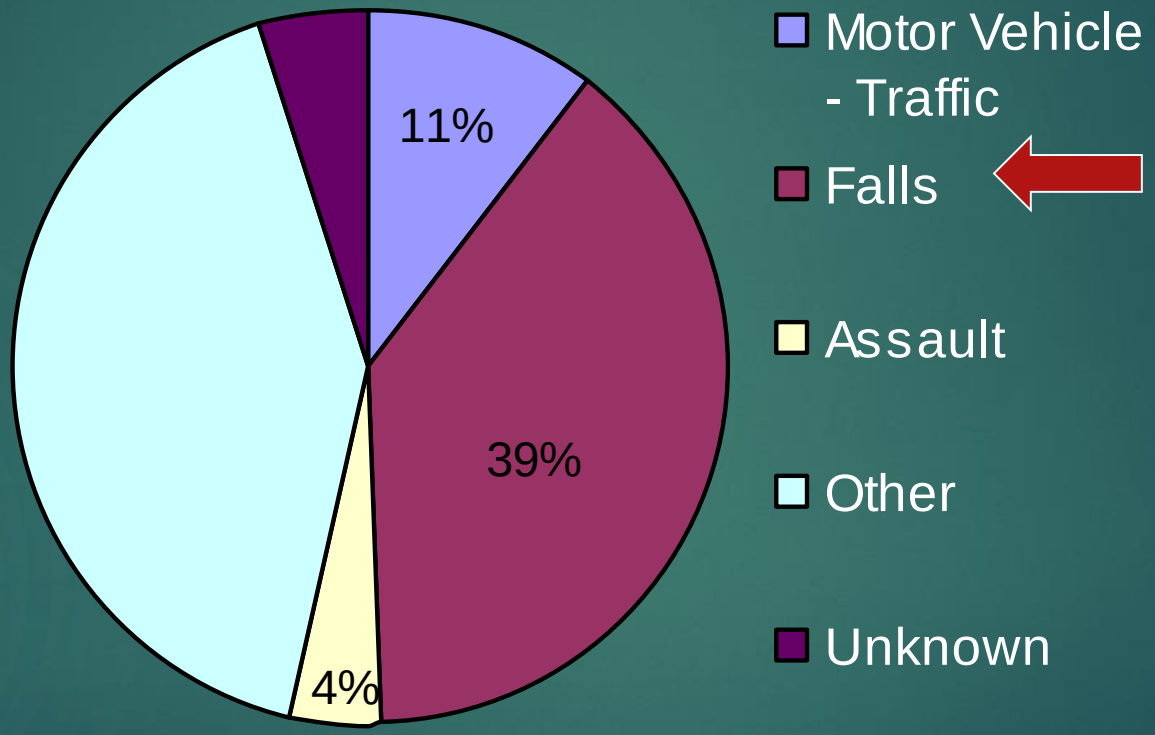
TBIs by Cause* 1995-2001



- Average annual rates, 1995-2001; ED visits, hospitalizations, and deaths combined;
  - Falls are leading cause of TBI

TBI in the United States (CDC)

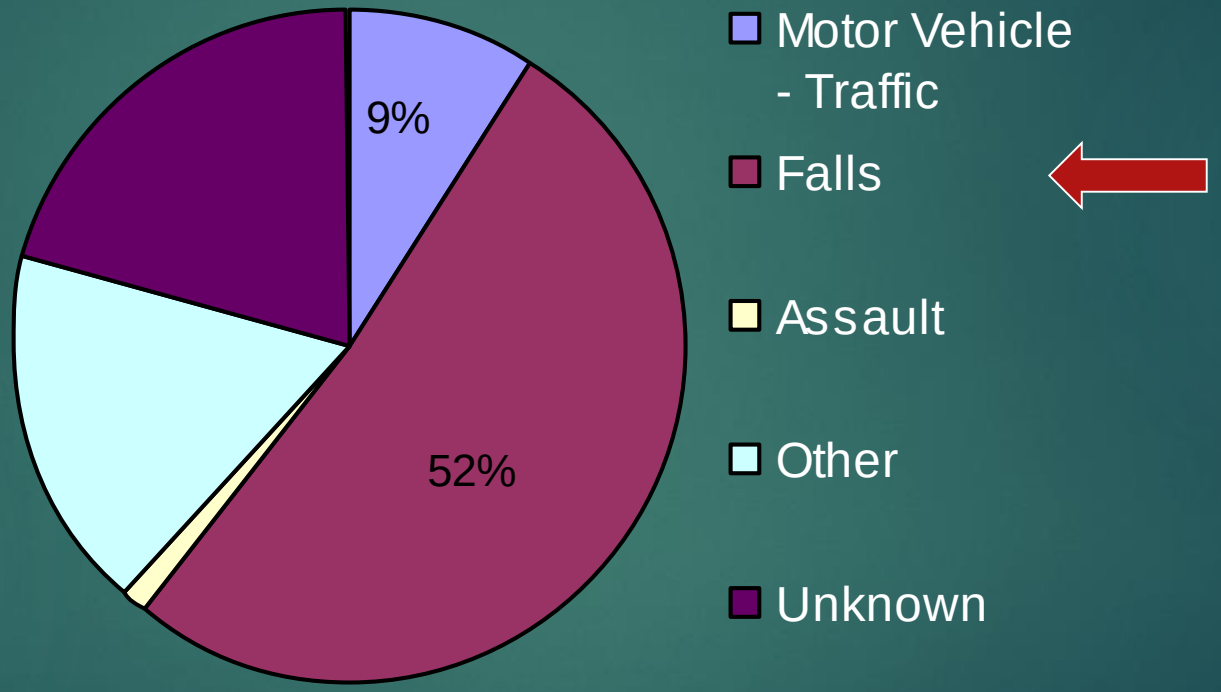
TBIs Among Children (0-14 years) by Cause 1995-2001



* Average annual percents, 1995-2001; ED visits, hospitalizations, and deaths combined

TBI in the United States (CDC)

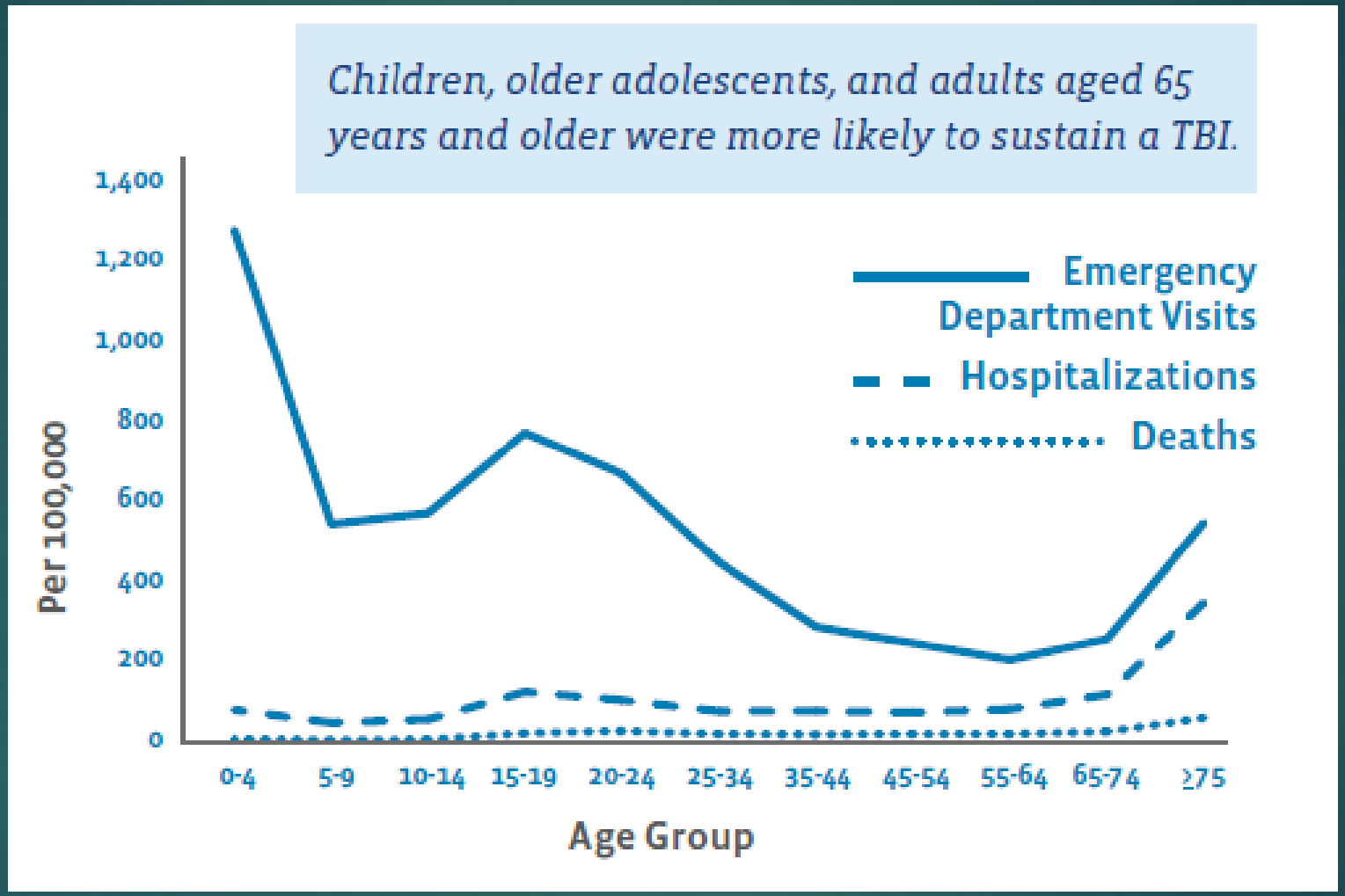
TBIs Among Older Adults (>65 years) by Cause 1995-2001



* Average annual percents, 1995-2001; ED visits, hospitalizations, and deaths combined

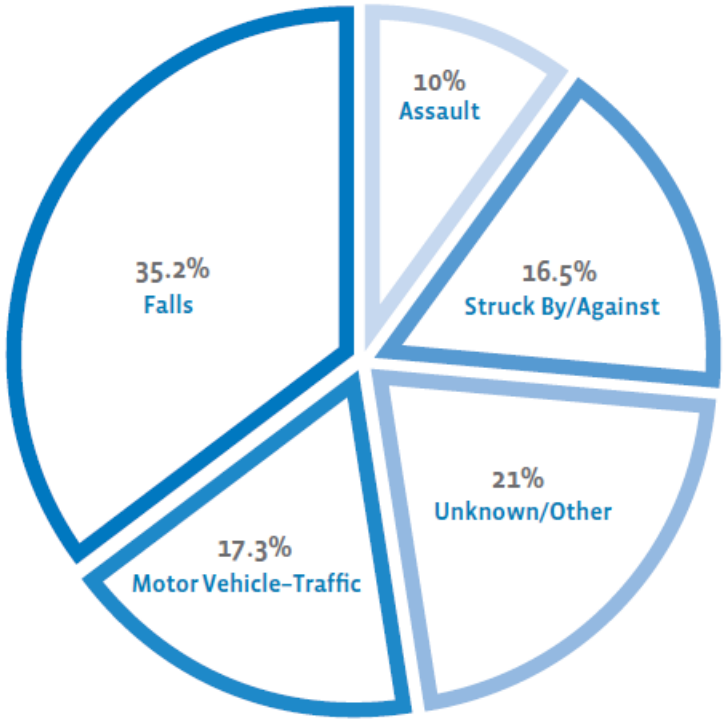
New 2002-2006 CDC TBI Report

Highest in Kids, teens, 65+



Correction: Hospitalization (350/100K) & death (100/100K) rates in 75+ is significantly higher than all other groups

FIGURE 5: Estimated Average Percentage of Annual Traumatic Brain Injury-Combined Emergency Department Visits, Hospitalizations, and Deaths, by External Cause, United States, 2002–2006



Falls and TBI: Child and Adolescents vs. 65+ Adults

FIGURE 6: Estimated Average Percentage of Annual Traumatic Brain Injury-Combined Emergency Department Visits, Hospitalizations, and Deaths Among Children 0 to 14 Years, by External Cause, United States, 2002–2006

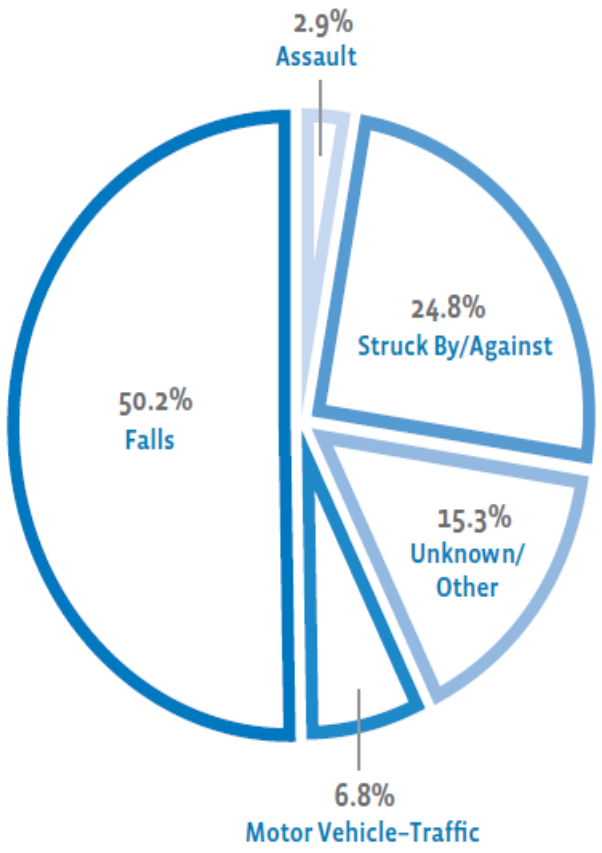
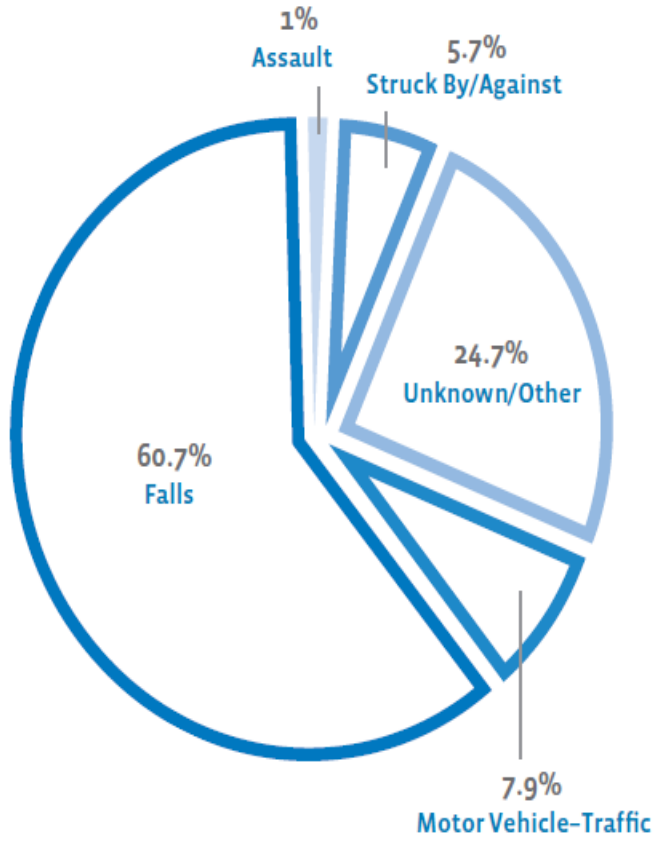


FIGURE 7: Estimated Average Percentage of Annual Traumatic Brain Injury-Combined Emergency Department Visits, Hospitalizations, and Deaths Among Adults 65 Years and Older, by External Cause, United States, 2002–2006



Skiing: Helmets!!

- About 5% to 10% of skiing accidents result in TBIs.
- Falls are the most common mechanism of injury, followed by skier-vs.-skier, then skier-vs.-tree. But skier-vs.-tree was far and away the most lethal.
- In 10 years, very few of the on-slope fatalities were helmeted (about 6%).
- Helmets reduced the risk of brain injury by 74%, and the risk of fatality by 80%.
- Only 1 of 19 helmeted snowboarders have a positive CT scan, compared to 25% of unhelmeted skiers and boarders.

TABLE 1. Estimated annual number of emergency department (ED) visits for all nonfatal injuries and nonfatal traumatic brain injuries (TBIs) related to sports and recreation activities, for all ages and for ages 5–18 years, by activity — National Electronic Injury Surveillance System–All Injury Program, United States, 2001–2005

	All ages					Ages 5–18 yrs						
	All injuries		TBIs			All injuries		TBIs			% of all injuries in age group[¶]	% of TBIs in age group[**]
Activity	No.[*]	(95% CI[†])	No.	(95% CI)	% of all injuries[§]	No.	(95% CI)	No.	(95% CI)	% of all injuries		
Bicycle	524,692	(434,500–614,883)	40,424	(25,293–55,555)	7.7	309,752	(263,097–356,407)	23,405	(16,860–29,950)	7.6	59.0	57.9
Football	398,369	(349,189–447,550)	22,689	(18,102–27,276)	5.7	320,542	(277,524–363,560)	20,293	(16,255–24,332)	6.3	80.5	89.4
Playground	226,091	(186,257–265,925)	16,130	(11,004–21,256)	7.1	160,621	(133,056–188,186)	10,414	(7,185–13,644)	6.5	71.0	64.6
Basketball	603,239	(528,121–678,357)	14,680	(10,782–18,579)	2.4	380,245	(328,566–431,924)	11,506	(8,528–14,485)	3.0	63.0	78.4
All-terrain vehicle	132,702	(101,439–163,964)	11,199	(5,856–16,542)	8.4	53,447	(40,562–66,332)	5,220	(2,462–7,979)	9.8	40.3	46.6
Baseball	163,215	(136,676–189,755)	10,103	(7,414–12,792)	6.2	113,649	(93,833–133,465)	7,433	(5,440–9,426)	6.5	69.6	73.6
Soccer	169,373	(120,915–217,830)	9,371	(5,753–12,989)	5.5	122,731	(87,045–158,417)	7,667	(4,747–10,588)	6.2	72.5	81.8
Horseback riding	74,096	(55,856–92,336)	8,650	(4,496–12,803)	11.7	20,903	(15,972–25,834)	2,648	(1,593–3,704)	12.7	28.2	30.6
Swimming/diving	99,514	(74,848–124,179)	5,878	(3,398–8,357)	5.9	52,684	(41,271–64,098)	3,846	(2,325–5,367)	7.3	52.9	65.4
Skateboard	109,550	(72,553–146,546)	5,292	(3,124–7,460)	4.8	86,765	(60,612–112,918)	4,408	(2,561–6,255)	5.1	79.2	83.3
Hockey[††]	70,548	(34,650–106,445)	5,194	(2,239–8,149)	7.4	45,127	(20,554–69,700)	4,111[§§]	(1,523–6,699)	9.1[§§]	64.0	79.1[§§]
Moped/minibike/ dirt bike[¶¶]	71,987	(55,405–88,569)	4,736	(3,023–6,449)	6.6	33,868	(25,092–42,643)	2,523	(1,677–3,370)	7.5	47.0	53.3
Softball	108,014	(87,021–129,007)	4,277	(2,967–5,588)	4.0	45,153	(35,611–54,695)	1,797	(1,171–2,423)	4.0	41.8	42.0
Exercise	230,966	(193,241–268,691)	4,163	(2,368–5,958)	1.8	62,226	(52,865–71,587)	1,469	(863–2,076)	2.4	26.9	35.3
Miscellaneous ball games[***]	89,469	(71,798–107,140)	3,814	(2,211–5,416)	4.3	59,338	(46,559–72,116)	2,470	(1,634–3,306)	4.2	66.3	64.8
Combative[†††]	77,088	(62,232–91,944)	3,682	(2,537–4,827)	4.8	46,488	(36,219–56,757)	2,456	(1,627–3,284)	5.3	60.3	66.7
Scooter	67,197	(53,340–81,054)	3,534	(2,558–4,511)	5.3	52,355	(42,058–62,652)	2,790	(2,061–3,518)	5.3	77.9	78.9
Gymnastics[§§§]	93,603	(77,169–110,037)	2,951	(2,038–3,864)	3.2	64,507	(51,567–77,447)	2,339	(1,579–3,100)	3.6	68.9	79.3
Toboggan/sled	32,279	(20,161–44,397)	2,687	(1,411–3,964)	8.3	21,292	(13,340–29,245)	1,873	(1,011–2,736)	8.8	66.0	69.7
Golf[¶¶¶]	40,578	(30,313–50,843)	2,687	(1,783–3,591)	6.6	13,058	(10,092–16,023)	1,125	(681–1,569)	8.6	32.2	41.9
Skating, ice	23,214	(15,810–30,618)	2,411	(1,546–3,275)	10.4	14,387	(9,666–19,108)	1,545	(909–2,181)	10.7	62.0	64.1
Trampoline	93,389	(73,452–113,325)	2,131	(1,382–2,881)	2.3	73,029	(57,219–88,839)	1,545	(1,013–2,078)	2.1	78.2	72.5
Skating, in-line	46,665	(32,989–60,342)	1,610	(982–2,238)	3.5	33,109	(23,692–42,525)	1,142	(742–1,542)	3.4	70.9	70.9
Skating, other[****]	53,795	(41,434–66,156)	1,457	(989–1,925)	2.7	36,609	(28,152–45,066)	1,087	(749–1,425)	3.0	68.1	74.6
Amusement attractions[††††]	21,273	(15,270–27,275)	1,391	(825–1,957)	6.5	10,947	(7,698–14,196)	859	(467–1,250)	7.8	51.5	61.7
Go-cart	16,951	(13,235–20,667)	1,243	(802–1,684)	7.3	11,336	(8,835–13,838)	872	(521–1,223)	7.7	66.9	70.2
Volleyball	56,620	(43,298–69,942)	1,170	(755–1,585)	2.1	31,694	(24,700–38,689)	904	(581–1,227)	2.9	56.0	77.2
Racquet sports[§§§§]	28,268	(20,428–36,108)	723	(294–1,151)	2.6	8,299	(6,408–10,191)	198[§§]	(71–325)	2.4[§§]	29.4	27.4[§§]
Bowling	18,978	(14,628–23,329)	394	(242–546)	2.1	4,863	(3,665–6,061)	93[§§]	(24–163)	1.9[§§]	25.6	23.7[§§]
Track and field	17,025	(13,528–20,523)	305	(129–481)	1.8	15,391	(12,179–18,602)	275	(116–435)	1.8	90.4	90.2

CDC Statistics

- Ages 5-18: two of every three sports-related TBI
- 135,000 of 208,000 sports-related TBIs occur in ages five to 18
- Basketball and bicycling accounted for the greatest number of emergency visits for all causes, with 603,239 and 524,692, respectively.
- But the proportion of brain injuries was highest for horseback riding (11.7%), ice skating (10.4%), all-terrain vehicles (8.4%), tobogganing or sledding (8.3%), and bicycling (7.7%).
- Boys, 10-14, account for 70%; girls in basketball

Highest TBI Risk Groups

- Elderly
- Males 15 - 24 years old
- Substance abuse users
- Minorities of lower SES: African Americans have the highest death rate from TBI
- Infants (64% are due to child abuse)
- Those who have received a prior brain injury
- Military: 1 in 5 of Iraq veterans

Outcome of TBI

- 91% of firearm TBI result in death
- 11% of fall-related TBI result in death
- 80,000 survive TBI hospitalization, but are disabled
- 8 mild to 1 severe: 80-85% mild TBI to 15-20% moderate-severe
- 20% of severe TBI are unresponsive for 1 month
- Coma: only 14% persistent; half regain consciousness in 1 year
- Hospitalized TBI: 73% mild, 16% mod-severe, 11% DOA

Trauma Centers

- Trauma Centers have reduced death rates by 89%
- 22% decline in TBI death rate from 1979 to 1992
- 13% increase in firearm deaths form 1984 to 1992
- 1% of severe TBI survive in PVS

Guns: Suicides outnumber homicides

- Firearms surpassed MVA as largest cause of TBI deaths in 1990
- Suicides accounted for 61 percent of the nation's nearly 31,000 firearm deaths in 2011 (vs. 816 people in Canada in 2002)
- Homicides accounted for 40 percent of gun deaths. Accidents accounted for 3 percent
- Gun-related suicides have outnumbered firearm homicides and accidents for 20 of the last 25 years.
- Guns are leading method of suicide in US

Cost

- $37-260 billion per year in US
- Lifetime cost for 1 person: $4 million
- Average rehab is 55 days, $1000+ per day
- Medical cost of $450,000 if fatal

Helmets

- Fewer than half of U.S. states require every motorcycle riders to wear a helmet; and four states have no helmet requirements whatsoever. Around the world, the same patchwork legal pattern exists.
- 29 states have no bicycle helmet laws
- Helmet use reduces both accidental death and injury, reducing head injury risk by 69 percent and death by 42 percent. (2008)
- California in 1992:
  - 37% decline in fatalities;
  - 99% compliance
- 40% increase in deaths in states that repealed helmet laws

Airbags

- 14% fatality decrease in adults
- 34% fatality increase in children in front seat

Volvo pledge: no one will die or be seriously injured in a Volvo by 2020 (structure, crushable engine, full radar)

Driverless cars are coming

CT vs. MRI

- CT is imaging technique of choice in ED (ID any neurosurgical needs); but poorest sensitivity in mTBI
- 24% with no signs on CT have MRI lesions
- Study of 16 TBI with minor-moderate severity:
  - 1CT was abnormal; 14 MRI abnormal

Risk Factors

- Age
  - For severe TBI, both ends of age spectrum
    - 0-4 years: child abuse, falls
    - 15-19: MVA
    - 65+: falls
- Alcohol/Substance abuse
- Male gender
- High risk behavior

Risk Factors 2

- Prior hx of TBI
- Psychiatric illness, incl. ADHD
- Lower SES or education
- Unemployment

Risk of TBI in Psychiatric Patients

- Psych. pts are at significant increased risk for TBI compared to non-psych. pts (60%)
- Highest risk in organic (psychotic & nonpsychotic), somatoform, schizophrenia, acute stress

Risk of TBI 2

- 1 TBI doubles the risk of 2nd TBI; 2nd TBI increases risk by 8
- Effects are cumulative
- Football players with 3-4 concussions have significantly greater AD risk
- Mohammed Ali was never knocked out
- Boxers: dementia, Parkinsonism, intention tremor, atrophy

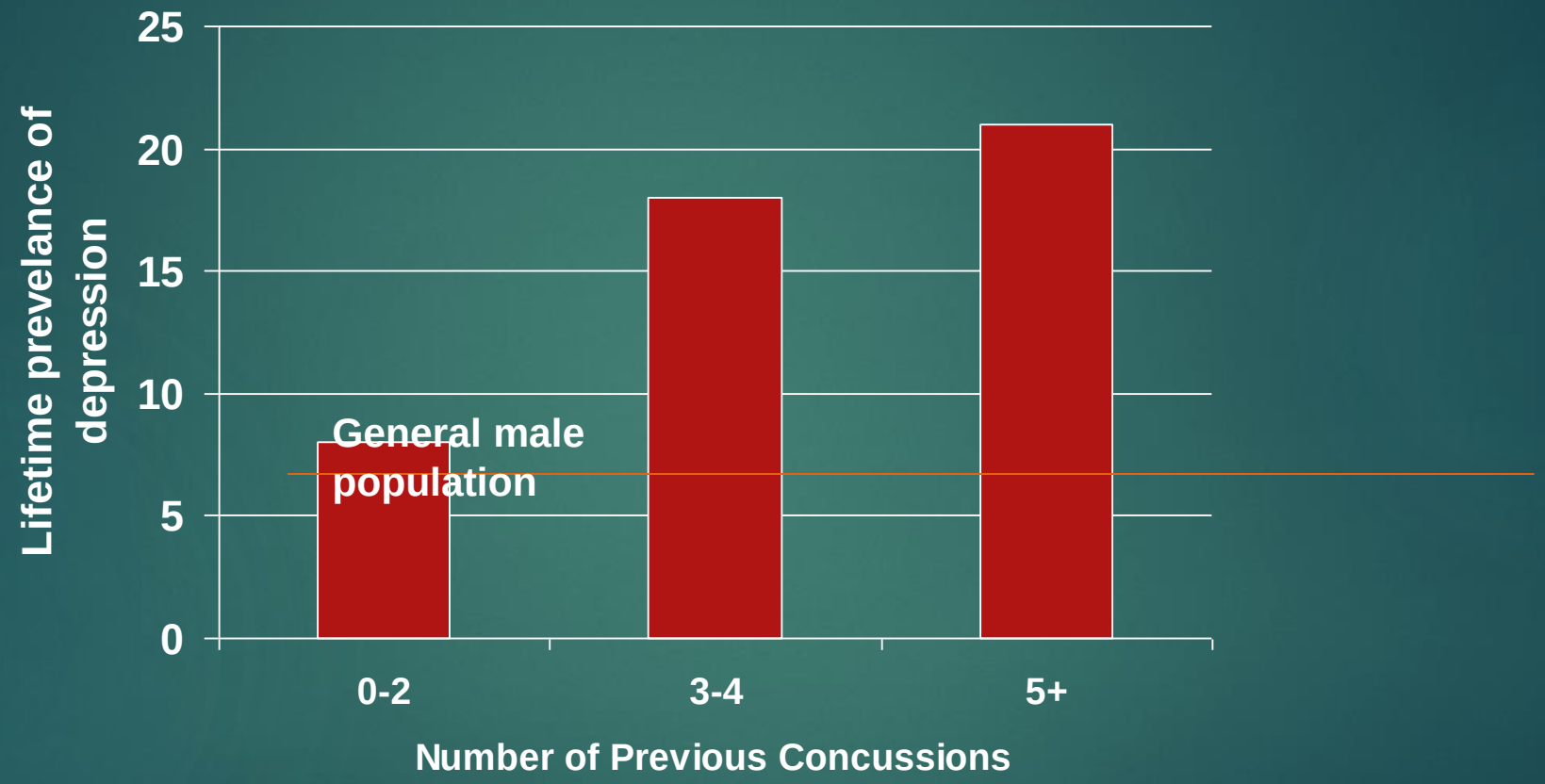
Risk of TBI 3

- 4-5 x greater rate of later dementia if significant TBI
- WWII severe TBI: 10 x greater rate of Alzheimer's
- Football players: 3 or more concussions had 5 x MCI, 3x significant memory disorder

TBI – risk for AD and PD

- 2016 study: n= 7,130; 865 of them reported having had a head injury with some loss of consciousness in the past; how long they thought they had been unconscious: 618 were out for less than 60 minutes, 142 for more than an hour, and 105 were unsure; 1,537 people developed dementia, of whom 1,322 had AD. New Parkinson's cases numbered 117
- A history of TBI had no bearing on whether a person developed mild cognitive impairment, Alzheimer's, or any type of dementia; head injury that knocked a person out for more than an hour increased the risk for PD by 4-fold; TBI with less than an hour of unconsciousness raised the chance that these symptoms would worsen by 65 percent; it more than doubled if a person had been out for more than an hour after the head trauma.
- Autopsy: Those with a TBI that knocked them out for less than an hour were 59 percent more likely to have Lewy bodies in the frontal or temporal cortex. That number rose to 78 percent for people who were unconscious for longer. Among samples from the ACT study, trauma also increased the likelihood of inclusions in the substantia nigra, a major site of pathology in Parkinson's. There was no increased risk for the typical Alzheimer's plaques specified by CERAD criteria, or tau tangles characteristic of Braak stage V/VI
- full-blown, autopsy-confirmed Alzheimer's disease is not necessarily associated with traumatic brain injury,

Concussion As Risk for Depression

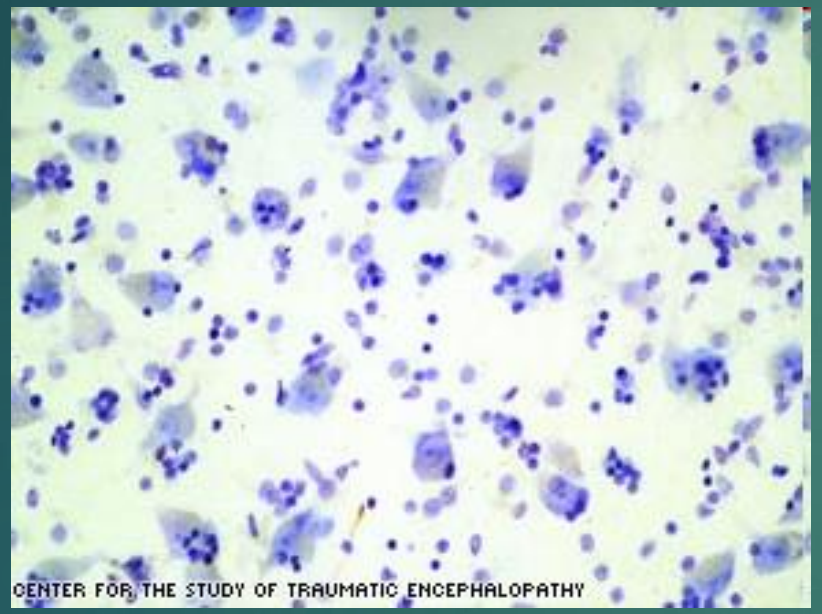


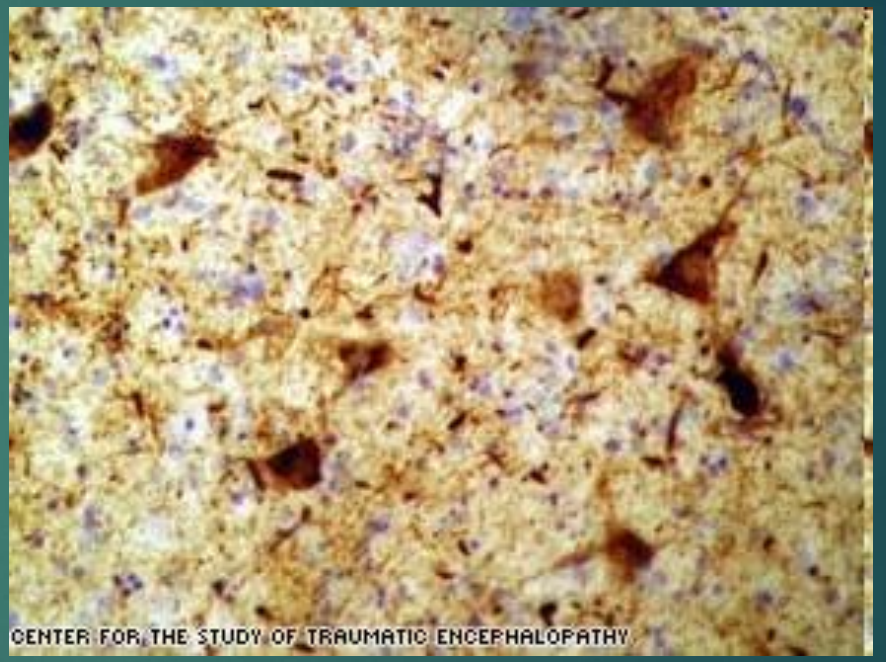
Survey N = 2,488
Depression N = 263

Chronic traumatic encephalopathy

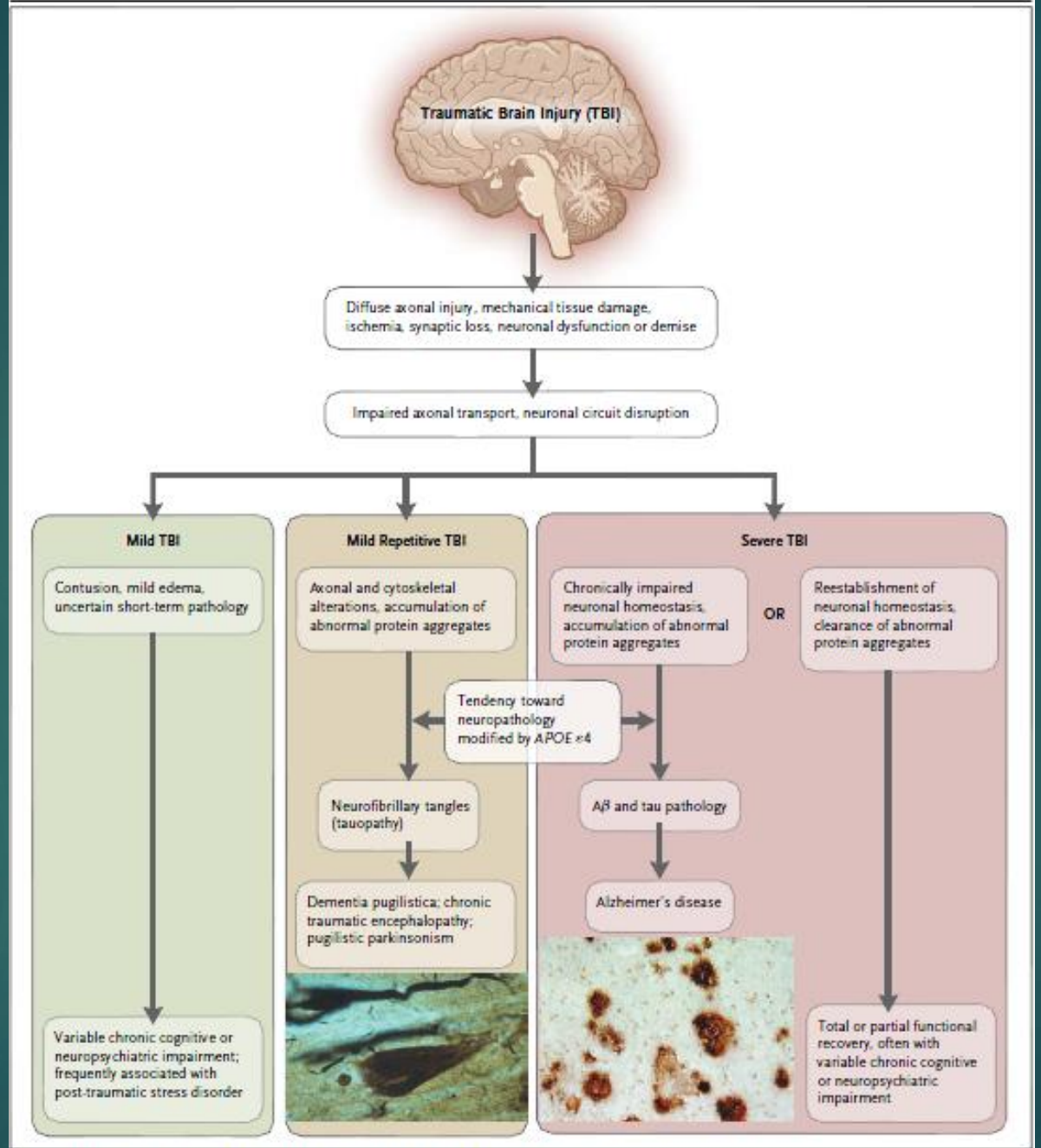
- CTE: a degenerative brain disease caused by head trauma.
- Build-up of toxic protein tau in the form of neurofibrillary tangles (NFTs) and neuropil threads (NTs) throughout the brain.
- CTE eventually progresses to full-blown dementia
- Initially referred to as dementia pugilistica

Normal vs. CTE tissue





Even without known concussion, i.e. Iron Mike Webster, center



Spectrum of Pathologic Features and Outcomes of Traumatic Brain Injury (TBI).

In the left inset, Bielschowsky silver stain shows intraneuronal and extracellular neurofibrillary tangles in temporal cortex from a retired boxer with dementia pugilistica.[1] The right inset shows diffuse Aβ plaque deposits in temporal cortex from a subject who sustained severe TBI.[2]

S. DeKosky, et al.,
NEJM, 2010

Mild TBI

- Uncomplicated mild traumatic brain injury (mTBI), often referred to as a concussion, involves:
  - a traumatically induced physiological disruption of brain function
  - that results in a graded set of clinical sxs
  - that most often resolve spontaneously.
- Relative to moderate or severe TBI, it has a very different recovery course

Controversy: Mild TBI is not a diagnosis

- Mild TBI is not a "diagnosis," and it is certainly not a diagnosis made over time.
- ALL definitions of mTBI make clear this definition describes a historical event, not a long term mental diagnosis. Mild traumatic brain injury (concussion) is an event involving physical trauma to the head resulting in alteration of consciousness, which is the result a physiological alteration of brain functioning.
- NONE of the definitions involve long term brain damage. That is a total misconception which plagues the literature and distorts the debate. The VAST majority of persons who suffer mTBI or concussion recover clinically just fine

Tom Kay, Ph.D., R. Psych.

Postconcussion Syndrome?

- There also is a major problem with the very concept of post-concussion syndrome, since many of the symptoms that are considered part of this syndrome (headache, nausea, balance, sensory sensitivity) have NOTHING to do with the concussion (mTBI, alteration of BRAIN functioning) at all, and are due to peripheral injuries, not the CNS.
- I have been an advocate of eliminating the term (and concept) of post-concussion syndrome.
- We need to get more rigorous in our distinctions between head injury and brain injury. Person who have concussions and other peripheral injuries often become dysfunctional long term. This is seldom because of permanent brain damage. Mild traumatic brain injury describes a historical event which may or may not result in long term dysfunction, which itself may or may not have neurological components.

Functional & NP Outcome with TBI

- NP and functional outcome related to injury severity.
- Acquired long term deficits are common in mod and severe brain injuries, but uncommon in mTBI
- Most recovery in mod to severe TBI occurs in 1st year; substantial recovery by 3+ years
- Recovery of sxs from mTBI occurs in first days to weeks following injury. No cognitive consequences in 1-3 wks in athletes & 1-3 mths in trauma pts.

Recovery in mTBI

- Early intervention as simple as education and reassurance can reduce number and frequency of post concussion sxs and increase return to work rates.
- Depression is fairly common. Many of these sxs mimic persistent post concussive sxs.

Factors affecting Recovery

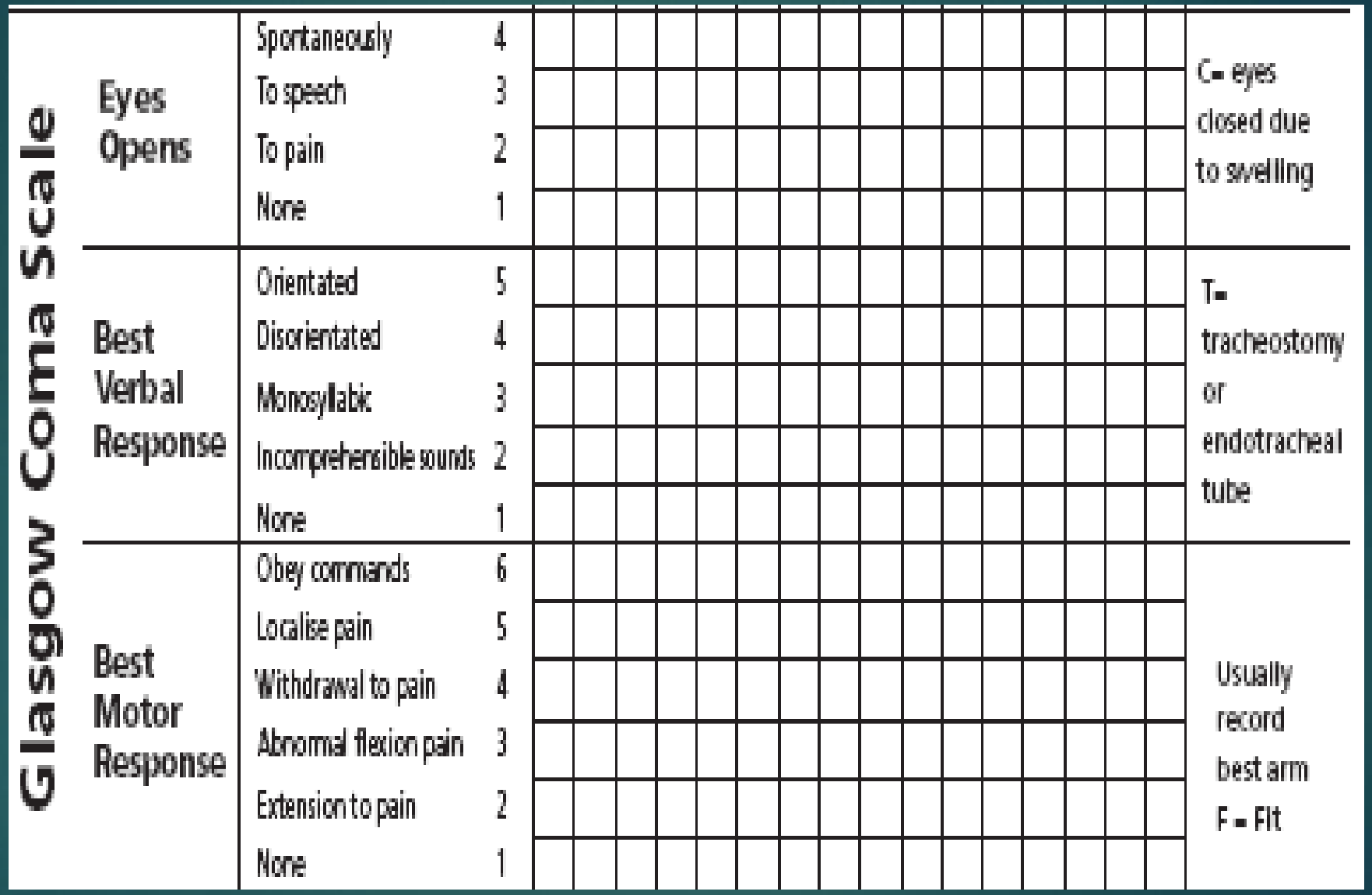
- Recovery following TBI is multifaceted
  - 1 year post TBI = significant individual differences
- Important factors premorbidly are:
  - Age, education, and injury severity
- Cognitive recovery past 1 year minimal and only in measures of complex attention
- Rate of recovery dependent on cognitive domain and location of injury

How to measure “severity”?

- Duration of loss of consciousness
- Initial score on Glasgow Coma Scale (GSC)
- Length of post-traumatic amnesia (PTA)
- Rancho Los Amigos Scale (1 to 10)

Outcomes Following TBI coma

- Coma & NP deficits: Dose-Response Relationship
  - Dikmen, et al. (1995) found a significant relationship between length of coma (Time to Follow Commands) and level of performance on sensitive neuropsychological measures at 1 year post-injury
  - Greater cognitive impairment is associated with longer periods of coma


Glasgow Coma Scale
Eyes Opens
Spontaneously 4
To speech 3
To pain 2
None 1
C= eyes closed due to swelling
Best Verbal Response
Orientated 5
Discrientated 4
Monosyllabic 3
Incomprehensible sounds 2
None 1
T= tracheostomy or endotracheal tube
Best Motor Response
Obey commands 6
Localise pain 5
Withdrawal to pain 4
Abnormal flexion pain 3
Extension to pain 2
None 1
Usually record best arm
F = Flt

Glasgow Coma Scale 1

- Eye Opening (1-4)
- Motor Response (1-6)
- Verbal Response (1-5)
- Lower score is worse:
  - 8 or less = severe
  - 9-12 = moderate
  - 13-15 = mild

Glasgow Coma Scale 2

- 50% of hospitalized TBI have scores of 13-15
- Alcohol decreases GCS scores
- Outcome of TBI by Glasgow rating:
  - Severe: 4% PVS, 8% severely disabled (dependent for ADLs)
  - Moderate: 22% independent, but not financially
  - Mild: 66% good recovery

Coma

- Coma duration: poor predictor of outcome if <30 min
- LH penetration have longer coma, more LOC than RH
- PVS: Persistent Vegetative State:
  - Wakeful, unresponsive, sleep-wake cycle, no cortical functioning, atrophy on CT

Amnesia

A PERIOD OF ANTEROGRADE AMNESIA IN WHICH NEW MEMORIES CANNOT BE CONSISTENTLY MADE AND RECALLED THAT FOLLOWS RECOVERY OF CONSCIOUSNESS IN HEAD INJURY OR OTHER NEUROLOGICAL TRAUMA.

THE DURATION OF PTA IS OFTEN USED AS A PREDICTOR OF THE DEGREE OF RECOVERY.

Post Traumatic Amnesia

- Amnesia (PTA)= resumption of continuous memory:
- Duration of PTA correlates with permanent memory disorder, tends to correlate with GCS, lasts 4 x length of coma; ask a collateral person
- Length of PTA more accurate than GCS, LOC, or coma duration in predicting cognitive status in 2 years
- < 10 minutes = very mild
- 10-60 min= mild
- 1-24 hours = moderate
- 1-7 days = severe
- >7 days = very severe
- With each additional day of PTA, greater the brain atrophy

Levels of Severity

- Mild Head Injury
  - Glasgow Coma Scale 13 – 15
  - PTA ≤ 24 hours
  - LOC ≤ 30 min; normal CT/MRI
- Moderate Head Injury
  - Glasgow Coma Scale 9 – 12
  - PTA ≤ 1-7 days
  - LOC ≤ 6 hrs; normal/abnormal CT/MRI
- Severe Head Injury
  - Glasgow Coma Scale < 9
  - PTA >7 days or longer
  - LOC > 6 hrs; normal/abnormal CT/MRI

Other predictors of cognitive deficit

- EVP in comatose
- Visual Field deficits (more severe)
- More severe MRI results
- Neuropsych. results (social competence decreased even when WNL)
- Quality of life correlated with severity of cognitive outcome

Smell as predictor

- There is a high correlation between anosmia and frontotemporal damage in head trauma.
- Green: After removing poor effort cases, smell test scores were by far the strongest statistical predictors of head injury severity. In fact, anosmia was 28 times more prevalent in those with more than 24 hours PTA compared with those with less than 24 hours of PTA.
- Anosmia (Varney: predicts unemployment)

Mechanism of Brain Injury

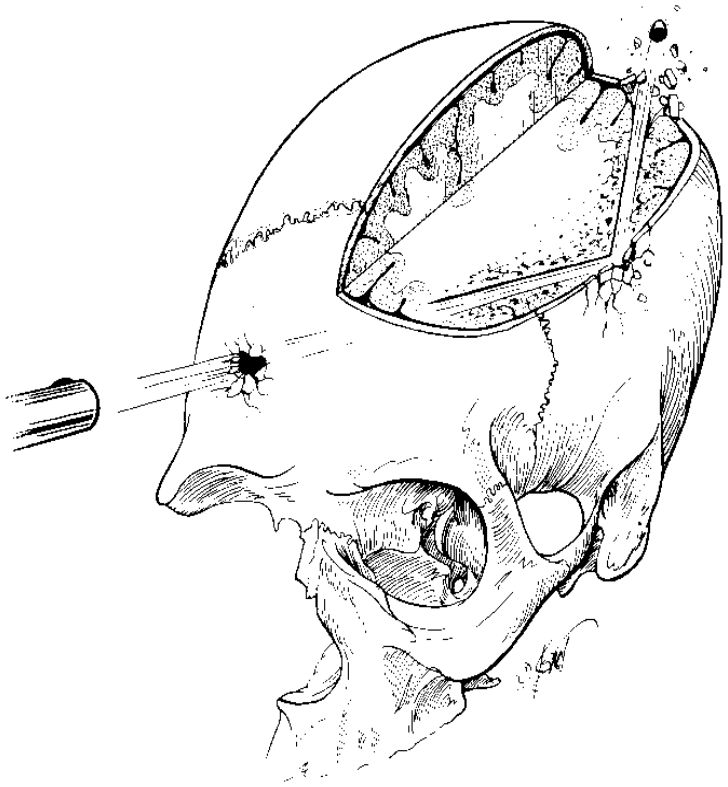
- Primary Injury
  - Damage that results from linear/rotational shear forces; seen in the initial minutes/hours after the insult
  - Skull fracture
  - Contusion
  - Cortical disruption
  - Axonal Injury
  - Vascular Injury
  - Subarachnoid Hemorrhage
  - Mostly frontal/temporal
- Secondary Injury
  - Cascade of brain events after injury; evolution of brain damage; gradual/quick
  - Neurometabolic cascade
  - Hypoxia
  - Post traumatic ischemia
  - Hypotension, poor cerebral perfusion pressure
  - Excitotoxicity
  - Mass lesions
  - Cell Death
  - Axonal Injury
  - Cerebral Swelling/edema
  - Raised intracranial pressure/herniation

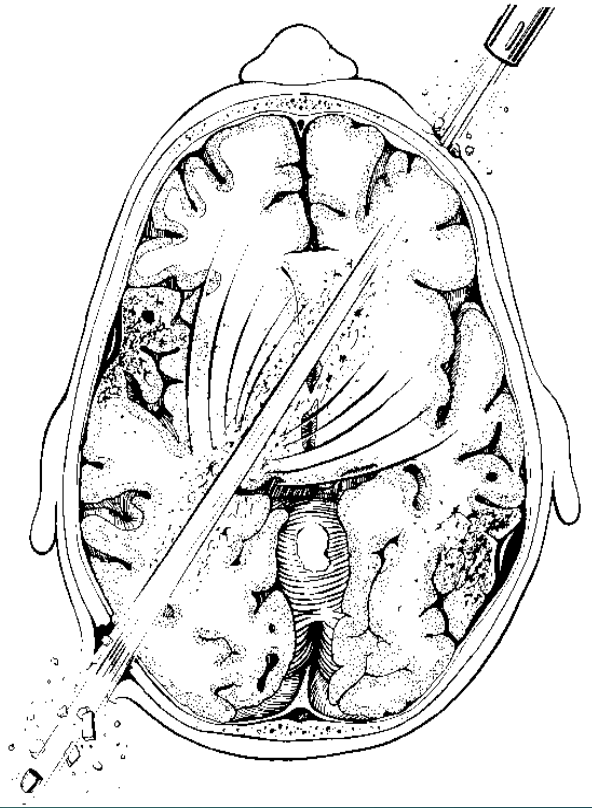
Closed Head Injury: Most Common TBI

- Resulting from falls, motor vehicle crashes, etc.
- Focal damage and diffuse damage to axons
- Effects tend to be broad (diffuse)
- No penetration to the skull

Open Head Injury

- Results from bullet wounds, etc.
- Largely focal damage
- Penetration of the skull
- Effects can be just as serious



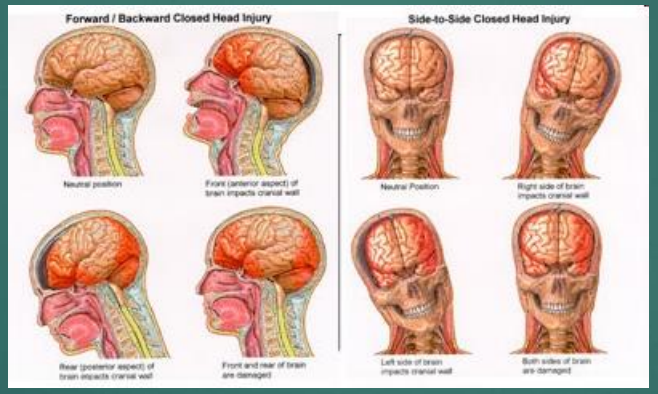


Pathophysiology: Primary

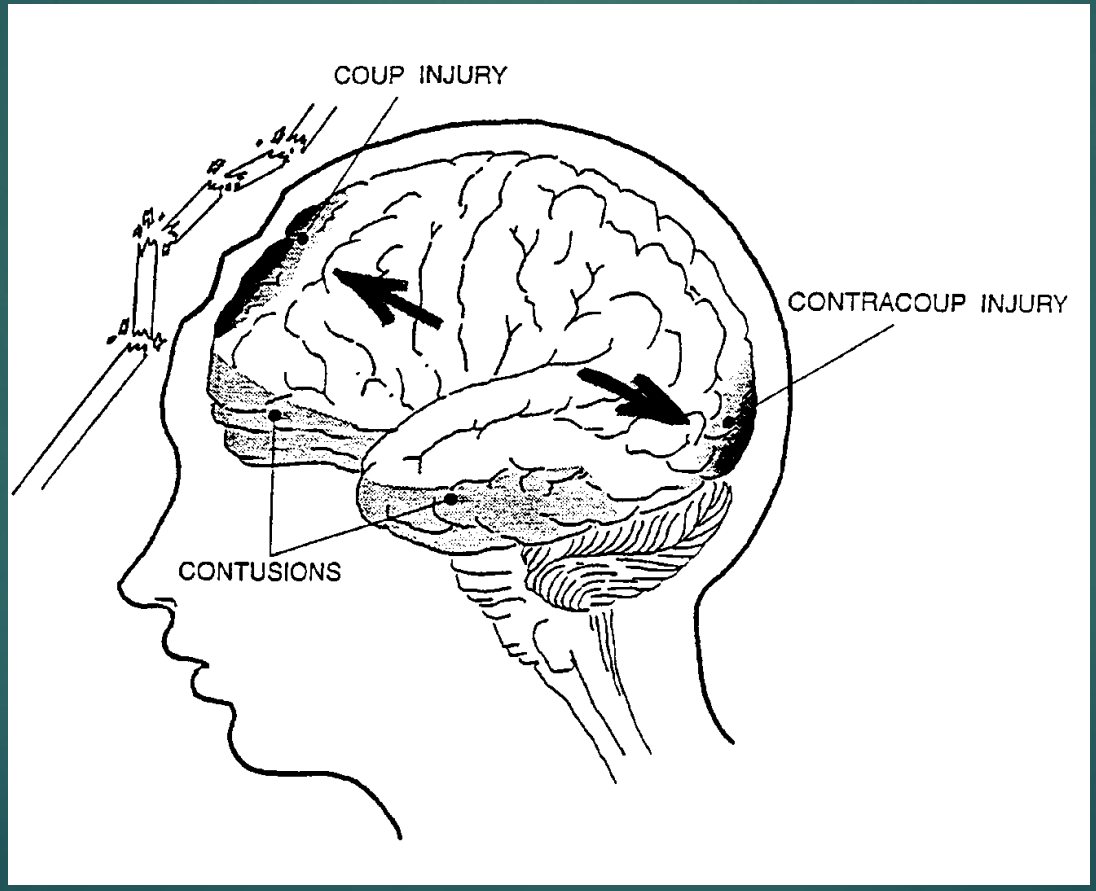
- Focal Contusions: damage at impact, bone-brain contact
- Coup – blow at point of impact
- Countercoup – contusion at opposite area of blow:
- 50-80% of damage is due to countercoup
- Frontal and Temporal poles and undersurfaces

Coup and Contracoup

TBI


Forward / Backward Closed Head Injury
Side-to-Side Closed Head Injury
Neutral position
Front (anterior aspect) of brain impacts cranial wall
Neutral Position
Right side of brain impacts cranial wall
Rear (posterior aspect) of brain impacts cranial wall
Front and rear of brain are damaged
Left side of brain impacts cranial wall
Both sides of brain are damaged

Coup and Countercoup



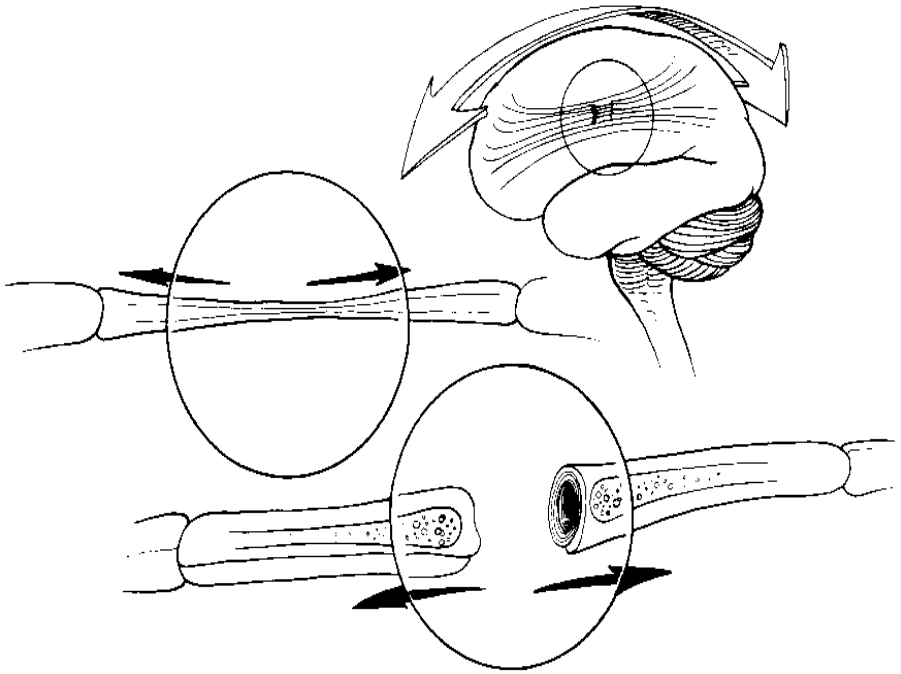
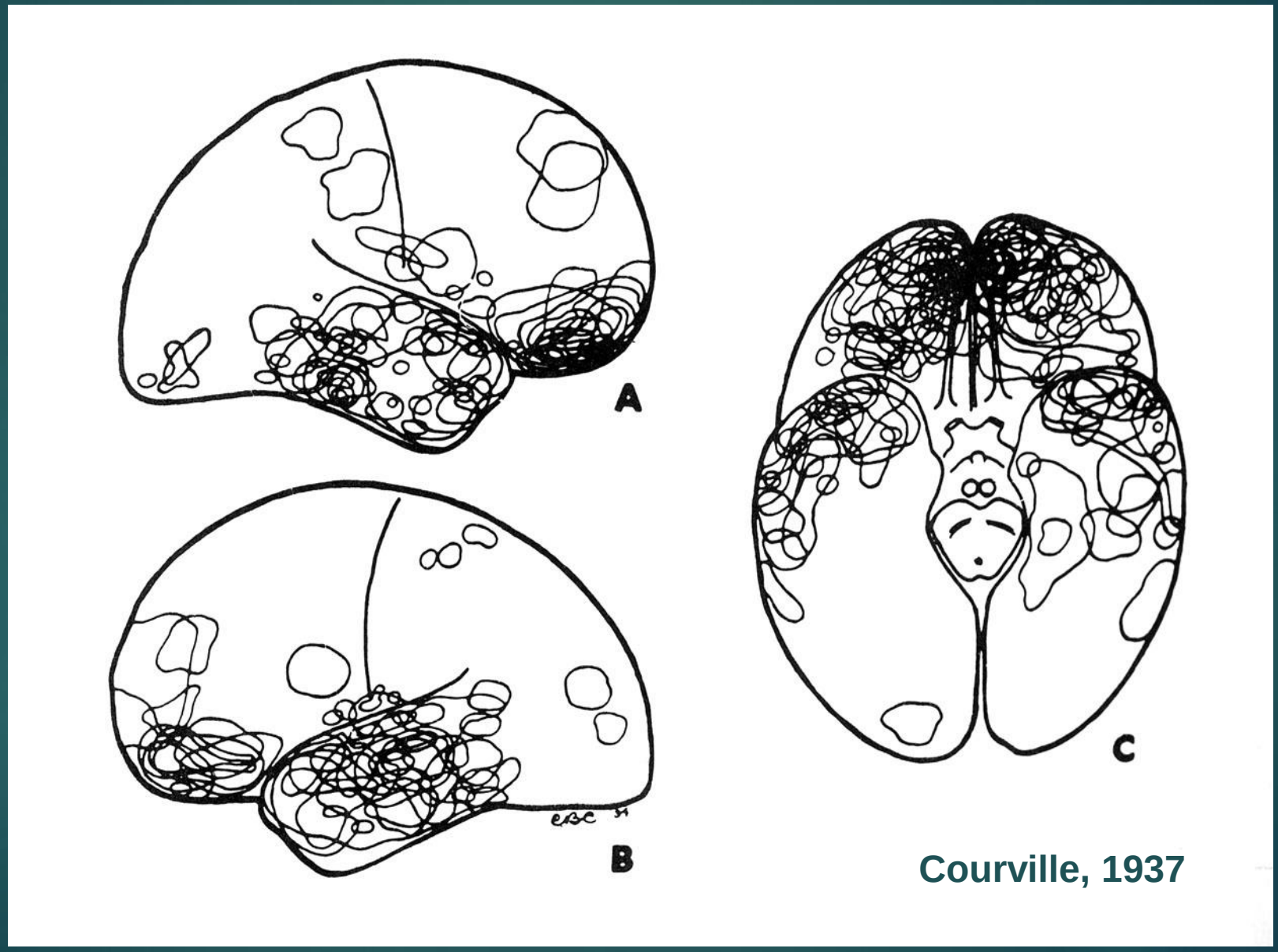


Figure 1-16. Axonal shearing may occur in acceleration as well as deceleration injuries. The nerve fiber may be stretched or completely severed, producing the manifestations of diffuse head injury.

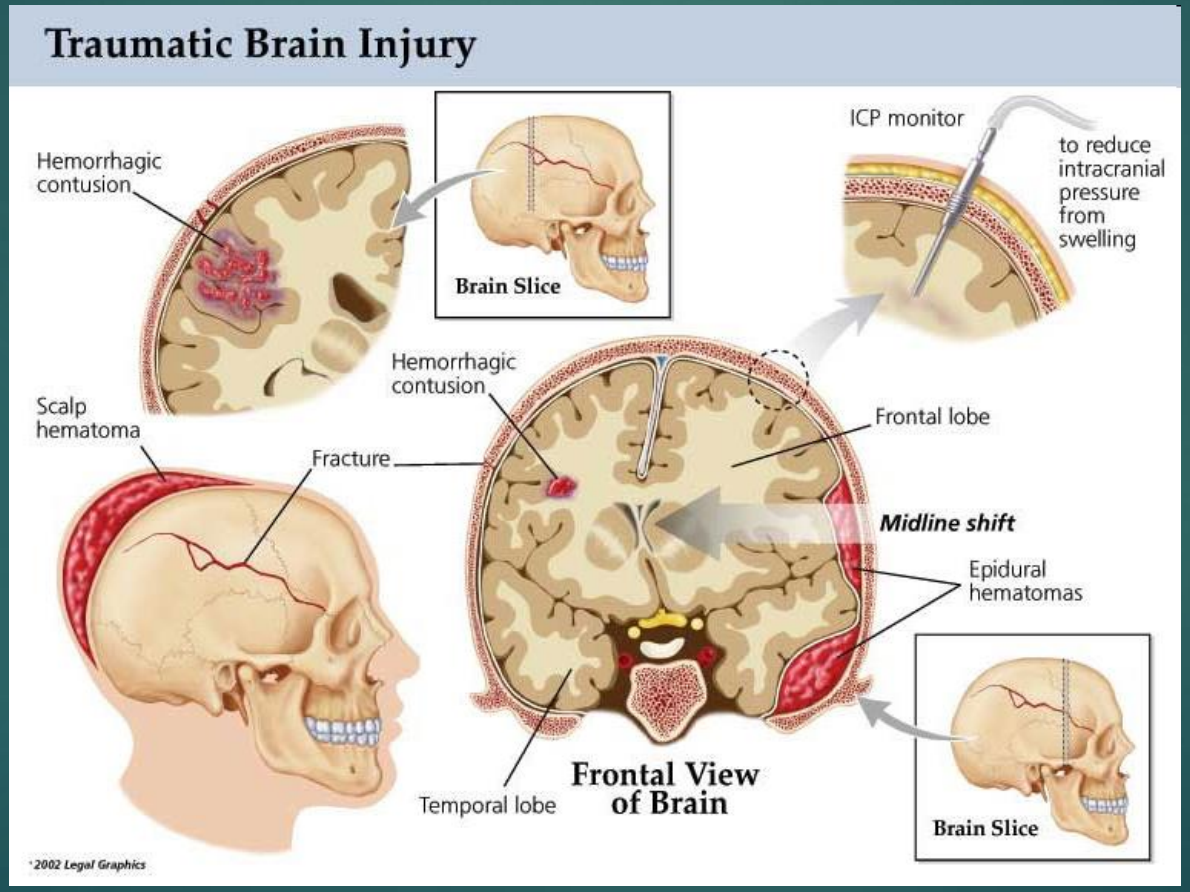
Axonal Shearing

Most Common: Frontal & Temporal Contusions



Courville, 1937

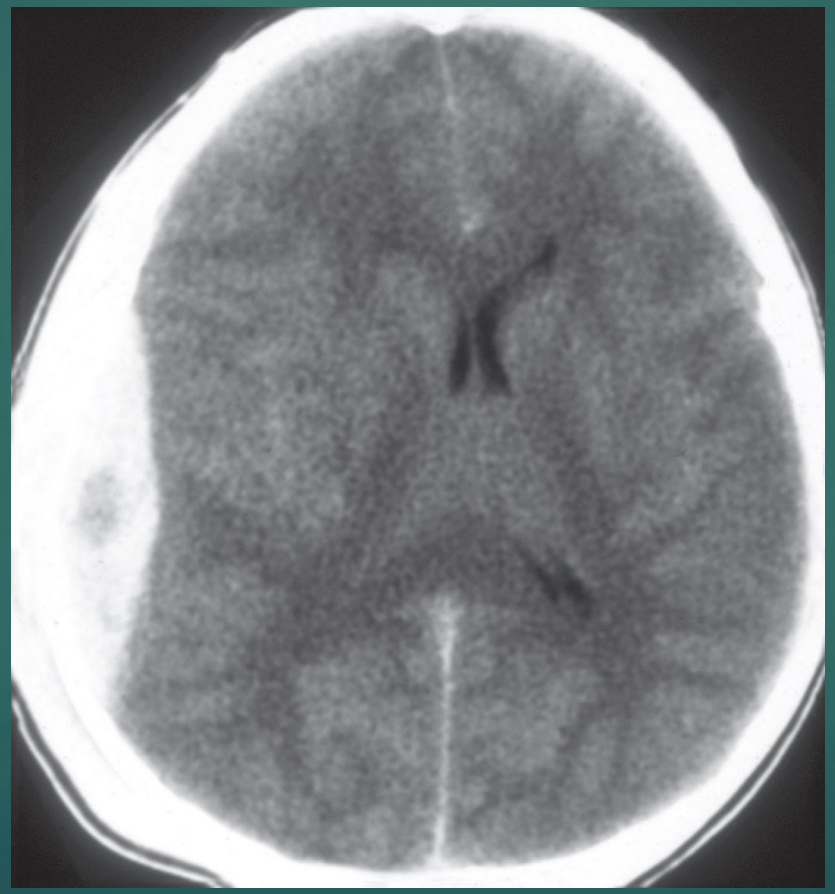
TBI


Traumatic Brain Injury
Hemorrhagic contusion
Brain Slice
ICP monitor
to reduce intracranial pressure from swelling
Hemorrhagic contusion
Scalp hematoma
Fracture
Frontal lobe
Midline shift
Epidural hematomas
Temporal lobe
Frontal View of Brain
Brain Slice
2002 Legal Graphics

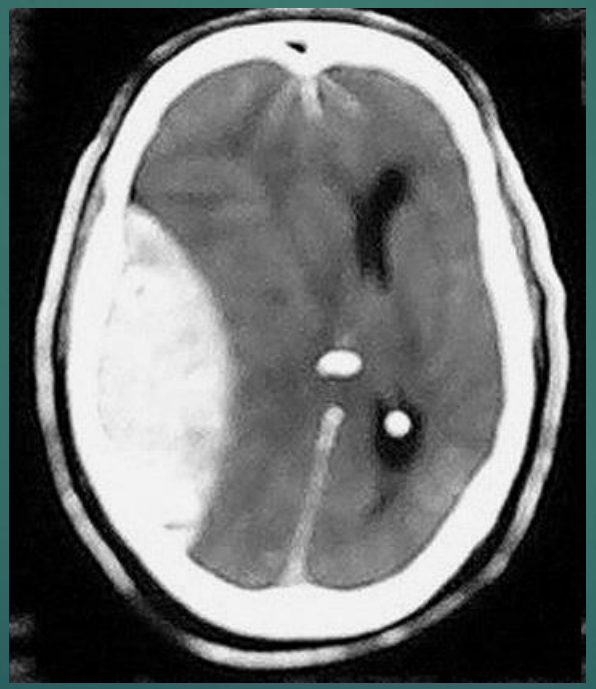
Hematomas (Bleeds)

- Intracranial hematomas: subdural, epidural; and edema; pressure effects
- Subdural hematomas: 75% ok if evacuated < 4 hours; age 60+ are 4x more likely to develop chronic SDH
- Epidural hematomas: Meningeal artery tear
- 68% coma < 24 hours, good recovery
- Closed HI with hemorrhage more severe than with DAI (Diffuse Axonal Injury) alone

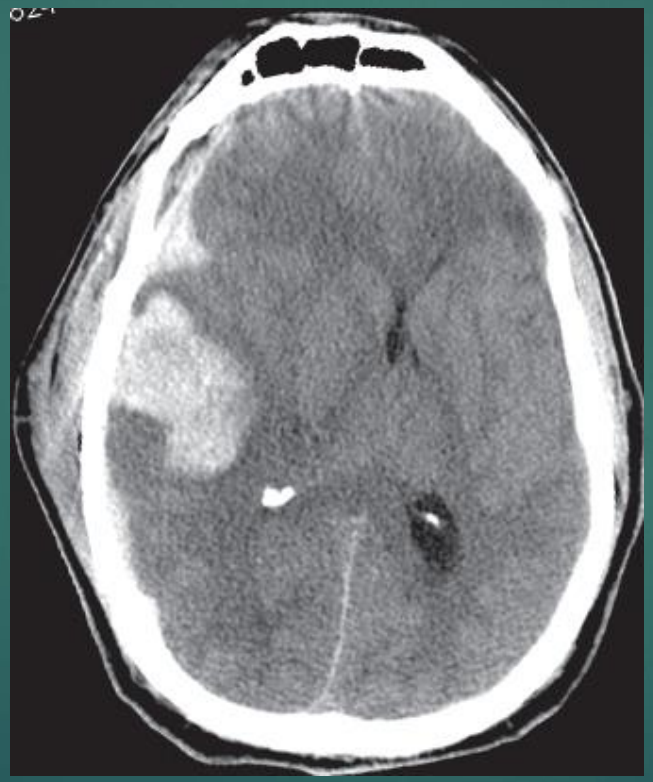
Epidural Hematoma



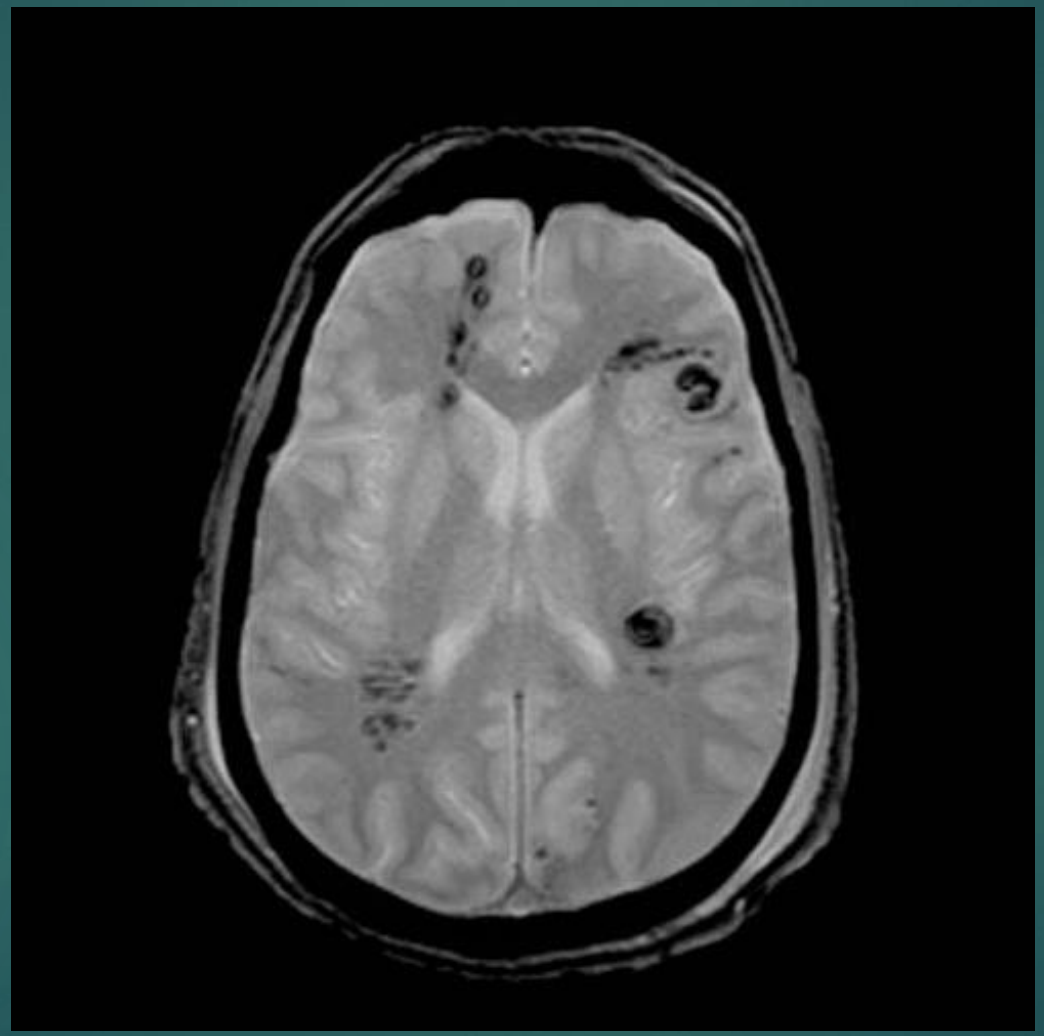
Epidural Hematoma



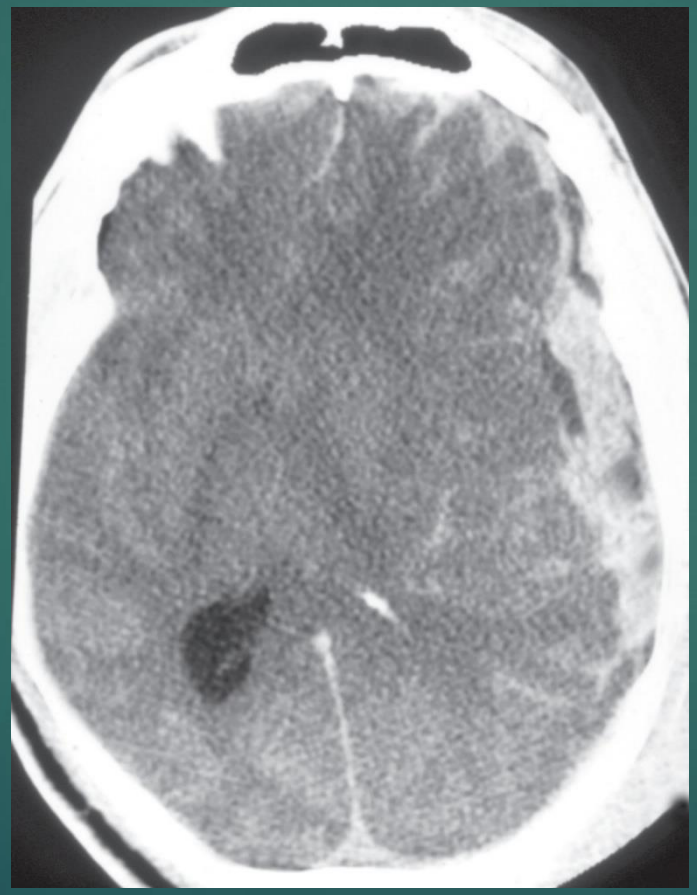
Contusion/Hematoma



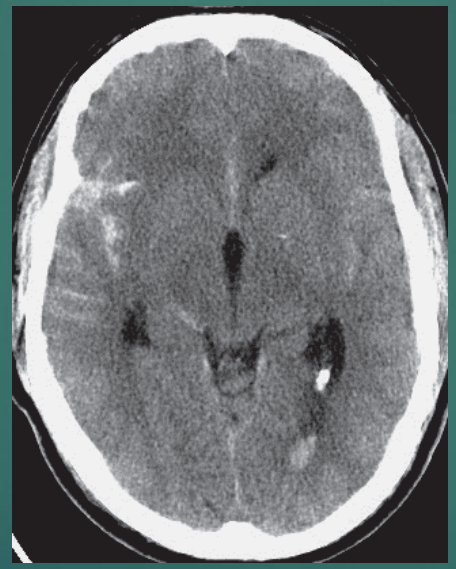
DAI: Diffuse Axonal Injury



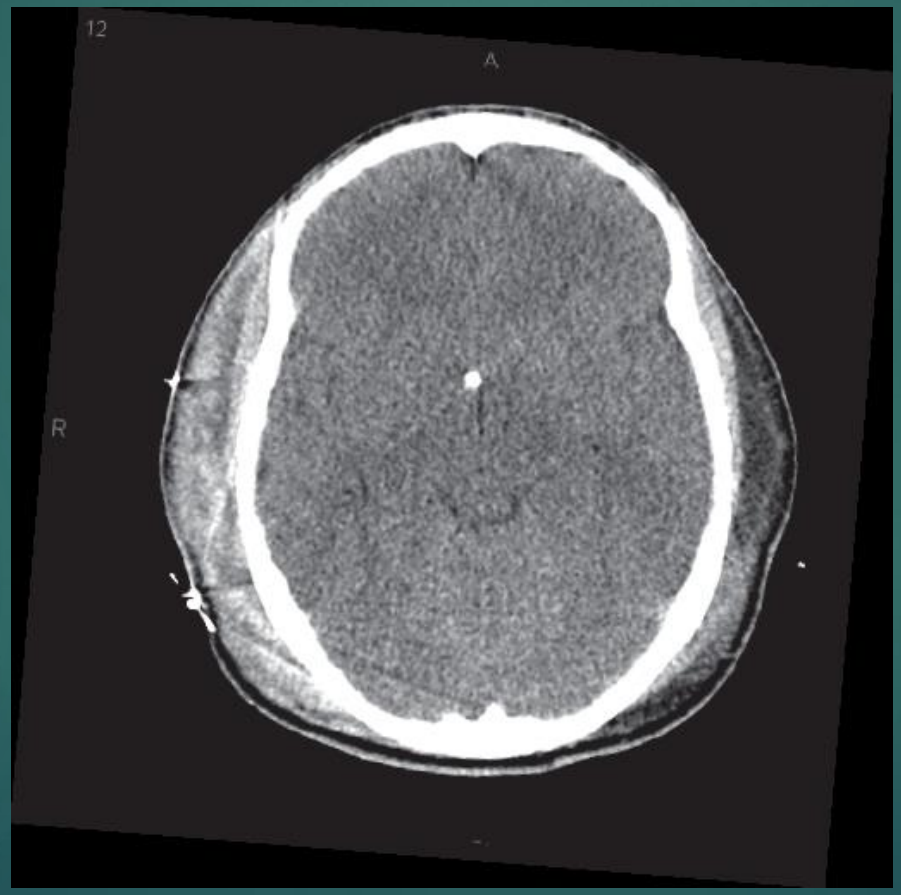
Subdural Hematoma: SDH



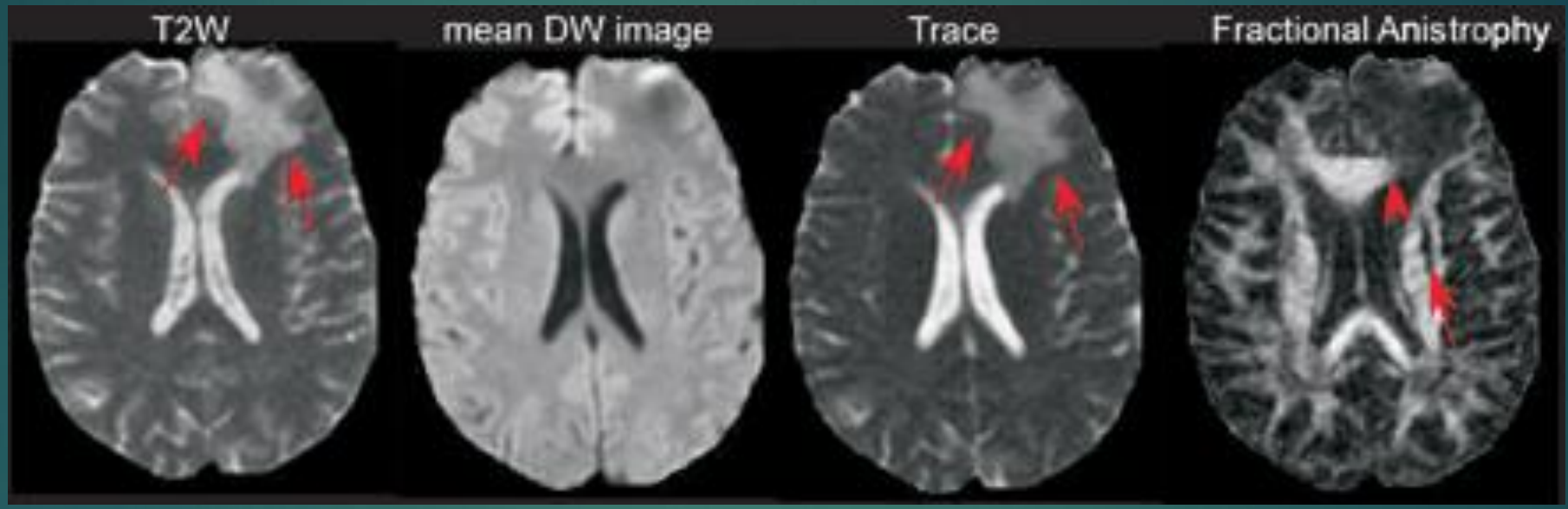
Subarachnoid hemorrhage



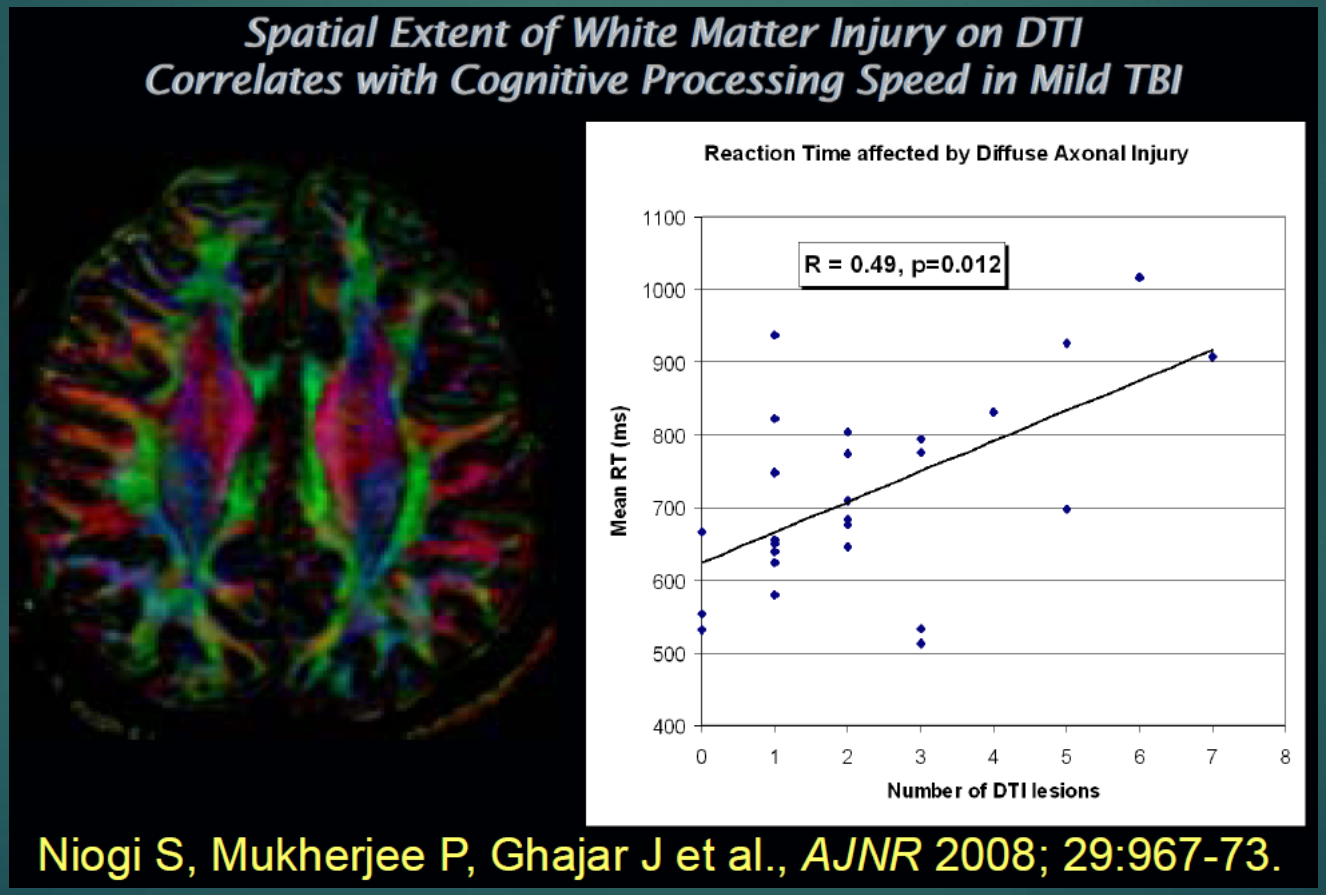
Diffuse Swelling



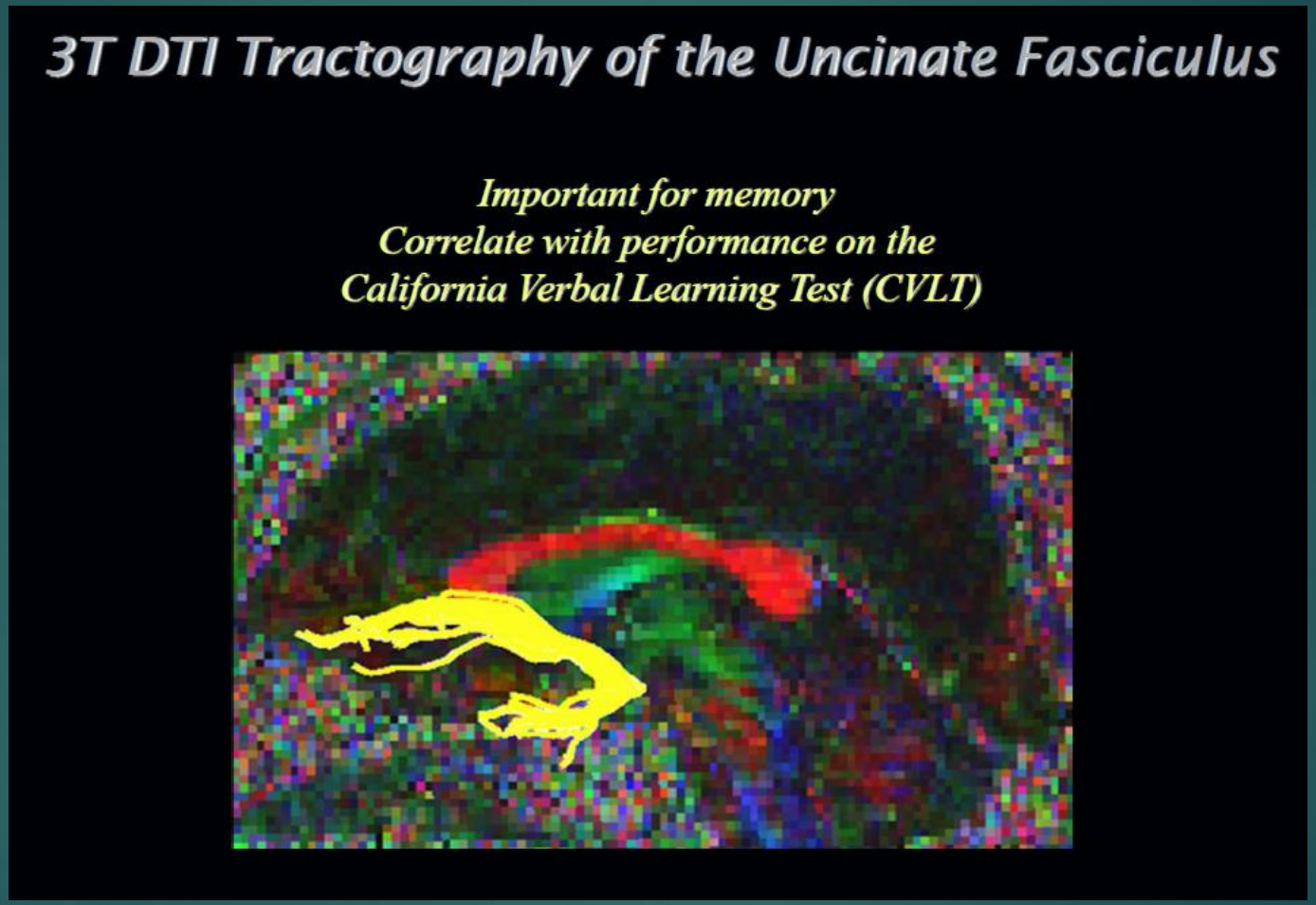
Frontal TBI: 4 types of MRI views



DTI: White matter injury produces PS ↓ ↓



Uncinate Fasciculus ↓ ↓ = CVLT ↓ ↓



Connects parts of the limbic system (hippocampus and amygdala in the temporal lobe) with frontal ones such as the orbitofrontal cortex

Acceleration - Deceleration

- Blow not necessary, only rapid deceleration
- Worse Scenario: Armored Truck
- Effects of momentum trauma:
  - multifocal
  - bilateral damage
  - diffuse
  - anterior > posterior
  - deeper> surface

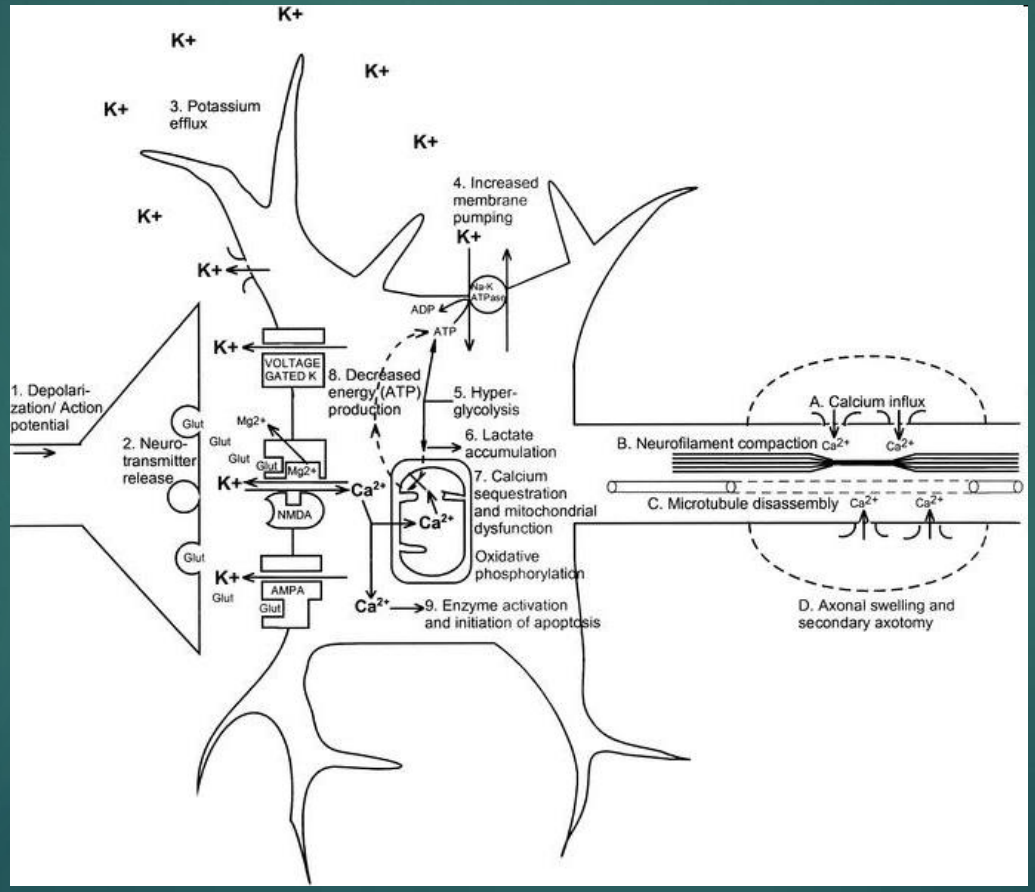
Concussion

- Trauma-induced alteration in mental status with or without LOC
  - Vacant stare
  - Delayed responses
  - Confusion, inability to focus attention
  - Disorientation
  - Slurred speech
  - Incoordination
  - Emotions out of proportion to circumstance
  - Memory deficits
  - LOC

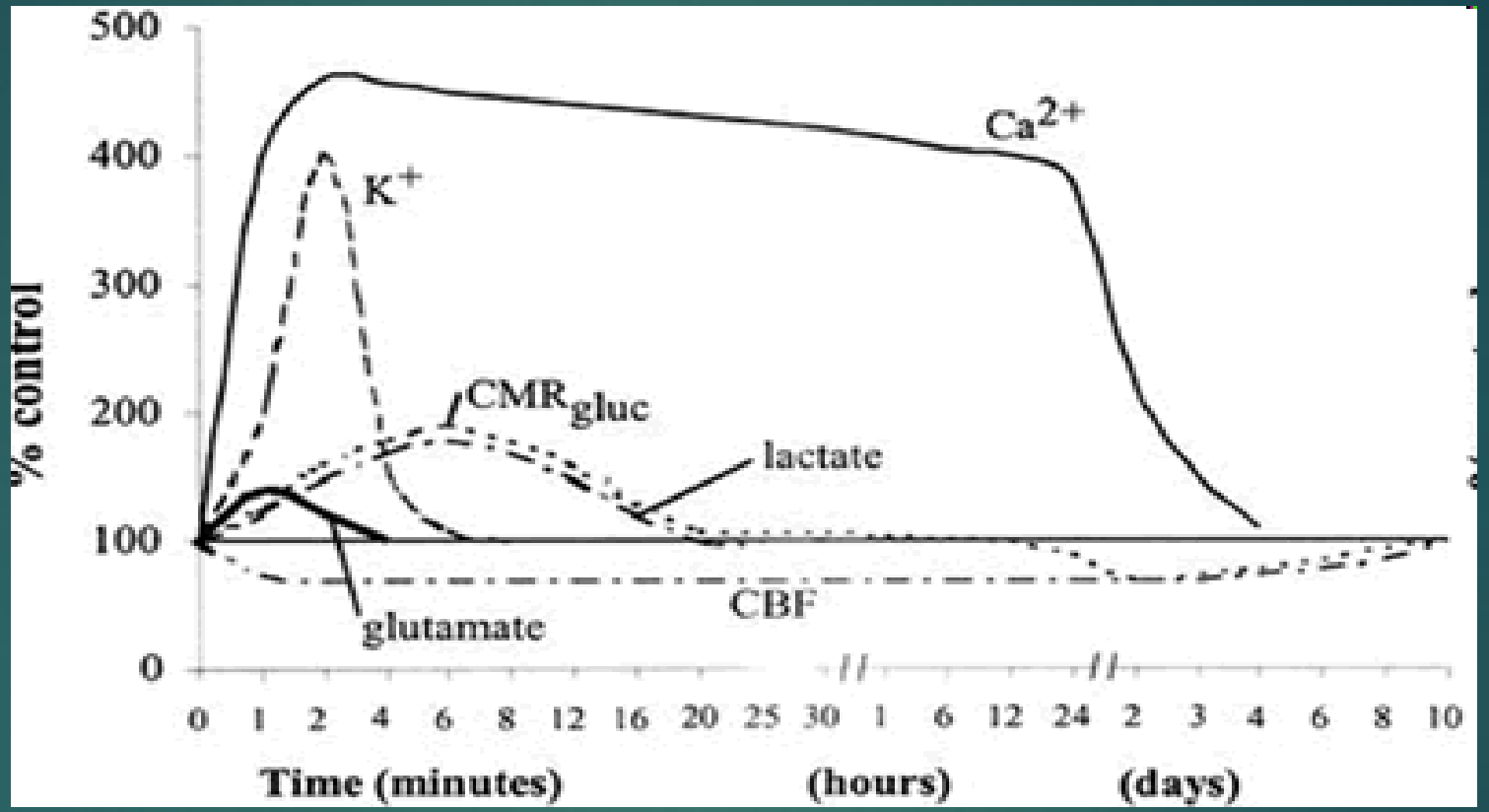
Concussion

- Injury effects of rapid acceleration – deceleration
- Mild = no LOC
- Classic severe= reversible coma, can incl. CV and pulmonary changes, neurological changes (decerebrate posturing, papillary changes, seizures) in 1st 30 min.
- Severe closed head injury: 72% have ventricular enlargement, esp. with prolonged coma

Neurometabolic Cascade of mTBI



Neurometabolic Cascade of mTBI 2



Late events in the cascade include recovery of glucose metabolism and CBF, delayed cell death, chronic alterations in neurotransmission, and axonal disconnection.

Diffuse Axonal Injury (DAI)

- Tear, stretch, rotation and twisting of axons and blood vessels from sudden deceleration cause microscopic lesions, esp. in Frontal and Temporal regions, as well as in white matter, midbrain, pons, corpus callosum, cerebellum
- Most prominent at gray-white matter junctions.
- Disconnection from subcortical structures
- No blow needed, i.e. whiplash

TBI: A White Matter Disease

- Since the time of Geschwind's seminal work, great progress has been made in understanding connectivity among brain regions that work in concert to bring about goal related behavior, as well as the consequences of damage to these networks as a result of traumatic axonal injury. Although post-mortem and histology work has been pivotal in understanding the TBI-associated abnormalities to network connections, recent advances in neuroimaging have helped to rapidly move the field forward. As TBI is primarily a white matter disorder, the advent of diffusion imaging has allowed visualization and quantification of disrupted white matter connections and axonal injury.

- Our review of the literature suggests that TBI, even of mild severity, is associated with acute and chronic white matter abnormalities that likely involve the loss of axonal integrity and myelin, and non-specific mechanisms of injury including inflammation and edema. Although there may be some tracts that are particularly vulnerable to axonal injury such as the corpus callosum, TBI is a spatially heterogeneous injury, characterized by diffuse damage to white matter fibers. Longitudinal studies have demonstrated that, although there is evidence for axonal recovery, persistent white matter abnormalities are observed in the chronic state of injury, particularly in more severe injuries. These white matter abnormalities are not without consequences; the review of the literature reveals that structural abnormalities are associated most often with executive dysfunction and reduced memory performance.

Microbleeds in TBI: Heavily Frontal

Cerebral microbleeds produce small deposits of the iron-storing protein hemosiderin.

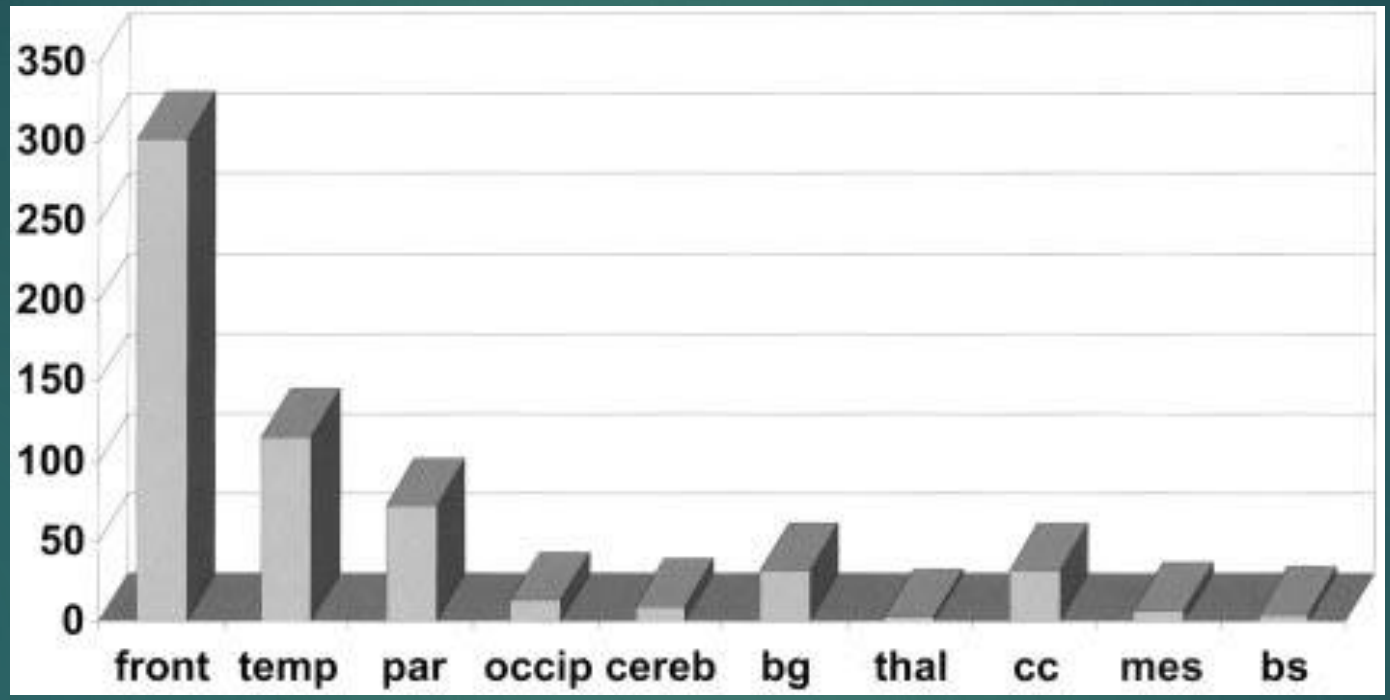


FIG 1. Frequency and site of traumatic microbleeds according to 10 brain areas. Shown is the total number of traumatic microbleeds in each brain area

AJNR, 24, June/July 2003

Secondary Pathophysiology

- Hypoxia – ischemic insufficiency
- Focal infarcts, ICP elevation, PVS
- Severe Consequences: amnesia, dementia
- Intracranial hypertension
- Infection
- Hydrocephalus
- Herniation
- Neurochemical changes

Nutrition and Increased Cranial Pressure

- Nutrition: 1.5-2.5 x higher than normal caloric requirement; basal metabolism increases in TBI
- Increased cranial pressure (ICP) increases:
  - Reduced profusion, hippocampus vulnerable to hypoxia
  - Ischemic necrosis in hippocampus common in fatal cases
  - Most common cause of death
  - Predicts chronic impairment

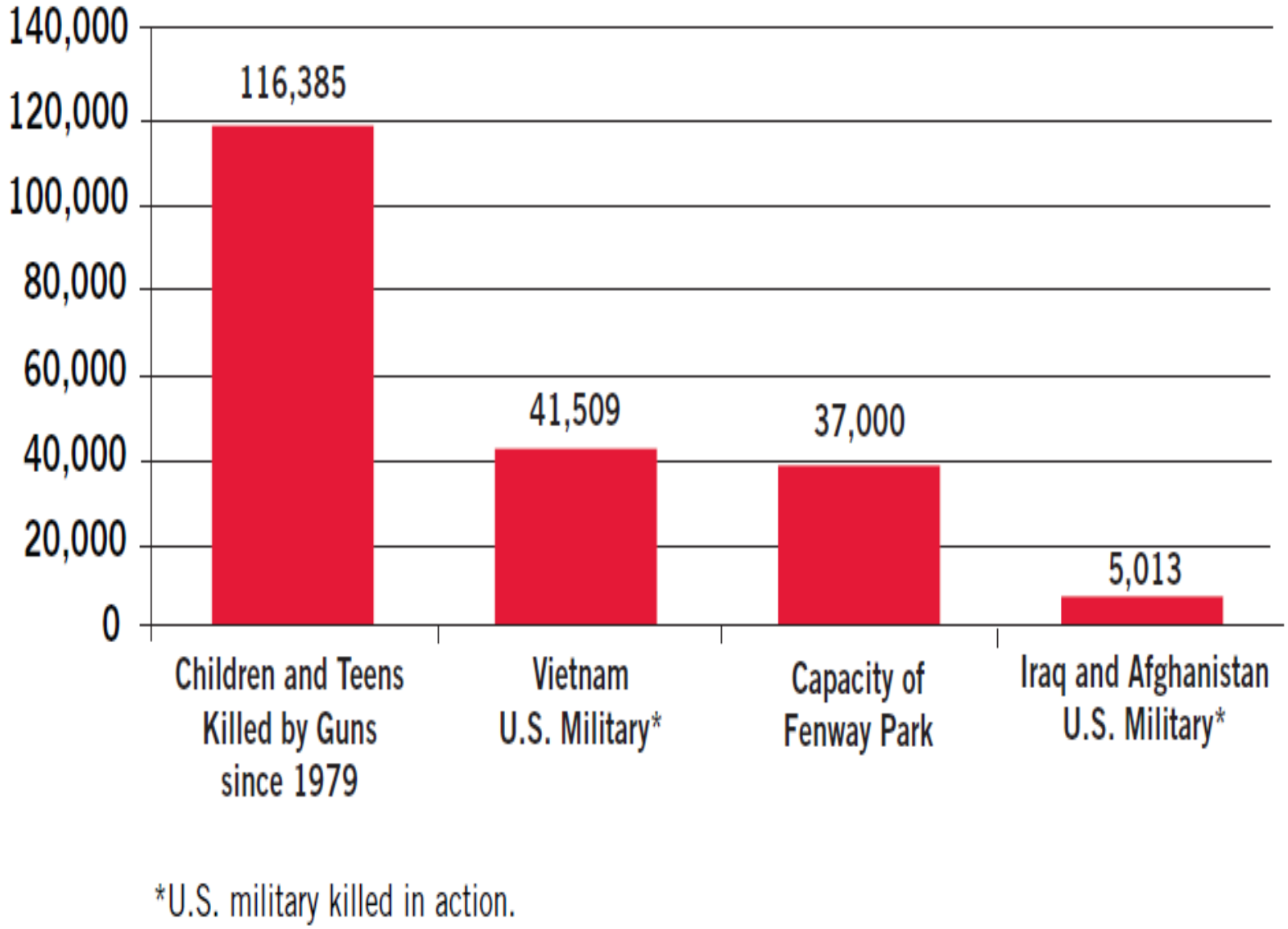
Pediatric TBI

- More deaths (4000 per year) than all other pediatric diseases: 1 in 800 die in MVA TBI before age 25
- 15,000 require prolonged hospitalization with long term sequelae
- Duration of coma directly related to long term outcome.
- 1 child dies of gunshot wound every 3 days

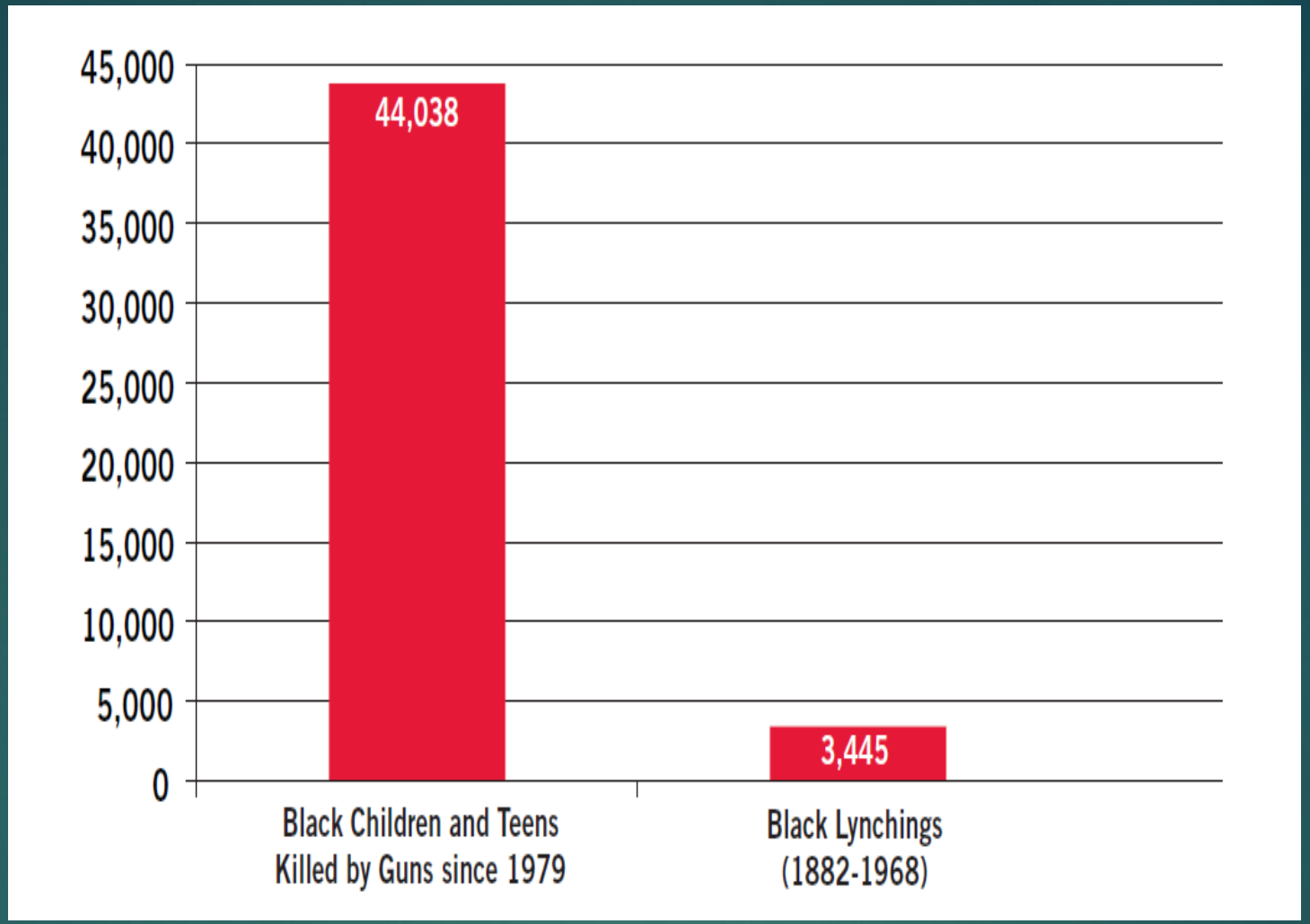
Guns at home: Children

- One-third of homes with children have guns, with many guns stored unsafely.
- 55% of U.S. homes with children and firearms have one or more firearms in an unlocked place

- 1500 to 3000 children are treated in hospital ERs for unintentional firearm injuries each year
- 1 child dies every 3 days in gun accidents in homes in US (usually at the hands of a brother or friend)
- 1 child or teenager dies every 3 hours in homes and on street (87% of all children under 15 killed by guns in 23 industrialized countries die in USA); injured by



Children's Defense Fund, 2012



Firearm homicide is the leading cause of death for African Americans ages 1-44.

African Americans suffer 54% of all firearm homicides.

Penetrating TBI

- Brain damage depends on energy translated to brain
- Teuber: 85% of WWII TBI employed 20 years later (bias: low survival of more severe TBI then)
- Puncture wounds: clean, damage in a focal pathway, rest intact
- Shock wave/pressure effects: ischemic and edema affects, diffuse effects

Penetrating TBI 2

- Increased seizure rates (2-5%; half with 24 hours):
- 33% of WWI and WWII with penetrating TBI
- 53% of Vietnam TBI had seizures (15 years later: motor slowing and word list recall impaired)
- Focal seizures in 75%
- Left hippocampus most susceptible to sz (Complex Partial szs mostly)

Shock Wave TBI: IUDs in Iraq War

- The detonation of an explosion generates a blast wave of high pressure that spreads out at 1,600 feet per second from the point of explosion and travels hundreds of yards. The lethal blast wave is a two-part assault that rattles the brain against the skull. The initial shock wave of very high pressure is followed closely by the "secondary wind": a huge volume of displaced air flooding back into the area, again under high pressure.

- No helmet or armor can defend against such a massive wave front.

Shock wave 2

- Shock waves damage the brain at a microscopic, sub-cellular level. When the sound wave moves through the brain, it causes little gas bubbles to form. When they pop, it leaves a cavity
- As many as one-third of all combat forces are at risk of TBI. 30% of all VA diagnosis after combat
- Soldiers walking away from IED blasts have discovered that they often suffer from memory loss, short attention spans, muddled reasoning, headaches, confusion, anxiety, depression and irritability.

Coma
(Alexander, ’82)

- 1 - Coma: unresponsive, eyes closed
- 2 - Unresponsive Vigilance: unresponsive, eyes opening, off sleep- wake cycle; 2% stay in persistent vegetative state
- 3 - Mute responsiveness: simple commands, sparse speech, elicit hand movements
- 4 - Confusional state: poor attention and cognition, PTA, hypo/hyper arousal
- 5 - Emerging Independence: continent, self care, but impaired STM, limited insight, poor cognition, inappropriate behavior
- 6 - Competence: higher level cognitive deficits, personality change, more impulsivity

TBI and Substance Abuse: Alcohol Does not Help

- 16-66% have pre-injury alcohol problems
- 36-51% BAL at injury ≥ .10
- 30-50% have post-injury problems
- 9-37% have history of other drug abuse; 12% have post injury use
- Higher rates of medical complications
- Longer periods of post-injury agitation
- Poorer cognitive functioning at discharge
- Greater neurological impairment

Long Term Complications of Alcohol

- Increased risk of another TBI
- Lowered seizure threshold
- Lowered levels of recovery
- Degradation of rehabilitation gains
- Increased risk of psychological adjustment difficulties
- Exacerbation of premorbid behavioral characteristics (impulsivity, risk taking, psychiatric disorders)

Need for Comprehensive Rehabilitation

- Physical Therapy
- Occupational Therapy
- Speech Therapy
- Medical Management
- Psychological/Neuropsychological
- Emotional/Psychiatric Management as appropriate
- Family Support
- Case Management

Determining Recovery Potential

- Poorer recovery associated with:
  - Lower Glasgow Coma Scale (GCS) Score;
  - Longer coma duration (greater than 4weeks);
  - Longer duration of Post Traumatic Amnesia (PTA)(good recovery unlikely when <3months)
  - Older age assoc. with worse outcomes
  - Neuroimaging features (presence of SAH, cisternal effacement, significant midline shift, EDH or SDH on acute care CT = worse outcomes).

Recovery

- Variables: age, severity of lesion, time since injury, premorbid patterns
- Rapid recovery in first 3 to 6 months following PTA; consistent evidence that maximum recovery in 6-12 months

Recovery

- Uncomplicated mTBI has been shown to have little to no impact on the majority (i.e., 95% to 97%) of patients' behavior following a short (i.e., few weeks) recovery time.
- Bulk of recovery in first year for more serious TBI

Recovery 2

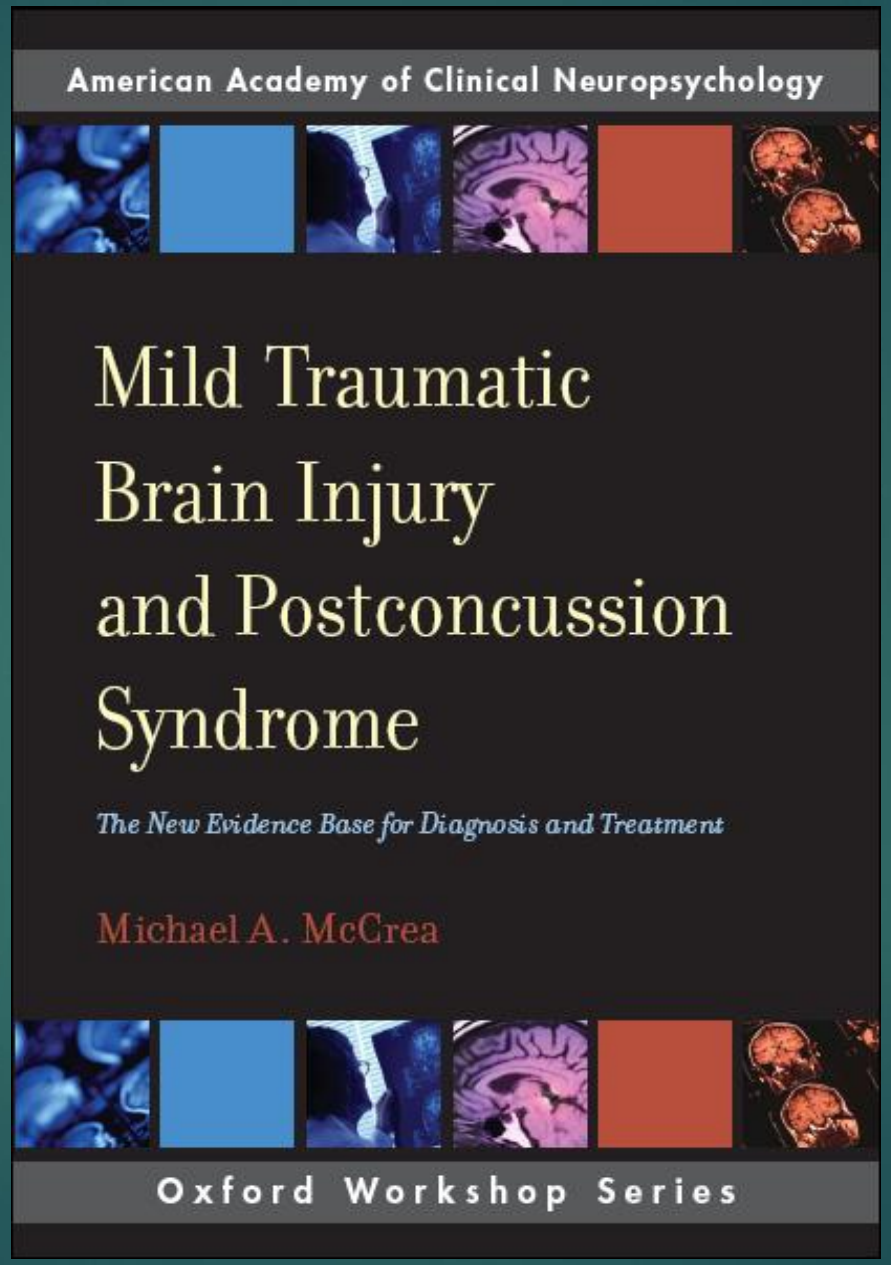
- Memory recovery slower than general intellectual ability and final memory ability lower than other intellectual ability
- Quality of life is significantly poorer post mod to severe TBI
- 2-4 years: in 50 %, social and leisure activities decreased and relatives report strain

Outcome

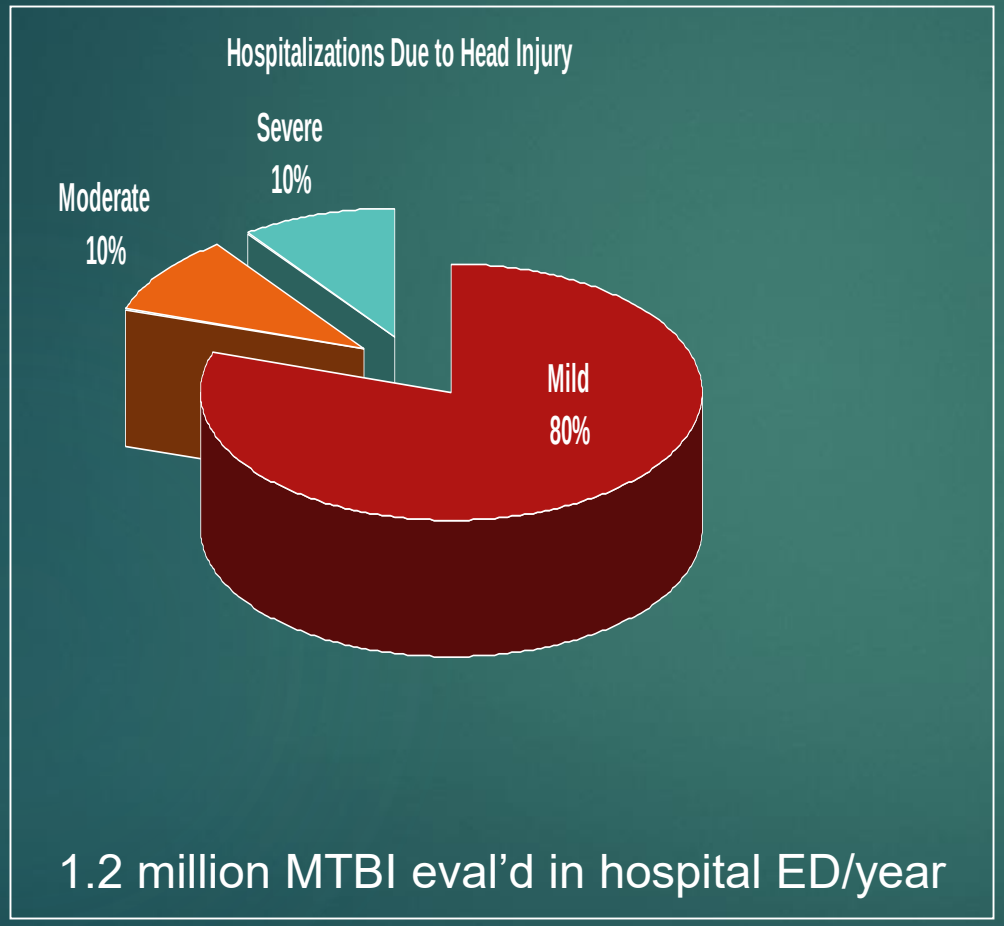
- Pre-injury status: personality, coping style, chemical dependency, education, IQ
- Rehabilitation: earlier the better:
  - If you start before 35th day post injury, you halve the hospital and rehab days
- PTA and outcome:
  - 83% have less than 2 weeks PTA: at 12 months, HA, dizziness, STM, tinnitus
  - 28 days + PTA: 27% good outcome, 43% moderate, 30% severe

Outcome 2

- Frontal damage and outcome:
  - 5 year outcome: 50% have mood and temper problems, memory impaired, concentration problems
- Personality and Interpersonal changes:
  - Loss of friends and isolation


American Academy of Clinical Neuropsychology
Mild Traumatic Brain Injury and Postconcussion Syndrome
The New Evidence Base for Diagnosis and Treatment
Michael A. McCrea
Oxford Workshop Series

Significance of MTBI: “Silent Epidemic”



1.2 million MTBI eval’d in hospital ED/year

- > 2.5 million MTBI/yr U.S
- True incidence unclear: 30-50% no medical attention; < 10% of ED MTBI admitted
- Total incidence as high as 500/100K population
- Few to neuropsychologist
- Persistent symptoms and disability (“PCS”)
- Costly public health issue (> $30 billion)

What is mTBI

- Traumatically induced physiologic disruption of brain function as indicated by at least one of the following:
  - Any period of loss of consciousness
  - Any loss of memory for events immediately before or after the accident
  - Any alteration in mental state at the time of the accident
  - Focal neurological deficits that may or may not be transient

- Severity of the injury does not exceed:
  - Loss of consciousness of 30 min
  - GCS score of 13-15 after 30 min
  - Posttraumatic amnesia of 24 hr.

Complicated mTBI

When any clinical neuroimaging findings are present following a mTBI, the classification changes to “complicated mTBI,” which has a 6-month outcome more similar to moderate TBI.

TBI Prognosis:
Some Things Are Crystal Clear…

Injury Severity is Strongest Predictor of Recovery after *moderate and severe* TBI

… And Some Things Are Not So Clear

MTBI is a Different Animal All Together

"*All MTBI Are Not Created Equally*" –

Grant Iverson, 2005

Biomechanics of MTBI:
Establishing a minimal biomechanical threshold

How much is enough to cause brain injury?

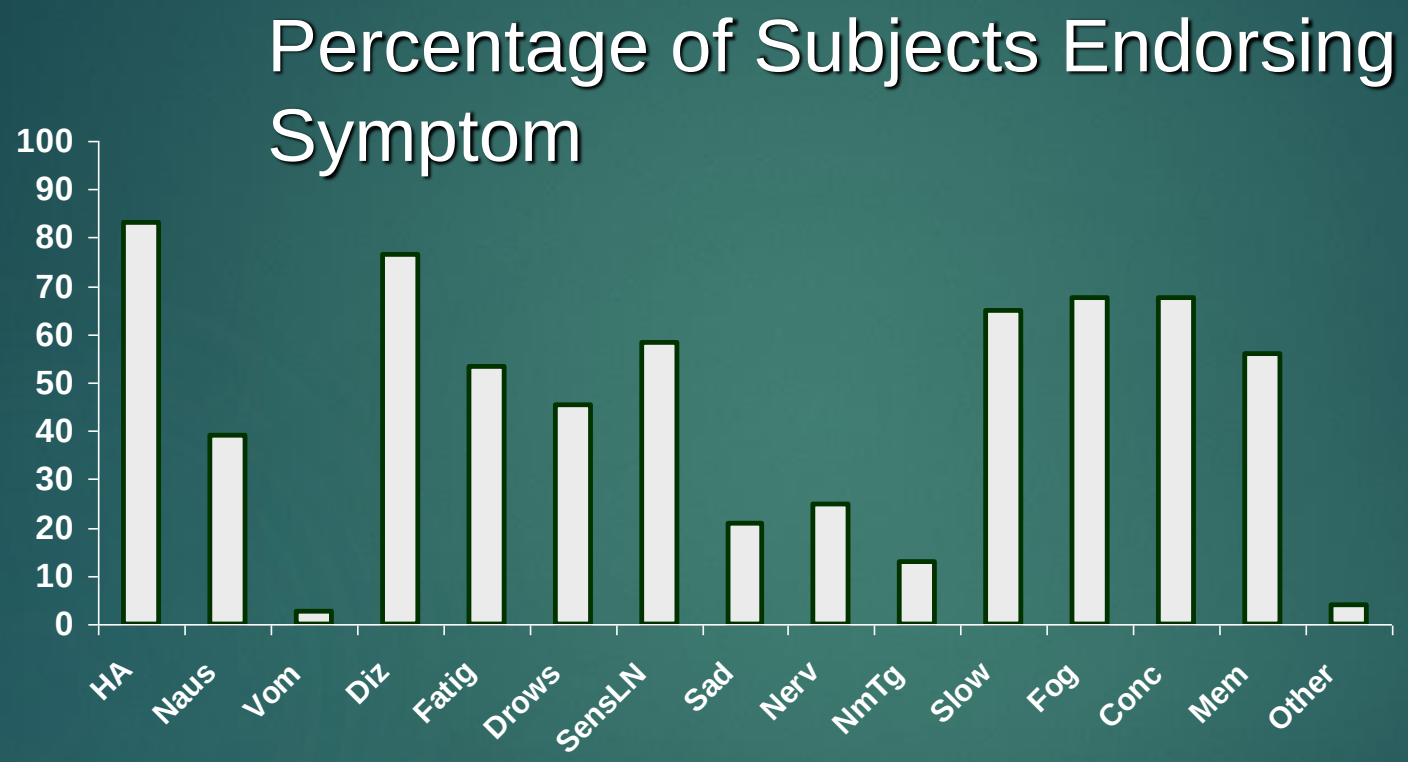
Accelerometry Instrumentation

Measures and records blows to the head:

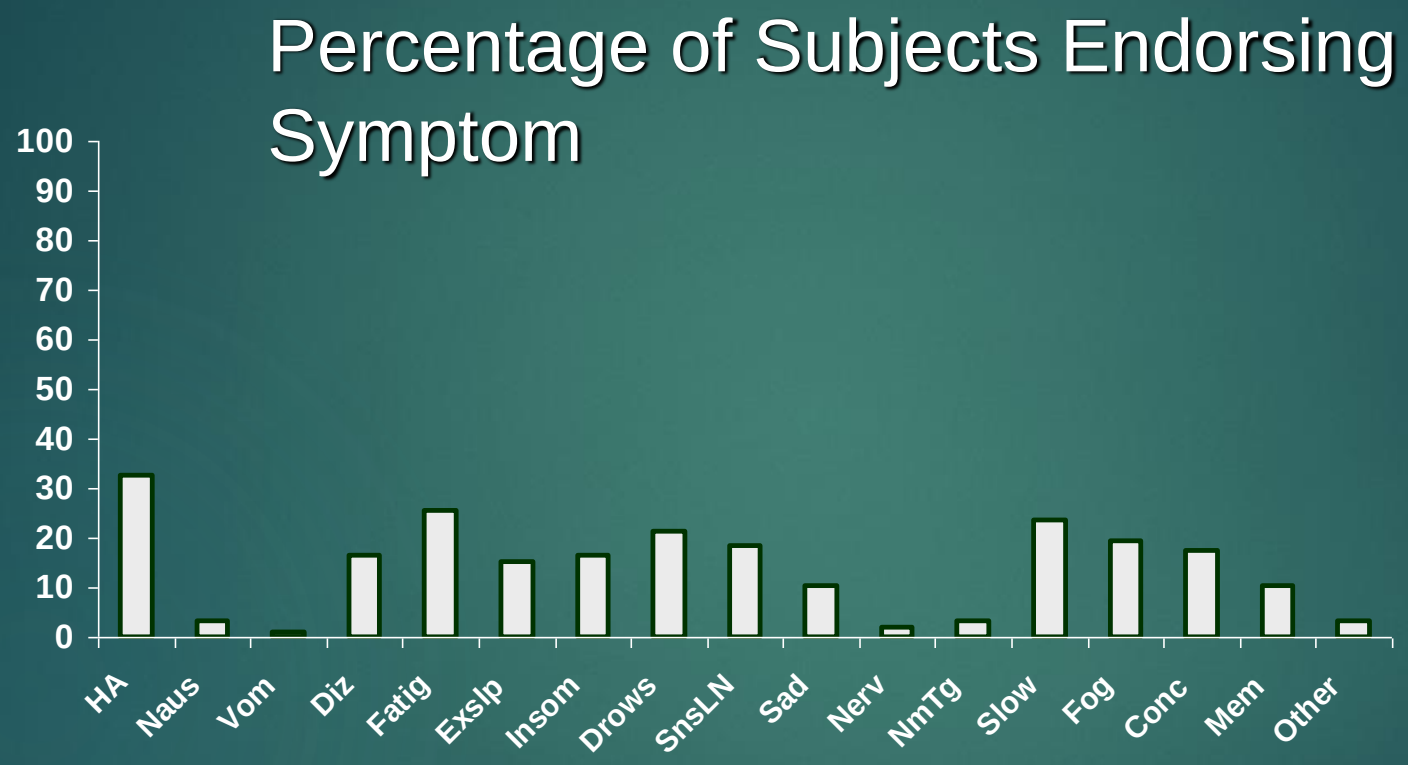
- Impact location
- Impact magnitude
- Impact duration
- Linear and angular acceleration components

-Exact times of impacts

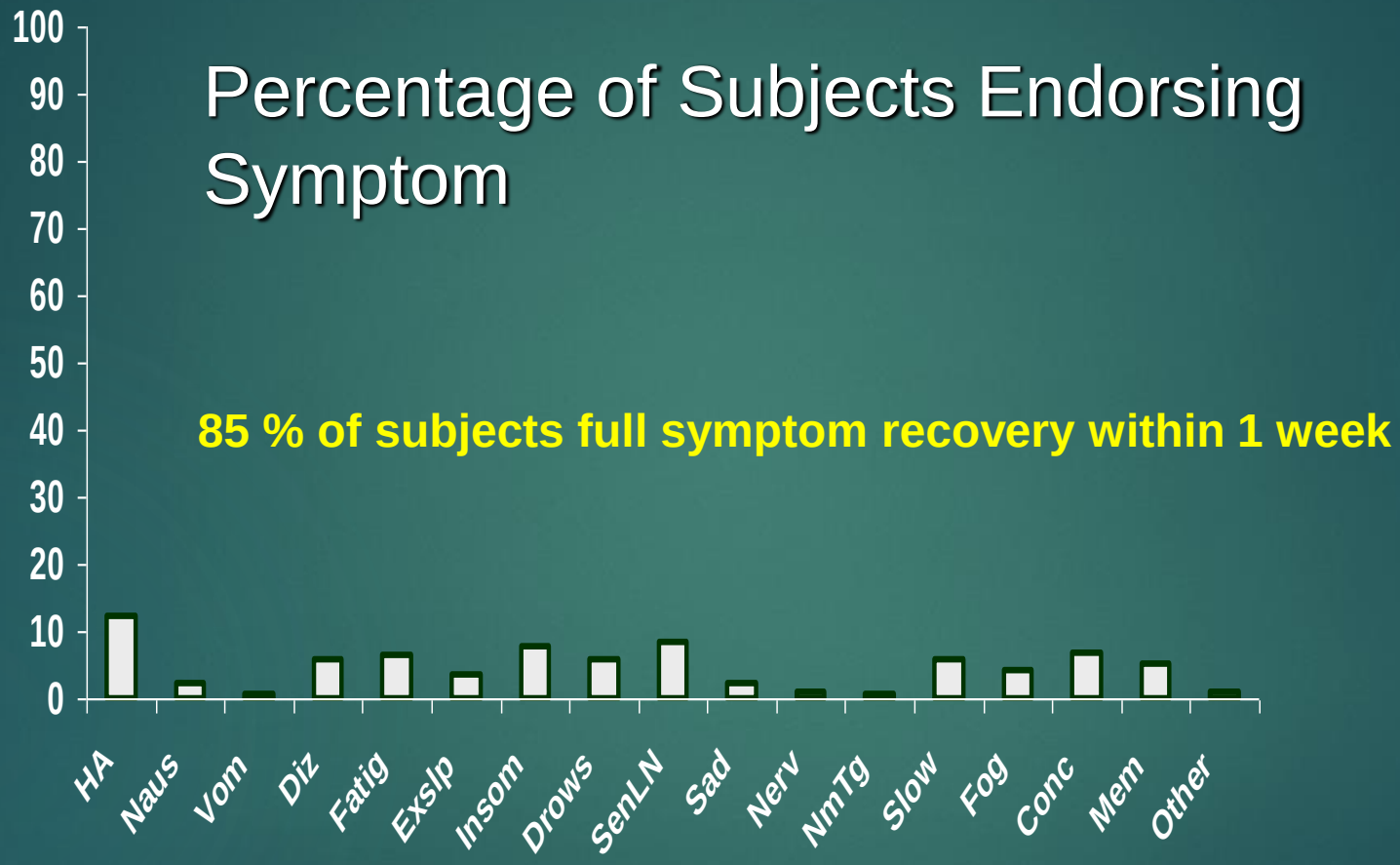
Immediate Symptoms

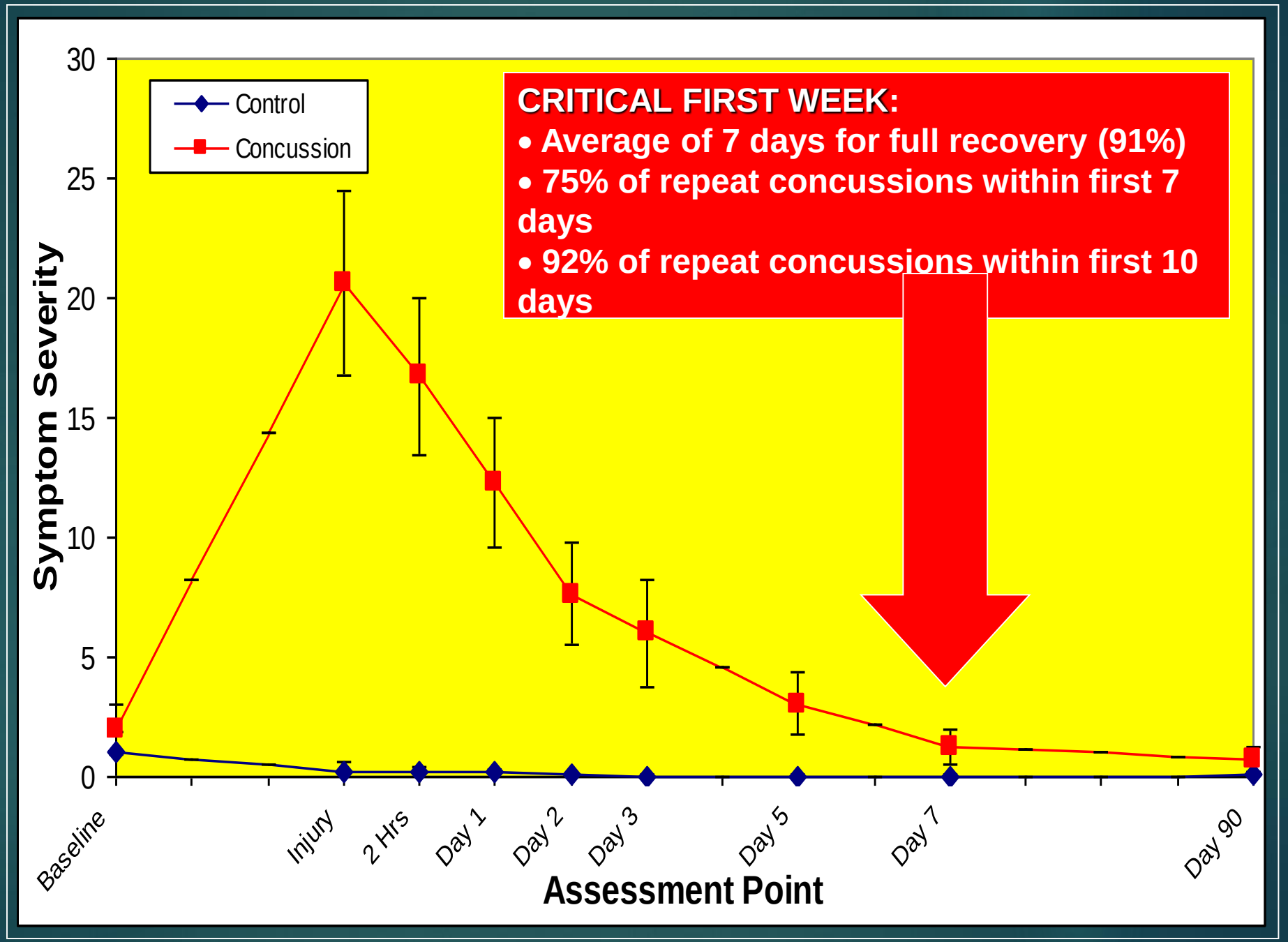


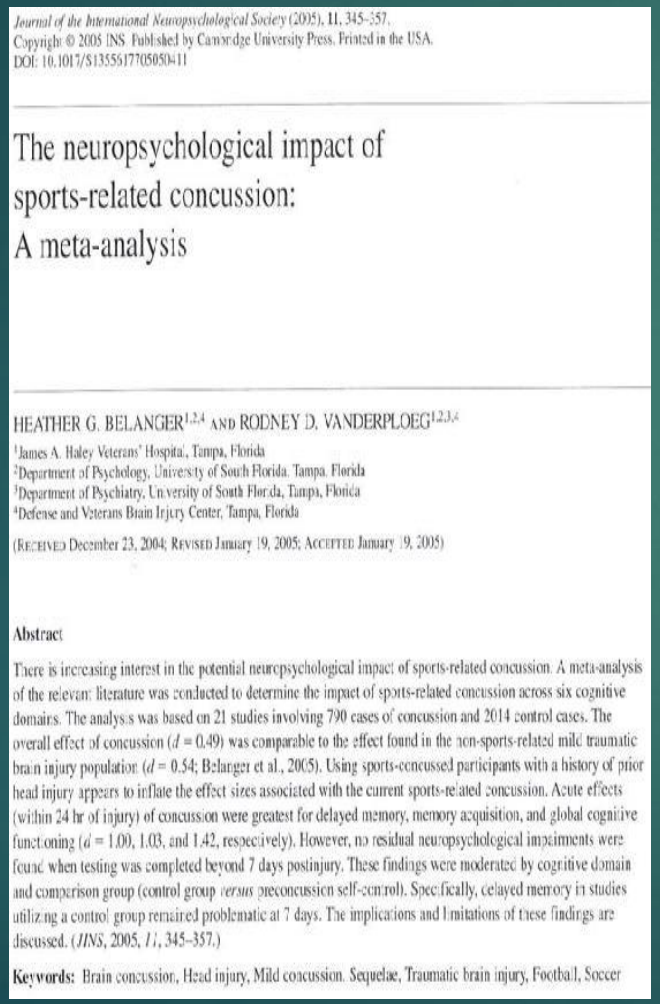
Day 3 Symptoms



Day 7 Symptoms




CRITICAL FIRST WEEK:
• Average of 7 days for full recovery (91%)
• 75% of repeat concussions within first 7 days
• 92% of repeat concussions within first 10 days
Control
Concussion
Symptom Severity
30
25
20
15
10
5
0
Baseline
Injury
2 Hrs
Day 1
Day 2
Day 3
Day 5
Day 7
Day 90
Assessment Point



Journal of the International Neuropsychological Society (2005), 11, 345–357.
Copyright © 2005 INS. Published by Cambridge University Press. Printed in the USA.
DOI: 10.1017/S1355617705050411

The neuropsychological impact of sports-related concussion: A meta-analysis

HEATHER G. BELANGER[1,2,4] AND RODNEY D. VANDERPLOEG[1,2,3,4]

[1]James A. Haley Veterans' Hospital, Tampa, Florida
[2]Department of Psychology, University of South Florida, Tampa, Florida
[3]Department of Psychiatry, University of South Florida, Tampa, Florida
[4]Defense and Veterans Brain Injury Center, Tampa, Florida

(RECEIVED December 23, 2004; REVISED January 19, 2005; ACCEPTED January 19, 2005)

Abstract

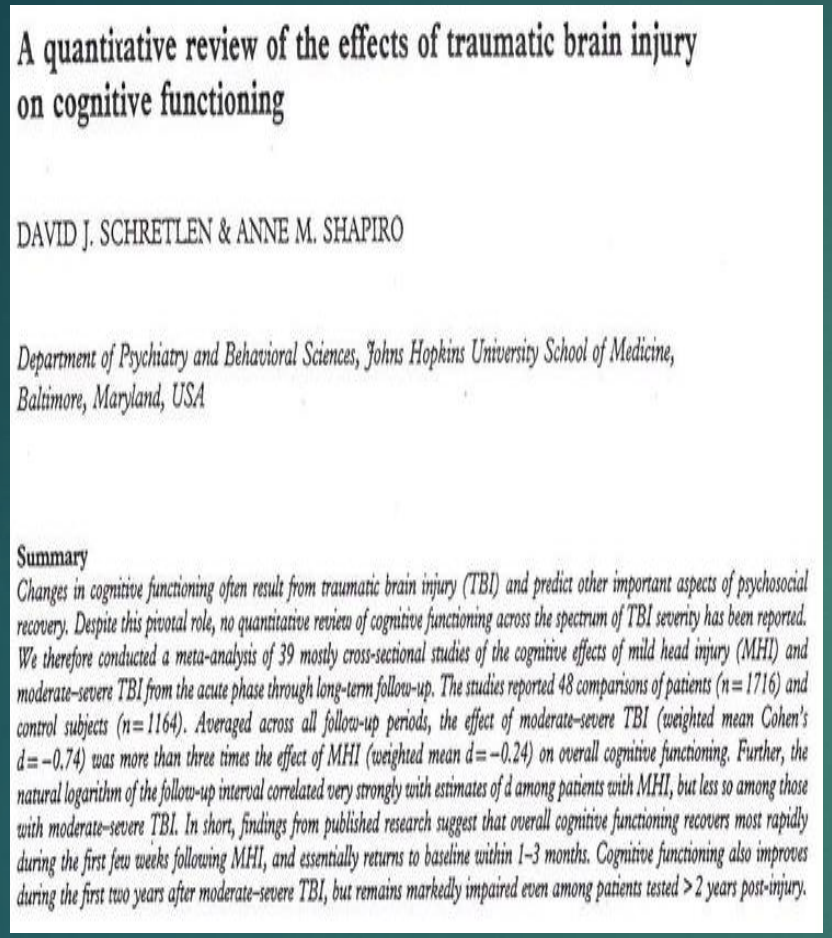
There is increasing interest in the potential neuropsychological impact of sports-related concussion. A meta-analysis of the relevant literature was conducted to determine the impact of sports-related concussion across six cognitive domains. The analysis was based on 21 studies involving 790 cases of concussion and 2014 control cases. The overall effect of concussion ($d = 0.49$) was comparable to the effect found in the non-sports-related mild traumatic brain injury population ($d = 0.54$; Belanger et al., 2005). Using sports-concussed participants with a history of prior head injury appears to inflate the effect sizes associated with the current sports-related concussion. Acute effects (within 24 hr of injury) of concussion were greatest for delayed memory, memory acquisition, and global cognitive functioning ($d = 1.00$, 1.03, and 1.42, respectively). However, no residual neuropsychological impairments were found when testing was completed beyond 7 days postinjury. These findings were moderated by cognitive domain and comparison group (control group *versus* preconcussion self-control). Specifically, delayed memory in studies utilizing a control group remained problematic at 7 days. The implications and limitations of these findings are discussed. (*JINS*, 2005, *11*, 345–357.)

Keywords: Brain concussion, Head injury, Mild concussion, Sequelae, Traumatic brain injury, Football, Soccer

- *Meta-analysis:* *21 studies, 790 concussions, 2014 controls*
- Acute effects (w/n 24 hrs) greatest for delayed memory (d=1.00), memory acquisition (d=1.03), and global cognitive functioning (d=1.42)
- No residual neuropsych impairment > 7 days postinjury

Conclusions from Dikmen et al. 1997

- mTBI in general is not associated with long term persistent neuropsychological impairments
- In cases of poor outcome from mTBI, other causes need to be explored



A quantitative review of the effects of traumatic brain injury on cognitive functioning

DAVID J. SCHRETLEN & ANNE M. SHAPIRO

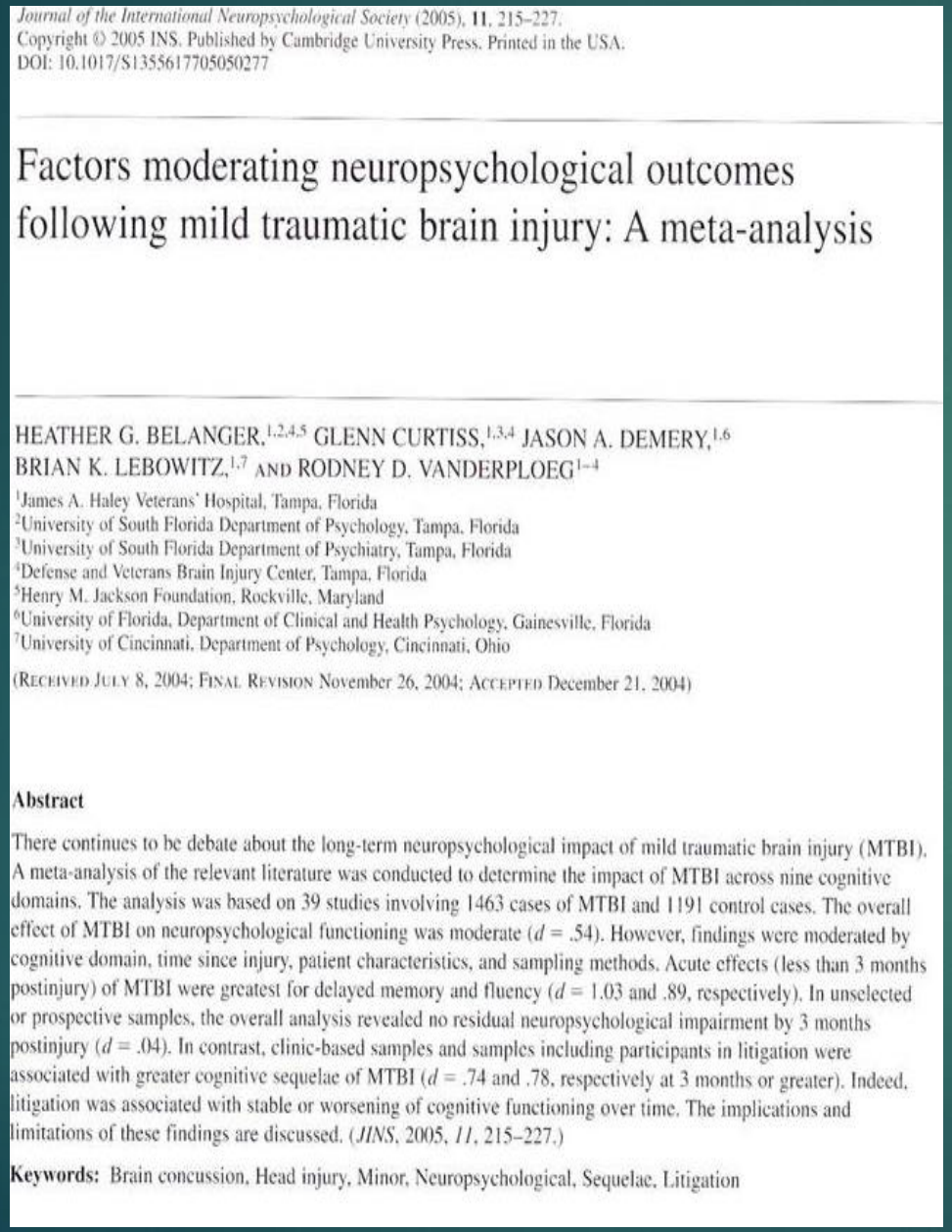
Department of Psychiatry and Behavioral Sciences, Johns Hopkins University School of Medicine, Baltimore, Maryland, USA

Summary

Changes in cognitive functioning often result from traumatic brain injury (TBI) and predict other important aspects of psychosocial recovery. Despite this pivotal role, no quantitative review of cognitive functioning across the spectrum of TBI severity has been reported. We therefore conducted a meta-analysis of 39 mostly cross-sectional studies of the cognitive effects of mild head injury (MHI) and moderate–severe TBI from the acute phase through long-term follow-up. The studies reported 48 comparisons of patients (n = 1716) and control subjects (n = 1164). Averaged across all follow-up periods, the effect of moderate–severe TBI (weighted mean Cohen's d = −0.74) was more than three times the effect of MHI (weighted mean d = −0.24) on overall cognitive functioning. Further, the natural logarithm of the follow-up interval correlated very strongly with estimates of d among patients with MHI, but less so among those with moderate–severe TBI. In short, findings from published research suggest that overall cognitive functioning recovers most rapidly during the first few weeks following MHI, and essentially returns to baseline within 1–3 months. Cognitive functioning also improves during the first two years after moderate–severe TBI, but remains markedly impaired even among patients tested > 2 years post-injury.

Summary

- 39 studies, 48 MTBI (n=1716) vs. control (n=1164) comparisons;
- "Moderate" ↓ in overall cognitive functioning <7 days post-injury
- Learning/memory, RT, and attention show greatest acute effects
- Effects diminish by 7-29 days, disappear by 30-89 days post
- "Recovery" of overall functioning follows a logarithmic course with rapid recovery in first few weeks following MTBI and returns to baseline within one to three months



Journal of the International Neuropsychological Society (2005), **11**, 215–227.

DOI: 10.1017/S1355617705050277

Factors moderating neuropsychological outcomes following mild traumatic brain injury: A meta-analysis

HEATHER G. BELANGER,[1,2,4,5] GLENN CURTISS,[1,3,4] JASON A. DEMERY,[1,6] BRIAN K. LEBOWITZ,[1,7] AND RODNEY D. VANDERPLOEG[1–4]

[1]James A. Haley Veterans' Hospital, Tampa, Florida
[2]University of South Florida Department of Psychology, Tampa, Florida
[3]University of South Florida Department of Psychiatry, Tampa, Florida
[4]Defense and Veterans Brain Injury Center, Tampa, Florida
[5]Henry M. Jackson Foundation, Rockville, Maryland
[6]University of Florida, Department of Clinical and Health Psychology, Gainesville, Florida
[7]University of Cincinnati, Department of Psychology, Cincinnati, Ohio

(RECEIVED JULY 8, 2004; FINAL REVISION November 26, 2004; ACCEPTED December 21, 2004)

Abstract

There continues to be debate about the long-term neuropsychological impact of mild traumatic brain injury (MTBI). A meta-analysis of the relevant literature was conducted to determine the impact of MTBI across nine cognitive domains. The analysis was based on 39 studies involving 1463 cases of MTBI and 1191 control cases. The overall effect of MTBI on neuropsychological functioning was moderate (d = .54). However, findings were moderated by cognitive domain, time since injury, patient characteristics, and sampling methods. Acute effects (less than 3 months postinjury) of MTBI were greatest for delayed memory and fluency (d = 1.03 and .89, respectively). In unselected or prospective samples, the overall analysis revealed no residual neuropsychological impairment by 3 months postinjury (d = .04). In contrast, clinic-based samples and samples including participants in litigation were associated with greater cognitive sequelae of MTBI (d = .74 and .78, respectively at 3 months or greater). Indeed, litigation was associated with stable or worsening of cognitive functioning over time. The implications and limitations of these findings are discussed. (*JINS*, 2005, *11*, 215–227.)

Keywords: Brain concussion, Head injury, Minor, Neuropsychological, Sequelae, Litigation

- Meta-analysis: 39 studies, 1463 MTBI cases, 1191 controls
- Overall effect of MTBI on neuropsychological functioning moderate (d=.54)
- Acute: greatest affect on memory (d=1.03), fluency (d =.89)
- Unselected or prospective samples: No residual NP effects by 3 mos. (d=.04)
- Clinic samples (.74) & litigants (.78) at 3 mos.
- Litigation associated with stable *or worsening* cognition

Outcome Research: Mild TBI

- Appropriately designed research studies indicate that virtually 100% mild head injured subjects show no cognitive impairment within about 3 months to a year (outside) post-injury
- EXCELLENT Prognosis for mTBI

Symptom Persistence in mTBI

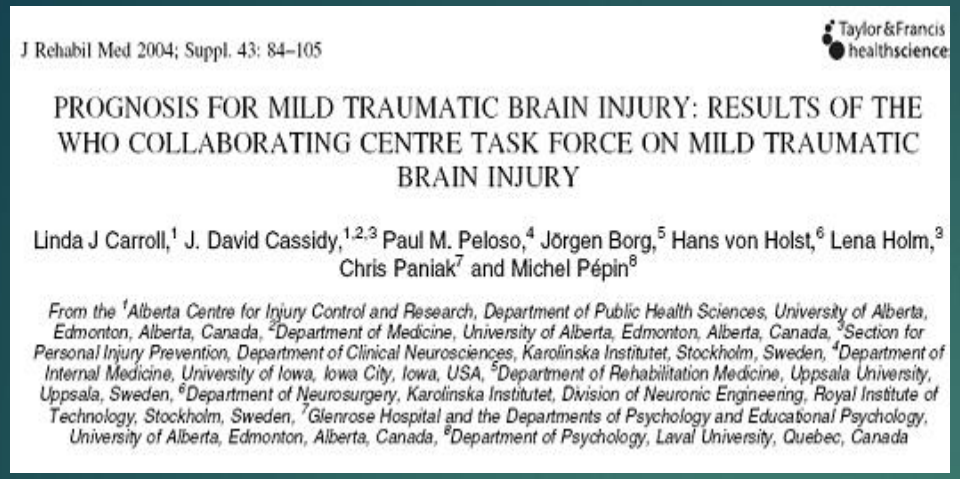
- When sxs persist beyond 3 months, diagnostic & treatment challenges are formidable due to the fact that persistence is multifactorial & typically not primarily associated with brain injury.

Risk factors for mTBI sx persistence

- Prior hx of neurological injury/reduced cerebral reserve
- Hx of recent or multiple mTBIs
- Hx of chronic medical condition
- Polytrauma &/or chronic pain
- *Misattribution bias*
- Potential for secondary gain (litigation, seeking disability)
- History of psychopathology (stress, depression, anxiety, SA)

Functional Outcome After MTBI

- Overwhelming majority of MTBI resume normal independent social, occupational, educational function within days to weeks of injury
- Non-injury factors associated with poor functional outcome
- Higher risk of depression, anxiety (12-44%), which receive insufficient attention



J Rehabil Med 2004; Suppl. 43: 84–105

Taylor & Francis healthscience

PROGNOSIS FOR MILD TRAUMATIC BRAIN INJURY: RESULTS OF THE WHO COLLABORATING CENTRE TASK FORCE ON MILD TRAUMATIC BRAIN INJURY

Linda J Carroll,[1] J. David Cassidy,[1,2,3] Paul M. Peloso,[4] Jörgen Borg,[5] Hans von Holst,[6] Lena Holm,[3] Chris Paniak[7] and Michel Pépin[8]

From the [1]Alberta Centre for Injury Control and Research, Department of Public Health Sciences, University of Alberta, Edmonton, Alberta, Canada, [2]Department of Medicine, University of Alberta, Edmonton, Alberta, Canada, [3]Section for Personal Injury Prevention, Department of Clinical Neurosciences, Karolinska Institutet, Stockholm, Sweden, [4]Department of Internal Medicine, University of Iowa, Iowa City, Iowa, USA, [5]Department of Rehabilitation Medicine, Uppsala University, Uppsala, Sweden, [6]Department of Neurosurgery, Karolinska Institutet, Division of Neuronic Engineering, Royal Institute of Technology, Stockholm, Sweden, [7]Glenrose Hospital and the Departments of Psychology and Educational Psychology, University of Alberta, Edmonton, Alberta, Canada, [8]Department of Psychology, Laval University, Quebec, Canada

We searched the literature on the epidemiology, diagnosis, prognosis, treatment and costs of mild traumatic brain injury. Of 428 studies related to prognosis after mild traumatic brain injury, 120 (28%) were accepted after critical review. These comprise our best-evidence synthesis on prognosis after mild traumatic brain injury. There was consistent and methodologically sound evidence that children's prognosis after mild traumatic brain injury is good, with quick resolution of symptoms and little evidence of residual cognitive, behavioural or academic deficits. For adults, cognitive deficits and symptoms are common in the acute stage, and the majority of studies report recovery for most within 3–12 months. Where symptoms persist, compensation/litigation is a factor, but there is little consistent evidence for other predictors. The literature on this area is of varying quality and causal inferences are often mistakenly drawn from cross-sectional studies.

Key words: mild traumatic brain injury, epidemiology, prognosis, recovery.

J Rehabil Med 2004; suppl. 43: 84–105

Correspondence address: Linda J Carroll, Department of Public Health Sciences, University of Alberta, 3080 RTF, 8308-114 St, Edmonton, AB, Canada, T6G 2E1.
E-mail: lcarroll@ualberta.ca

Science of MTBI Recovery

- Clear, sound evidence
- Kids: rapid recovery, no residual cognitive, behavioral, academic deficits
- Adults: rapid symptom, cognitive recovery; no impairments 3-12 mos
- Non-injury factors predict persistent symptoms

Natural History of MTBI
Main Conclusions

1. Symptom recovery in days to weeks in most cases
2. Measurable cognitive impairments w/o LOC, PTA, neuro
3. Favorable cognitive recovery overlapping symptom recovery; no permanent impairment
4. Neurophysiological recovery c/w clinical recovery (days to wks)
5. Focal lesions indicate more severe gradient, not perfectly predictive of outcome
6. Favorable functional outcome is expected
7. Non-injury factors best predictors of poor outcome
8. Exceptions to the rules: Recurrent MTBI

mTBI

- LOC of greater than 1-2 minutes atypical; 20-30 minutes rare
- Brief LOC and PTA may indicate more severe mTBI, but is not predictive of eventual recovery
- Symptoms: vertigo (most common sx), tinnitus, hearing loss, diplopia, attention↓; headache (10%): sx that lingers the longest
- Gradual symptom recovery over 7-10 days in 80-90%; 97% by 1 month
- Symptoms beyond this time range are nonspecific to mTBI and often due to non-injury related factors (difference between athletes and litigators)

mTBI 2

- CT usually negative
- Complicated mTBI (structural lesions on imaging) may increase risk of slow or incomplete recovery, but is not predictive of outcome.
- Subjective symptoms disproportionate to objective evidence only in litigators
- Less severe complain more than more severe TBI
- LOC unrelated to presence and severity of NP deficits
- Levin: at 3 and 6 months, normal MRI, but hypoperfusion on SPECT related to NP deficits

mTBI 3

- Measurable NP impairments evident in acute phase in absence of LOC, amnesia, or focal neurologic deficits.
- Several meta-analyses and prospective studies indicate that uncomplicated mTBI is most often followed by a favorable course of NP recovery over period of days to weeks with no indication of permanent impairment on NP testing by 3 months post-injury.

McCrea, 2008

mTBI 4

- Functional recovery after mTBI follows same course as symptom and NP recovery.
- Overwhelming majority of mTBI pts return to normal independent functioning, social functioning, and work within days to weeks after injury.
- Non-injury related factors often play significant role in functional outcome.
- mTBI pts with preexisting medical or psychological problems, high stress, and poor social support after injury are at risk for poorer functional outcomes.

mTBI 5

- Single, uncomplicated mTBI is a transient neurologic event followed by gradual and rapid recovery in sxs, cognition, and functioning in vast majority of cases.
- Recurrent mTBI may be associated with longer recovery and persistent symptoms, and potential of increasing lifetime risk of psychiatric and neurologic problems.

Postconcussion Symptoms

- Physical
  - Headache, dizziness, fatigue, noise/light intolerance, insomnia
- Cognitive
  - Memory complaints, poor concentration
- Emotional
  - Depression, anxiety, irritability, lability

PCS-Like Complaints of NP Dysfunction

- Common
- Nonspecific
- Potentially related to non-neurological factors (anxiety, depression, fatigue, stress)
- Correlate better with distress than with objective indicators of CNS injury
- Susceptible to attribution bias

What is “PCS”?

- Post Concussive Syndrome
- DSM-5 mild NCD due to TBI
- ICD-10: F07.2 (part of class of disorders with “a demonstrable etiology in cerebral disease, brain injury, or other insult leading to cerebral dysfunction”).
  - Def: A syndrome that occurs following head trauma (usually sufficiently severe to result in loss of consciousness) and includes a number of disparate symptoms such as headache, dizziness, fatigue, irritability, difficulty in concentration and performing mental tasks, impairment of memory, insomnia, and reduced tolerance to stress, emotional excitement, or alcohol.

Non-specificity of PCS symptoms

- Symptoms are *not specific* to concussion/TBI; e.g.:
  - Trahan et al, 2001: Pts with depression endorse significantly more PCS Sxs than pts with mTBI.
  - Lees-Haley et al, 2001: Non-TBI personal injury claimants endorse PCS symptomatology at high rates, comparable on many symptoms to mTBI claimants (e.g., concentration impairments 63% mTBI, 65% other).
  - Iverson & McKraken, 1997: Chronic pain pts endorse PCS Sxs at high rate (34% meet criteria for PCS; 81% endorsing 3+ symptoms)
  - Gouvier et al., 1988: High base rates of “PCS” symptoms in normal (college) population

Prevalence of “PCS” Symptoms

	Headache	Dizziness	Irritability	Memory problems	Conc. problems
College students[1]	36%	18%	36%	17%	42%
Chronic pain[2]	80%	67%	49%	33%	63%
Depressed[3]	37%	20%	52%	25%	54%
PI claimants (non tbi)[4]	77%	41%	63%	46%	71%
mTBI[5]	42%	26%	28%	36%	25%

1. Sawchyn et al., 2000; 2. Radanov et al., 1992; 3.Trahan et al., 2001; 4. Dunn et al., 1995; 5. Ingebrigtsen et al., 1998

Contributing Risk Factors Account for Persistent Symptoms in cases of Mild Closed TBI

- Age
- Education
- Pre-existing conditions
  - Treatment for alcohol or substance abuse
  - CNS disorder (prior head injury)
  - Psychiatric condition (including PTSD)
  - Somatoform-Spectrum diagnoses

PCS: Neuropsychological Disorder

- Biopsychosocial basis for PCS (Iverson, Zasler, Lange)
- In most studies examining predictors of PCS symptomatology in TBI, injury severity is usually ***not*** predictive (sometimes relationship is found to be inverse), but non-injury variables are:
  - Blaming of other(s) for injury, Limited social support
  - Current levels depression/anxiety
  - Premorbid psychiatric Hx
  - Presence of PTSD
  - Somatization
  - Motivational factors (exaggeration,)

Implication: Psychological Theory and Treatment

Postconcussion Syndrome: Main Conclusions

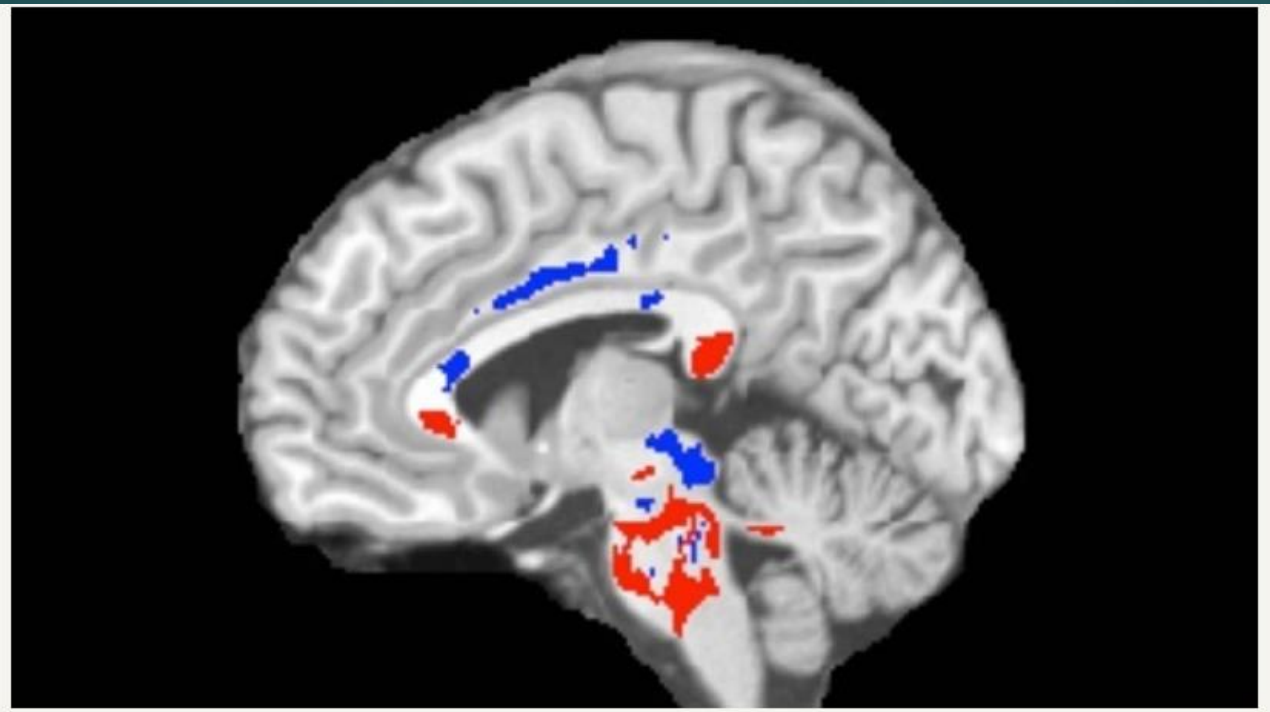
1. Sx-Based diagnosis of PCS problematic (poor reliability of criteria, nonspecificity of sx's)
2. PCS estimates severely inflated; true incidence ~ 1-5%
3. Science to rethink PCS: Neuropsychological Disorder
4. Psychological bases indicates psychological and educational interventions
5. Effective intervention will improve functional outcome and reduce disability from PCS
6. Need to rule out motivational factors
7. Neuropsychologists *the* key component

Warning: Commercial Re-Run

1. MTBI, more than any other clinical entity, is a *neuropsychological* construct
2. The contribution by neuropsychologists to MTBI research is unmatched by any other discipline
3. Neuropsychologists are uniquely suited to evaluate and treat MTBI
4. Neuropsychologists should not limit their role in MTBI just to neuropsych testing

Postconcussion Syndrome: Conclusions

- PCS as a neuropsychological disorder; 1-5% of mTBI
- While associated with transient neurological effects of mTBI, development and maintenance of persistent PCS are the result of psychological, psychosocial, and other non-mTBI-specific factors.
- More similar to somatoform disorders and PTSD
- Misattribution of common complaints to the head injury



A DTI scan of the brain of a patient with mild traumatic brain injury showing areas with low fractional anisotropy (FA) (red) and high FA (blue).

Photo reproduced from Strauss SB, Kim N, Branch CA. Bidirectional Changes in Anisotropy Are Associated with Outcomes in Mild Traumatic Brain Injury. AJNR Am J Neuroradiol 2016 Jun 9.

2016: mTBI biomarker?

- DTI scans of 39 patients diagnosed with mTBI within 16 days of the injury
- Follow-up assessments with 26 patients one year later found that those whose DTI scans showed abnormally high FA had better outcomes on tests of cognition and post-concussion symptoms. Study subjects who had high FA in the left frontal lobe and left temporal lobe performed better on tests of attention and those who had high FA in the right thalamus experienced fewer emotional post-concussion symptoms, for example.
- DTI imaging analyses of high FA in specific brain areas may be a good predictor of functional outcomes at one year after mild TBI.

Strauss S B, Kim N, Branch C A, et al. Bidirectional changes in anisotropy are associated with outcomes in mild traumatic brain injury. *AJNR Am J Neuroradiol*; Epub June 9 2016.

Moderate TBI

- 8-10% of all TBI
- 1-24 hrs. PTA, 9-12 GCS, <6 hours coma, skull fx
- 6 months: HA, STM, 2/3rds not working
- Functionally independent, but behavioral traces of prefrontal deficits, impulsivity, spontaneity/initiative↓, muted affect and empathy, STM ↓, planning ↓, poor self monitoring, loss of non-work activity
- It takes five years for the brain to return to the normal rate of loss due to aging after moderate to severe TBI

Severe TBI

- <10 % of all TBI
- Cognitive: poor attention, behavioral slowing, word finding ↓
- Memory: learning impaired, intrusions increased, worse in ETOH
- Frontal: executive dysfunction, neurogenic lack of awareness
- Talk better than they communicate
- Pragmatics impaired
- <25% return to work, primarily because of executive dysfunction.

Cognitive Impairments after TBI

- Post Traumatic Amnesia
- Information processing and attention;
- Anosognosia (unawareness of deficits);
- Intellectual functioning
- Memory
- Confabulation and delusions
- Spatial Cognition
- Chemical Senses (Olfaction and Taste)
- Executive Functions
- Social Cognition and Behavior

Symptom Validity & Suboptimal Performance

- Base rate of exaggeration estimated to be 27% of all TBI cases (Rohling et at., 2000)
- Base rate for symptom exaggeration higher in patients with mild TBI versus severe TBI (54% vs. 21%)
- Impairment equals 1.2 to 1.5 SD units
- mTBI patients often do worse than severe TBI and extremely low IQ kids

NP Assessment

- Heterogeneity in moderate to severe TBI = no single pathognomonic NP profile
- Some common patterns
- Some mTBI patterns immediately after injury; but majority have rapid improvement

NP Test Results 1

- Intelligence:
  - hold tests (overlearned skills, information, vocabulary) normal
  - subtests with processing speed & novel PS ↓
  - Infant & early childhood injury: lower IQ
- Attention/Concentration:
  - ubiquitous ↓ in PTA
  - complex attention can remain impaired
  - acquired ADHD
- Processing Speed: ↓ ↓
  - The most commonly affected cognitive ability, especially early on
  - WAIS Coding, Symbol Digits, TMT ↓ ↓

NP Test Results 2

- Language:
  - Persistent aphasia uncommon unless focal injury
  - Dysnomia & phonological processing in diffuse injury; language pragmatics ↓ common
  - Complex language impacted
- Visuospatial:
  - If more focal injury in adults; in kids, following both focal and diffuse
  - Need to clarify EF deficit contributions

NP Test Results 3

- Memory:
  - Frontal & Temporal injuries common = reduced declarative memory, both for encoding, retrieval, & organizational strategies
- Executive Functions:
  - Common frontal damage = EF deficits
  - Effects functional recovery
  - Reduced emotional regulation

NP Test Results 4

- Sensorimotor Functions:
  - Motor & sensory deficits if focal injury
  - Common reduced psychomotor speed & coordination in mod/severe TBI and in children with diffuse injury
- Emotion, Social Cognition & Personality:
  - Depression & anxiety common following TBI
  - Frontal deficits can produce apathy & mimic bipolar, OCD
  - Anosognosia; reduced self control, impulsivity, fatigue,
  - Reduced social skills, social cognition, moral reasoning

Frontal Consequences of TBI

Difficulty with “executive control” functions

- – Short-term memory
- – Money management
- – Time management
- – Judgment
- – Concentration
- – Planning
- – Frequent confusion or frustration

Psychosocial and Emotional Functions ↓↓

- Self-monitoring
- Reading social “cues”
- Impulse control
- Mood swings
- Anxiety
- Depression
- Anger management
- Sudden agitation
- Low self-esteem
- Substance abuse

Difficulties with physical abilities

- -- Motor coordination
- – Headaches **
- – Fatigue
- – Seizures
- – Muscle spasticity
- – Hearing, visual, and speech impairments
- – Attention Impairment
- – Bladder & Bowel
- – Dizziness
- – Nausea
- – Vertigo
- – Vomiting

Behavioral Deficits in TBI

- Disorientation and confusion initially
- Inflexibility
- Impulsivity
- Neurogenic lack of awareness
- Poor affective modulation

Anoxic Brain Damage

- 31 pt study, comatose >24 hours post cardiopulmonary arrest:
  - 17 regained ambulation
  - 20 regained oral communication
  - 13 regained full independence
  - 2 regained previous cognitive ability
  - 1 regained previous level of employment
- Outcomes strikingly worse than pts with TBI coma
- Age and coma duration correlation with outcome

Denial vs. Lack of awareness

- Pattern: never told info, told & does not understand, does not remember
- Psychogenic denial: voluntary repression from consciousness of ideas/feelings that cause anxiety; perceived and then repressed
- In rehab, common in family members
- Neurogenic denial/lack of awareness: neurologically based lack of self awareness due to memory deficit, anosognosia, neglect
- Cannot deny what you do not know, understand, or remember

Neuropsychiatric Syndromes Associated with Neuroanatomical Lesions

- Lateral orbital pre-frontal cortex
  - Irritability - Impulsivity
  - Mood lability - Mania
- Anterior cingulate pre-frontal cortex
  - Apathy- Akinetic mutism
- Dorsolateral pre-frontal cortex
  - Poor memory search
  - Poor set-shifting / maintenance

Neuropsychiatric Syndromes 2

- Temporal Lobe
  - Memory impairment - Mood lability
  - Psychosis- Aggression
- Hypothalamus
  - Sexual behavior- Aggression

Neuropathology in TBI and Depression

- Left dorsolateral frontal lesions or left basal ganglia lesions are associated with MDD in acute TBI and stroke (Federoff et al., 1992, Robinson et al., 1985)
- Disruption of frontal lobe - basal ganglia circuits is associated with MDD in TBI (Mayberg, 1994)
- Decreased glucose metabolism in orbital-inferior frontal and anterior temporal cortex is associated with MDD in TBI, CVA, Parkinson's (Mayberg, 1994)

Neuropathology in TBI and Depression 2

- Serotonergic fibers have been implicated in the pathogenesis of arousal, sleep and depression in both the general population and brain-injured patients
- Frontal lobe damage from TBI is associated with reduced brain serotonergic function (VanWoerkom et al., 1977)
- MDD is associated with reduced left prefrontal gray matter volumes, esp. ventrolateral & dorsolateral regions (Jorge et al., 2004)

TBI as Emotionally Traumatic Event

- PTSD Prevalence: 13-27% *
  - Possibly more prevalent in mTBI
  - Mediated by implicit memory or conditioned fear response in amnestic patients?
- PTSD Phenomenology: **
  - Intrusive memories: 0-19%
  - Emotional reactivity: 96%
  - Intrusive memories, nightmares, emotional reactivity had highest predictive power
- Anxiety often comorbid with and prolongs depression

* Warden 1997, Bryant 1995, Flesher 2001
** Warden et al 1997, Bryant et al 2000

TBI as Chronic Illness (the “Invisible Epidemic”)

- 80,000-90,000 new TBI survivors experience onset of long-term disability annually
- About 1 in 4 adults with TBI is unable to return to work 1 year after injury
- 5.3 million Americans (2% of U.S. population) currently live with TBI-related disabilities
  - Based on hospitalized survivors only
- 65% of costs are accrued among TBI survivors
- Annual acute care and rehab costs of TBI = $9 - $10 billion*
- Annual direct and indirect costs of TBI survivors who required hospitalization = $56.3 billion **

* NIH Consensus Development Panel on Rehabilitation, 1999
** CDC, 1991

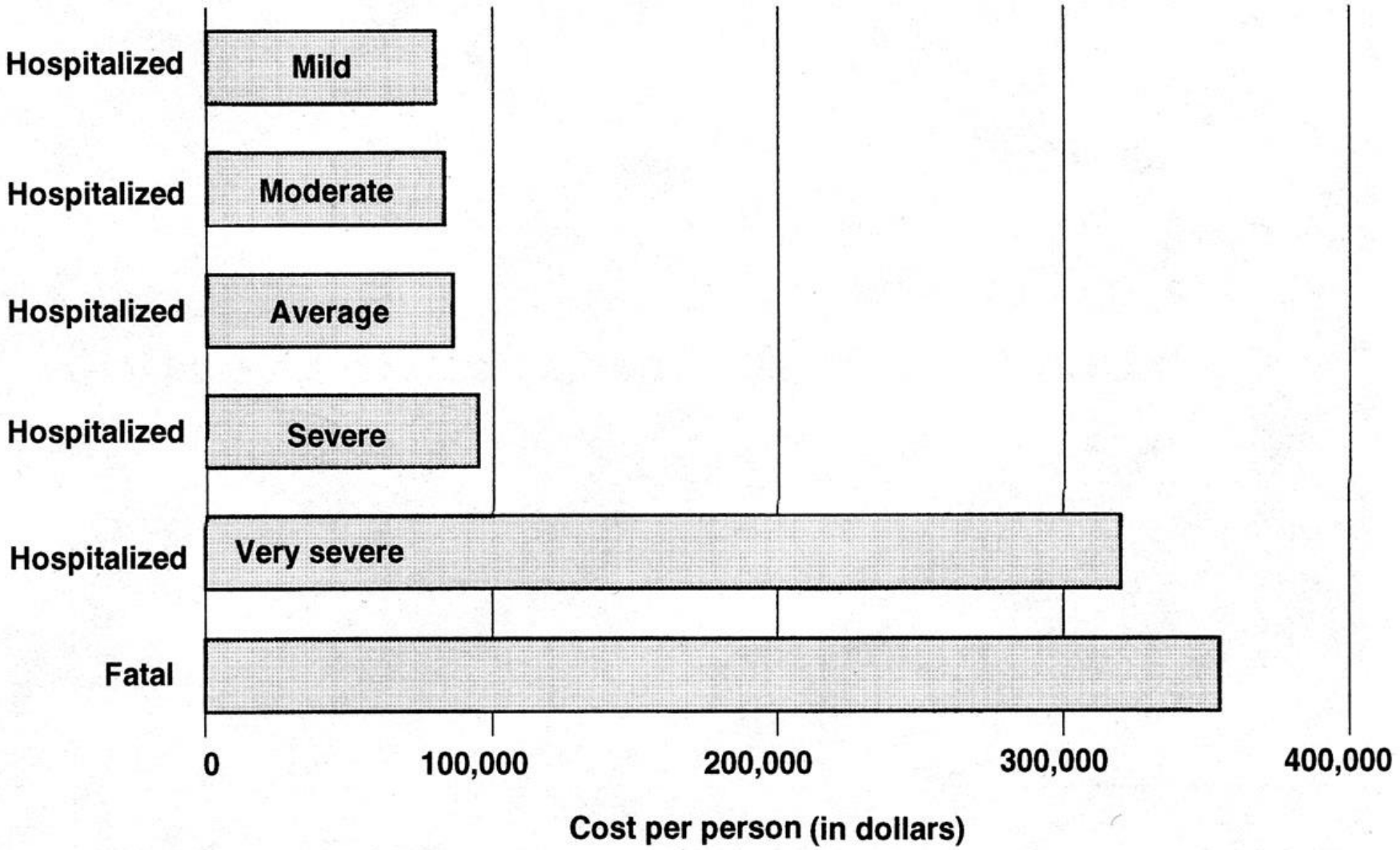


Figure 1–20. Lifetime costs of head injury, 1985 (by severity of injury).
Source. Max et al. 1991.

Neuropsychiatric Sequelae

- Delirium
- Depression / Apathy
- Mania
- Anxiety
- Psychosis
- Cognitive Impairment
- Aggression, Agitation, Impulsivity
- Postconcussive Symptoms

Depression / Apathy

- Prevalence of major depression: 44% *
  - Increased suicide risk
  - Assess pre-injury depression and alcohol use
  - Phenomenology may vary
  - May occur acutely or post-acutely
  - May be related to neuropsychological impairment and left frontal and basal ganglia lesions
  - Associated with increased functional impairment and post-concussive symptoms
- Apathy alone - prevalence 10%
  - Disinterest, disengagement, inertia, lack of motivation, lack of emotional responsivity

* van Reekum et al. *J Neuropsychiatry Clin Neurosci,* 2000;12:316-327

Depression Causes Increased Disability

- Depression is associated with worsening post-concussive symptoms (Fann et al., 1995)
- Depression is associated with more severe cognitive impairment and slower recovery after TBI and stroke (Levin et al., 1979; MacNiven et al., 1993; Robinson et al., 1986)
- Depression predicts lower health status and poorer functional outcome after TBI (Fann, et al., 1995; Christensen et al., 1994)
- There are no large controlled trials of pharmacotherapy or psychotherapy for depression in TBI populations

Anxiety

- Often comorbid with and prolongs course of depression
- Posttraumatic Stress Disorder: Prevalence 14.1% *
  - Re-experience, Avoidance, Hyperarousal
  - > 1 month, causes significant distress or impairment
  - Possibly more prevalent in mild TBI
- Panic Disorder: Prevalence 9.2% *
- Generalized Anxiety Disorder: Prevalence 9.1% *
- Obsessive-Compulsive Disorder: Prevalence 6.4% *

* van Reekum et al. J Neuropsychiatry Clin Neurosci 2000;12:316-327

Aggression, Irritability, Impulsivity

- Up to 70% within 1 year of TBI
- May last over 10-15 years
- Interview family and caregivers
- Characteristic features
  - Reactive - Explosive
  - Non-reflective - Periodic
  - Non-purposeful - Ego-dystonic
- Treat other underlying etiologies
- Also use behavioral interventions: Triggers

Manifestations of Impulsivity and Aggression

- Emotional lability
- Pathologic laughing and crying
- Rage and aggression
- Altered sexual behavior
- Bulimia
- Lack of concern over consequences of actions
- Social indifference
- Inappropriate joking and punning
- Superficiality of emotions

Treatment of TBI

- Education of family and patient
- Individual and group psychotherapy
- Psychopharmacology (mood stabilizers)
- Management of substance use
- Remember:
  - memory deficits are common (use visual aids, repetition, cure cards)
  - Impulsivity is common (slow down stimulus-response, discover triggers)

Rest Recommendation

- 1st randomized study:
- Recommending strict rest postinjury did not improve outcome and may have contributed to increased symptom reporting.
- Usual care (rest for 1–2 days with stepwise return to activity) is currently the best discharge strategy for pediatric mild traumatic brain injury/concussion.
- Patients in strict rest group reported more symptoms over the course of the study

Thomas et al., 2015

Free Helmets for Skaters

- The Ian Tilmann Foundation, Inc. has operated the "Helmet for a Promise" program Florida since 2006. In that time over 1600+ Skaters have made the promise and received a free skateboard helmet. The program is simple...if you promise to wear the helmet every time you skate...get a free helmet. The program operates in six Florida skate parks where helmets are picked up for free.
- Now The Helmet for a Promise program has gone national. Log on to website and download the Helmet Registration to make your promise. The FREE Helmet is direct shipped to you for a $7 fee. We offer both S-ONE and Bern helmets.
- GO TO..... www.theiantilmannfoundation.org
- Click the "Helmet for a Promise" icon..the rest is simple."

Books

- *Mild Traumatic Brain Injury and Postconcussion Syndrome* by Michael McCrea
- *Textbook of Traumatic Brain Injury* by Stuart C. Yudofsky
- *Traumatic Brain Injury: Methods for Clinical and Forensic Neuropsychiatric Assessment*, 2nd Ed. by Robert P. Granacher Jr.

Websites

- http://www.youtube.com/profile?user=TBINBD#p/u/5/x9Xso4qGdlI
- http://www.ninds.nih.gov/disorders/tbi/tbi.htm
- http://www.biausa.org/

The End