Head Injury: Classification Most Severe to Least Severe

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DISCLOSURE

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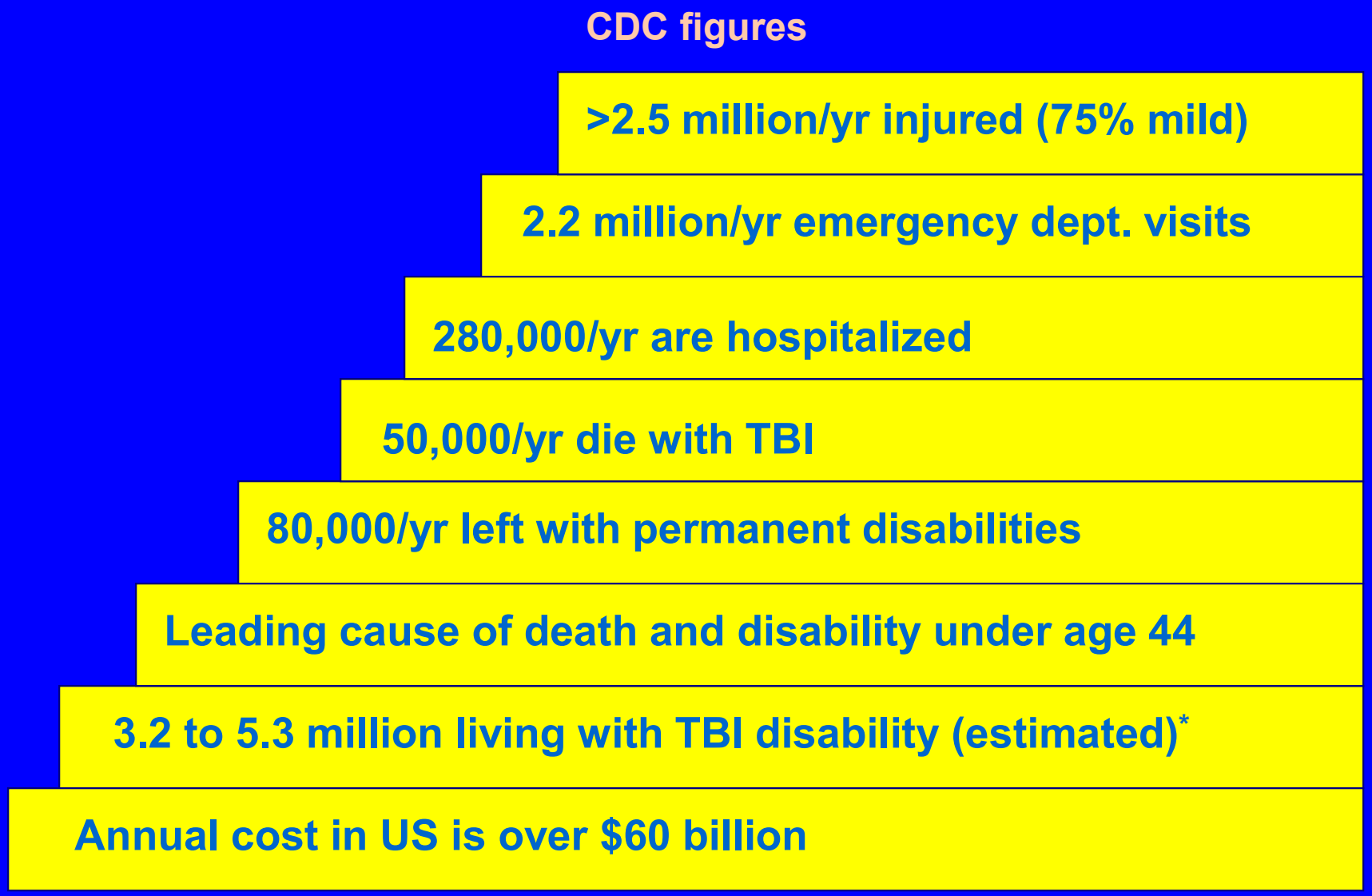
TBI Incidence & Prevalence in the USA



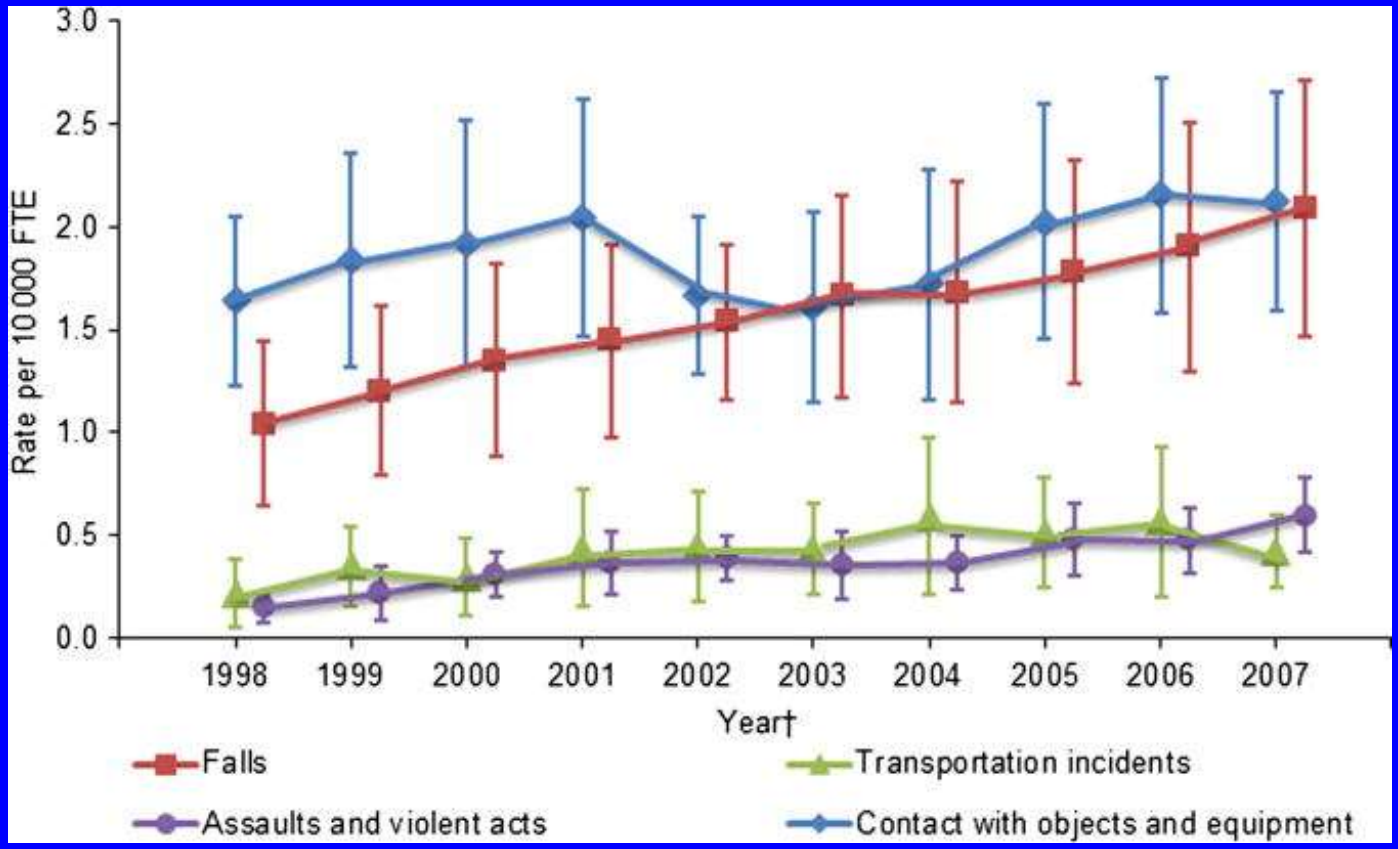
M Faul, et al.: CDC, 2010

*Zaloshnja E, et al.: J Head Trauma Rehab, 2008

Work-related TBI in the US

Konda et al., *Inj Prev*, 2015

Rates of non-fatal WRTBIs treated in emergency departments by year and event, US, 1998–2007



Trend of increasing rates 1998-2007.

Most common causes: Falls and Contact with objects/equipment.

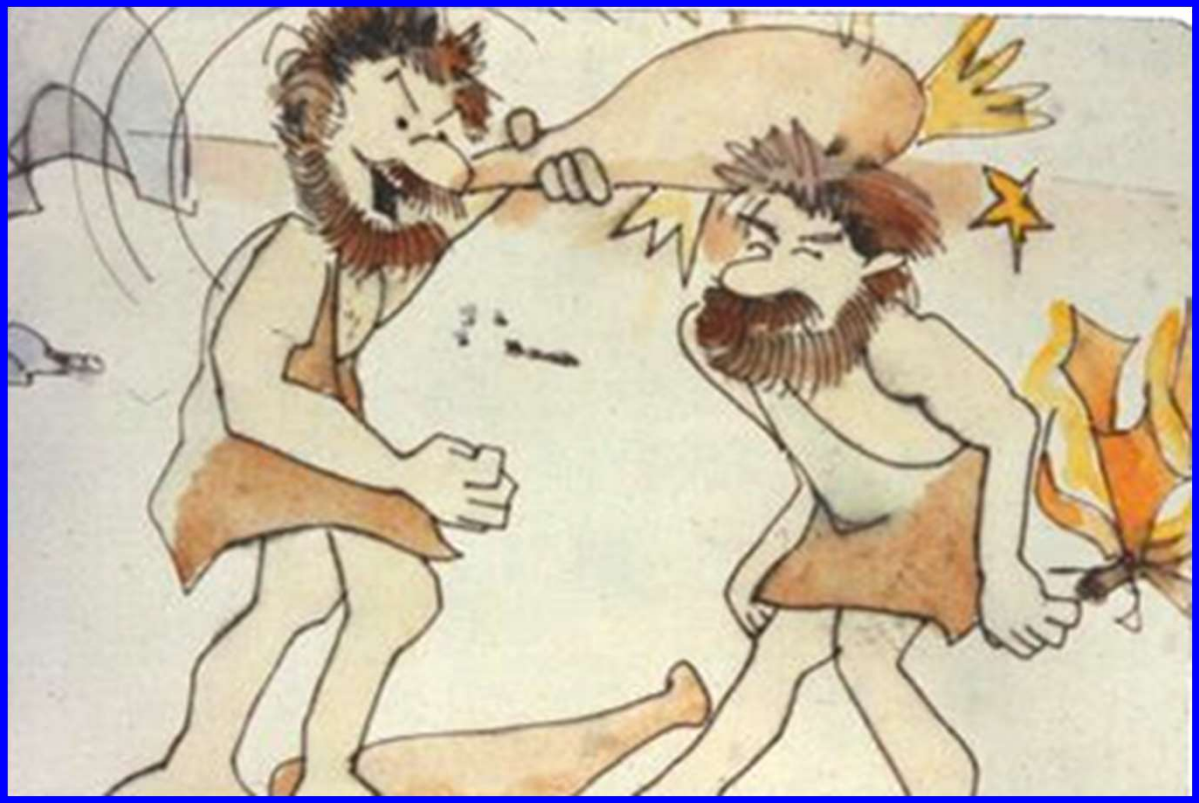
Work-related TBI in the US by industry

Highest rate of injury

Greatest number of injuries

Industry	Per cent	Rate per 10,000 FTE
Agriculture, forestry, fishing and hunting	5	16.8
Transportation and warehousing	8	8.9
Arts, entertainment and recreation	3	8.6
Construction	12	7.8
Accommodation and food services	8	7.2
Public administration	5	5.7
Retail trade	11	5.6
Healthcare and social assistance	11	4.9
Wholesale trade	3	4.3

Mechanisms of Injury: contact phenomena



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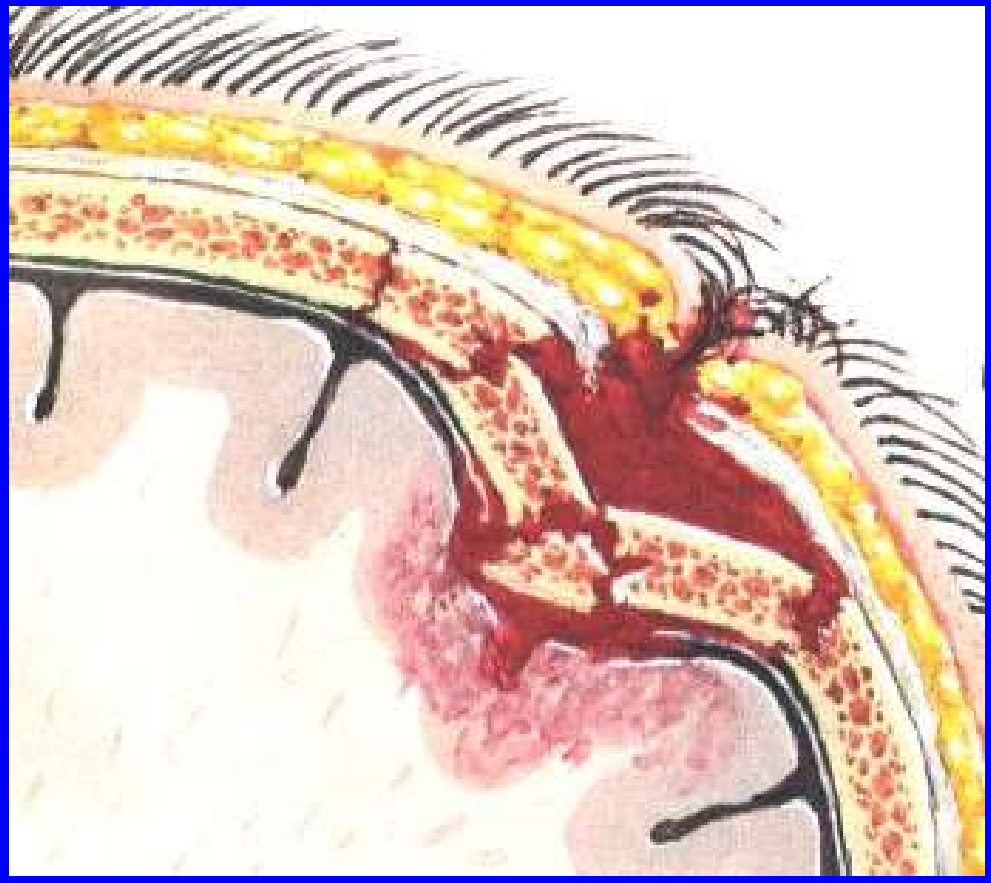

Mechanisms of Injury: Inertial Loading

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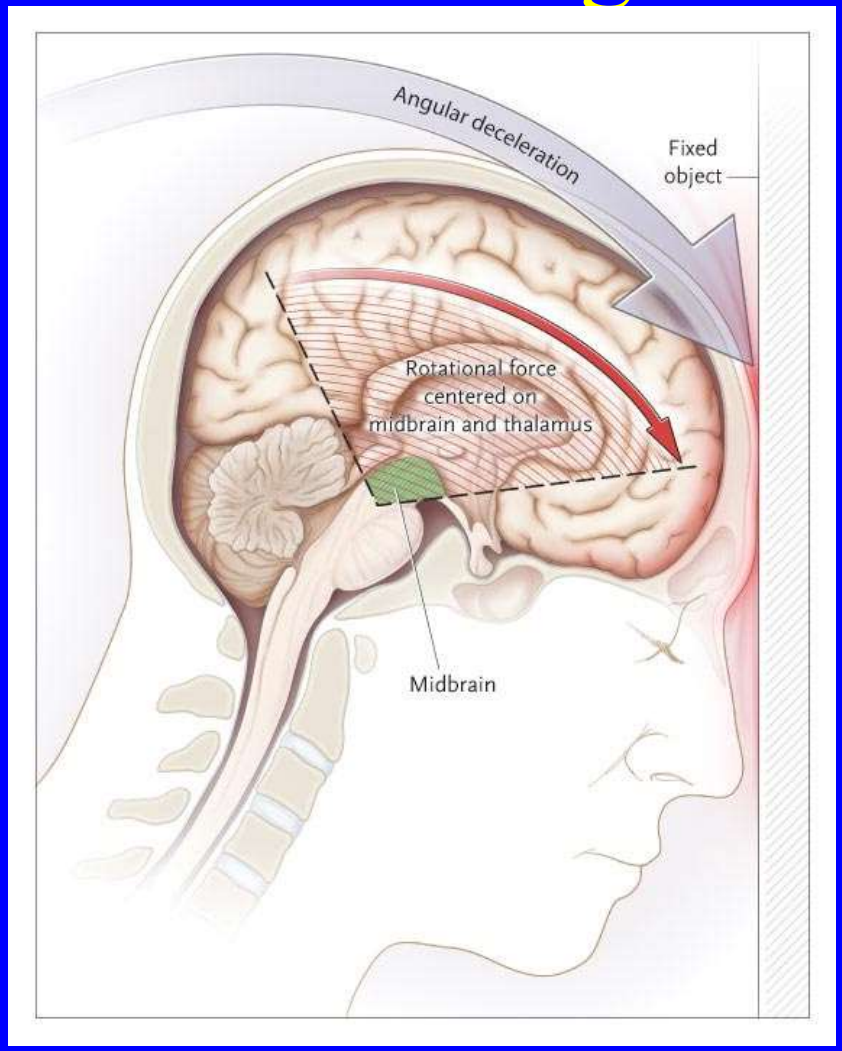
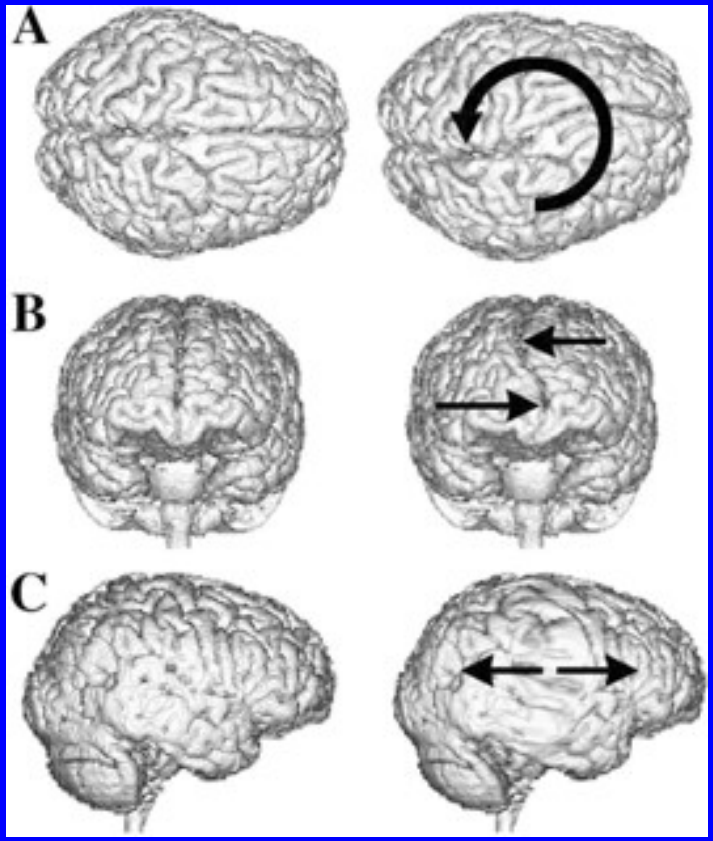


Mechanisms of Injury: Inertial Loading



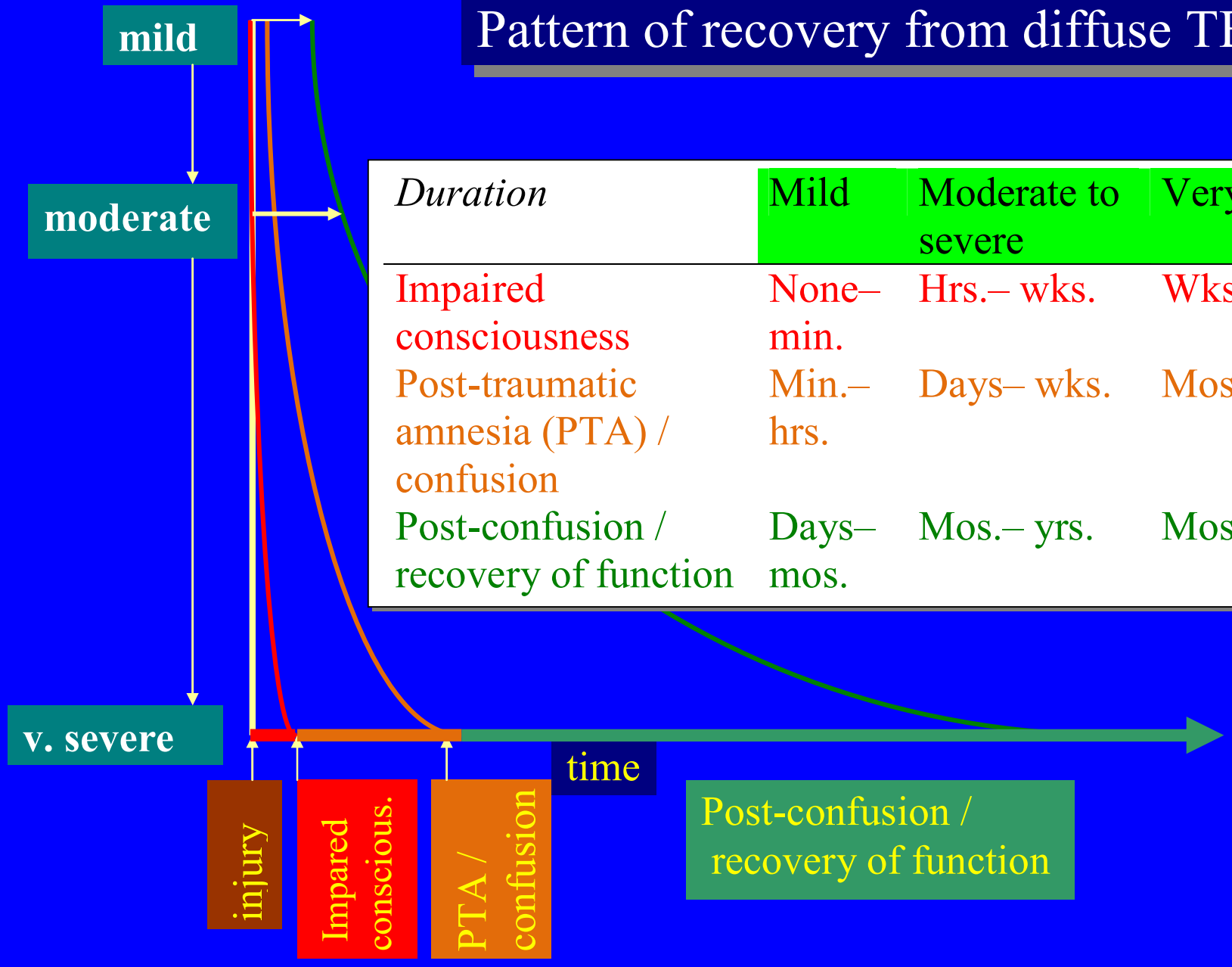
Neuropathological Consequences of TBI

	Diffuse	Focal
Primary	Diffuse axonal injury small white matter hemor.	Focal cortical contusion Deep cerebral hemorrhage (extracerebral hemorrhage)
Secondary	Hypoxic-ischemic injury Microvascular injury Swelling Excitotoxicity Delayed neuronal injury	Hypoxic-ischemic injury (stroke) Herniation damage Swelling Excitotoxicity & neuronal injury Late hemorrhages

Natural History of Recovery From Diffuse TBI

3 Main Phases of Recovery

Pattern of recovery from diffuse TBI



Duration	Mild	Moderate to severe	Very severe
Impaired consciousness	None– min.	Hrs.– wks.	Wks.- ?
Post-traumatic amnesia (PTA) / confusion	Min.– hrs.	Days– wks.	Mos. - ?
Post-confusion / recovery of function	Days– mos.	Mos.– yrs.	Mos.,yrs.- ?

Glasgow Coma Scale

Eye Opening Response	Verbal Response	Motor Response
4 = Spontaneous 3 = To verbal stimuli 2 = To pain 1 = None	5 = Oriented 4 = Confused 3 = Inappropriate words 2 = Incoherent 1 = None	6 = Obeys commands 5 = Localizes pain 4 = Withdraws from pain 3 = Flexion to pain or decorticate 2 = Extension to pain or decerebrate 1 = None

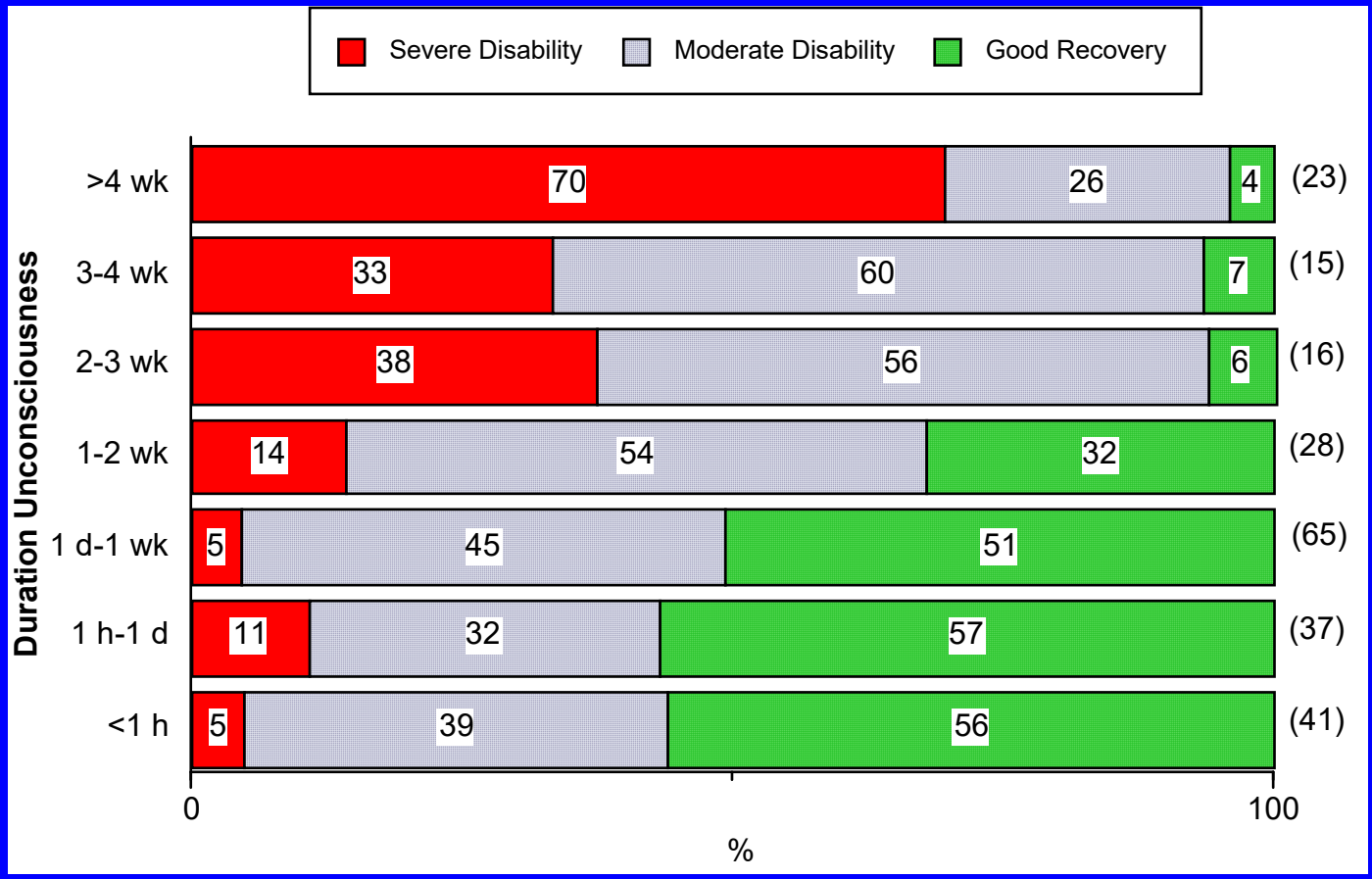
Total score = 3 to 15

TBI severity by GCS, LOC, PTA, and brain imaging

	GCS	LOC	PTA	Brain Imaging
Mild	13-15	0-30 min	<1 day	Usually normal
Moderate	9-12	>30 min to <24 hrs	>1 day to <7 days	Normal or abnormal
Severe	3-8	>24 hrs	>7 days	Normal or abnormal

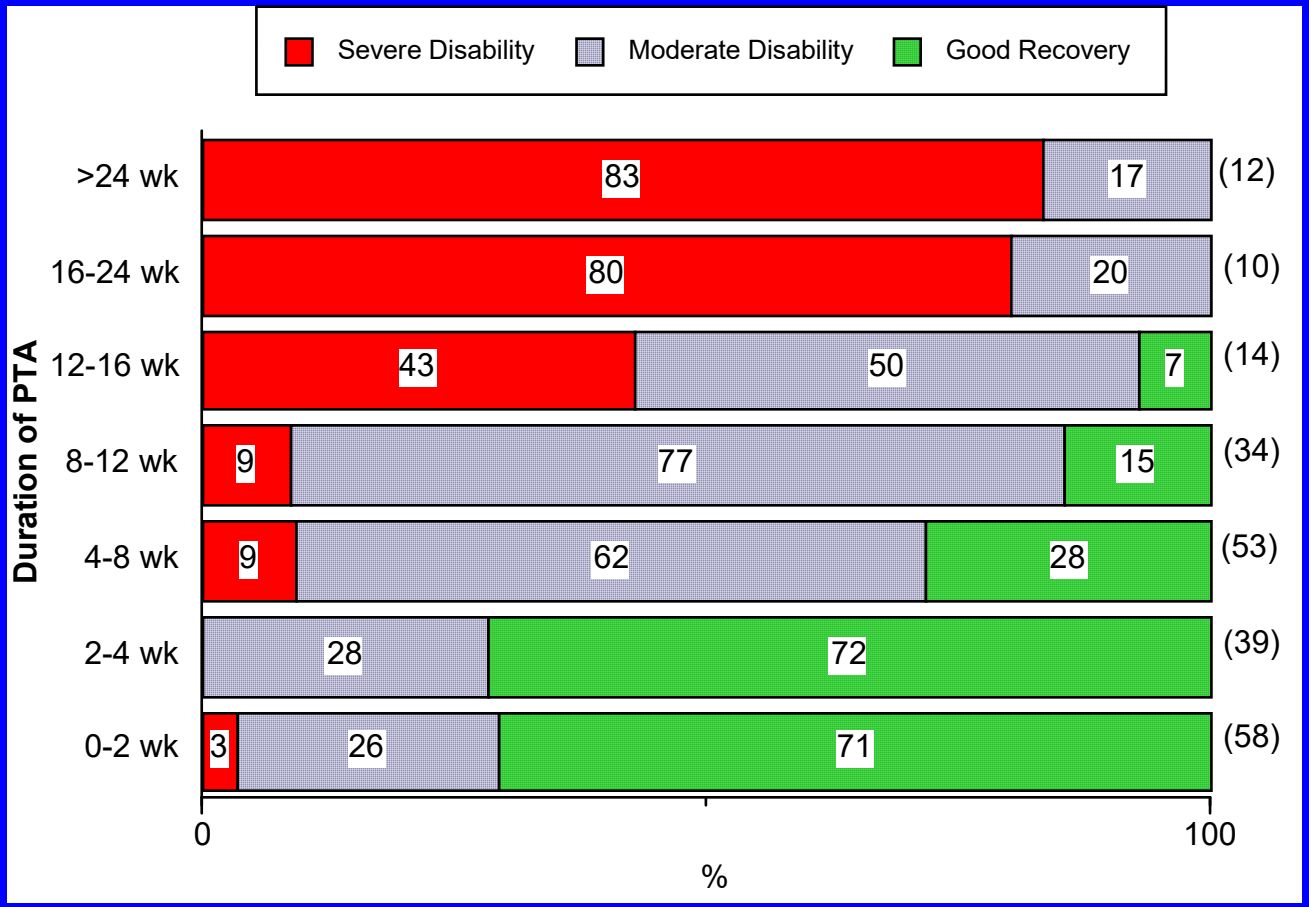
Functional Outcome:

1 year outcome related to duration of unconsciousness (n= 225 rehab. admissions w/ diffuse TBI)



Katz & Alexander, Arch Neurol, 1994

1 year outcome
related to duration of PTA
(n=220 rehab. admissions with diffuse TBI)

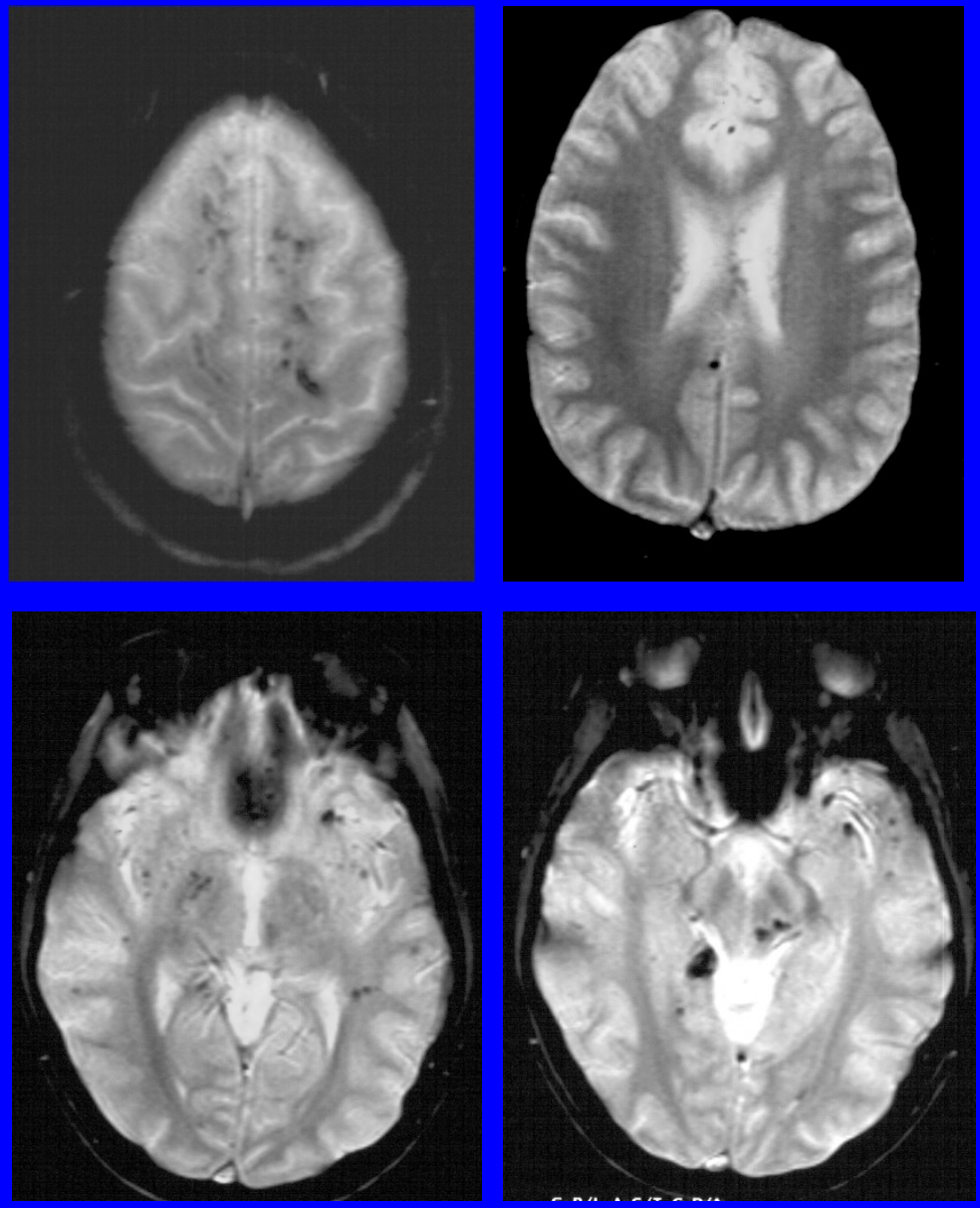


Diagnosis of DAI

- acceleration/deceleration mechanism
- immediate loss of consciousness (no lucid interval)
- supportive findings on CT/MR (petechial white matter hemorrhages, subarachnoid or intraventricular hemorrhage diffuse swelling)

17yo TBI in MVA

DAI:
Gradient echo or Susceptibility Weighted MRI demonstrates petechial hemorrhages



In parasagittal subcortical WM, temporal WM, corpus callosum, dlat. Midbrain, R int. capsule, et al.

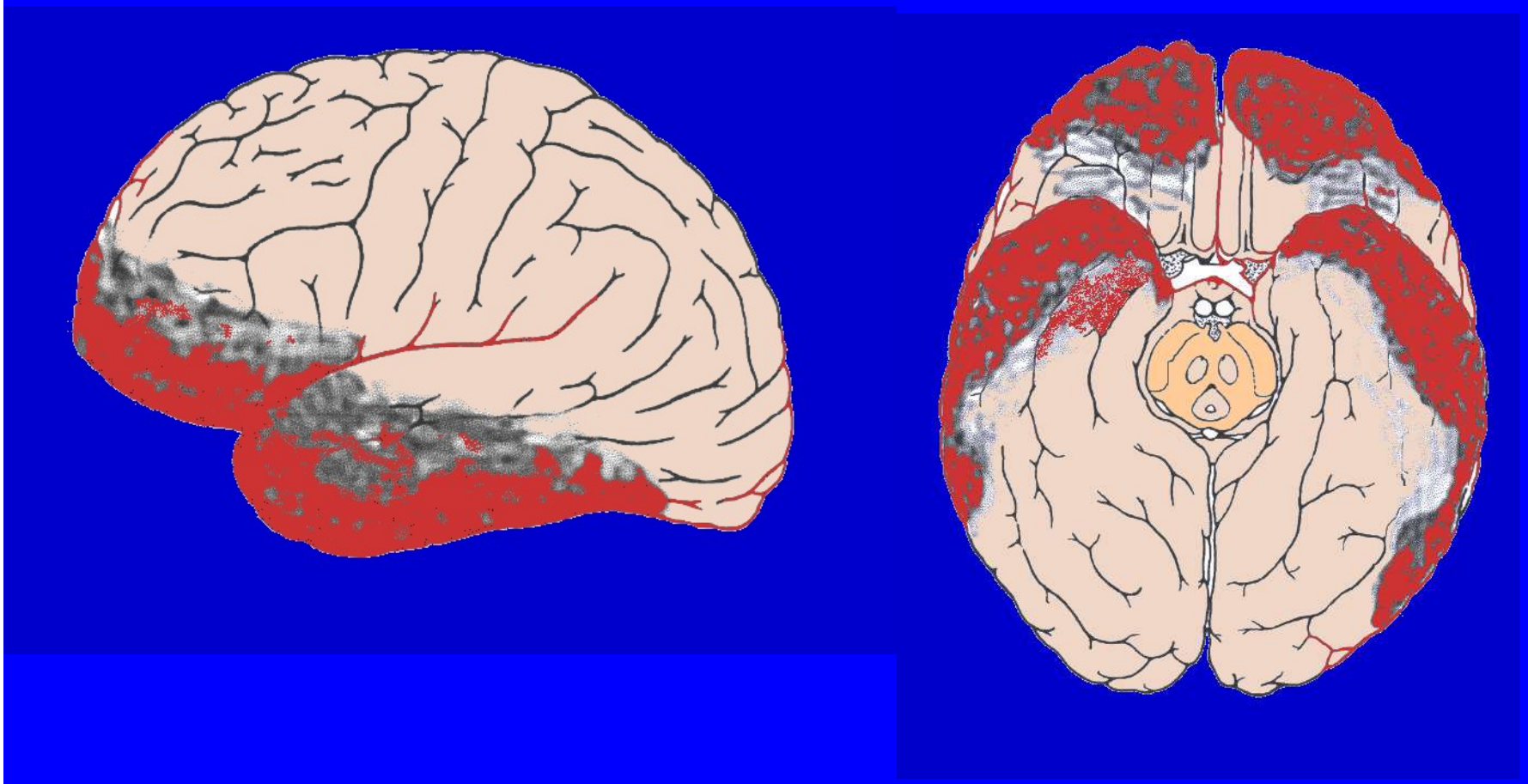
AF

Potential clinical affects of TBI

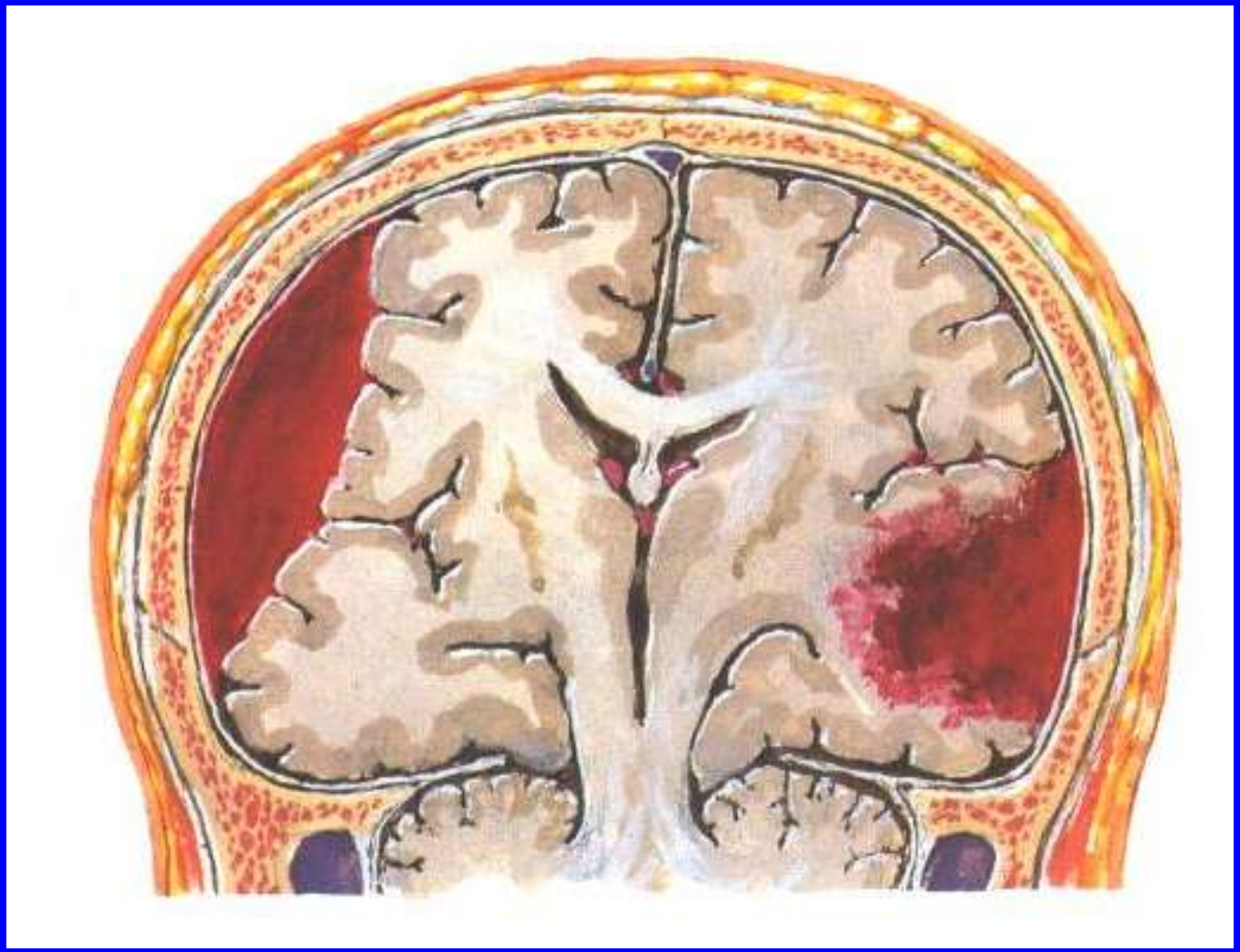
- Cognitive Function (e.g., attention, memory and executive functioning)
- Emotion (e.g., depression, anxiety, aggression, impulse control, personality changes)
- Motor function (e.g., extremity weakness, impaired coordination and balance)
- Sensation (e.g., hearing, vision, impaired perception and touch)

Focal Cortical Contusions

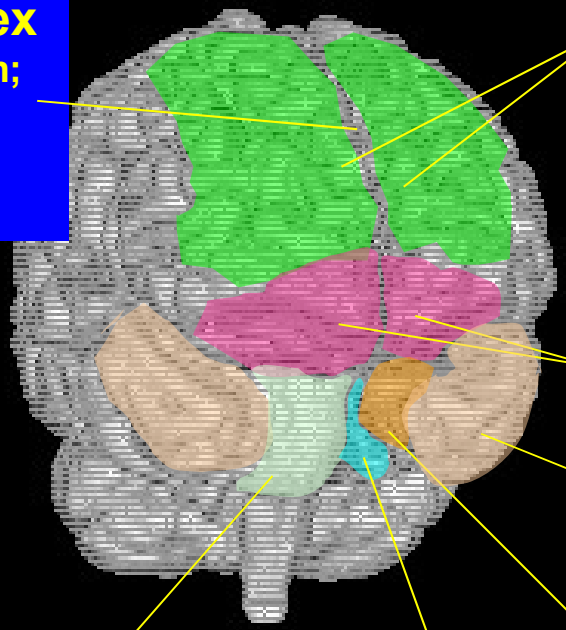
Predisposed Locations: Anterior-inferior Frontal and Temporal Lobes



Focal Injury: SDH and Focal Cortical Contusion



Regional Cortical Vulnerability to TBI Predicts Neurobehavioral Syndromes



Anterior medial prefrontal cortex (sustained attention; initiation and maintenance of behavior)

Dorsolateral prefrontal cortex (executive function, including sustained and complex attention, memory retrieval, abstraction, judgement, insight, problem solving)

Orbitofrontal cortex (emotional and social responding)

Anterior temporal cortex (memory, language, emotional responding, face recog. [temp.-occip.])

Amygdala (emotional learning and conditioning, including fear/anxiety)

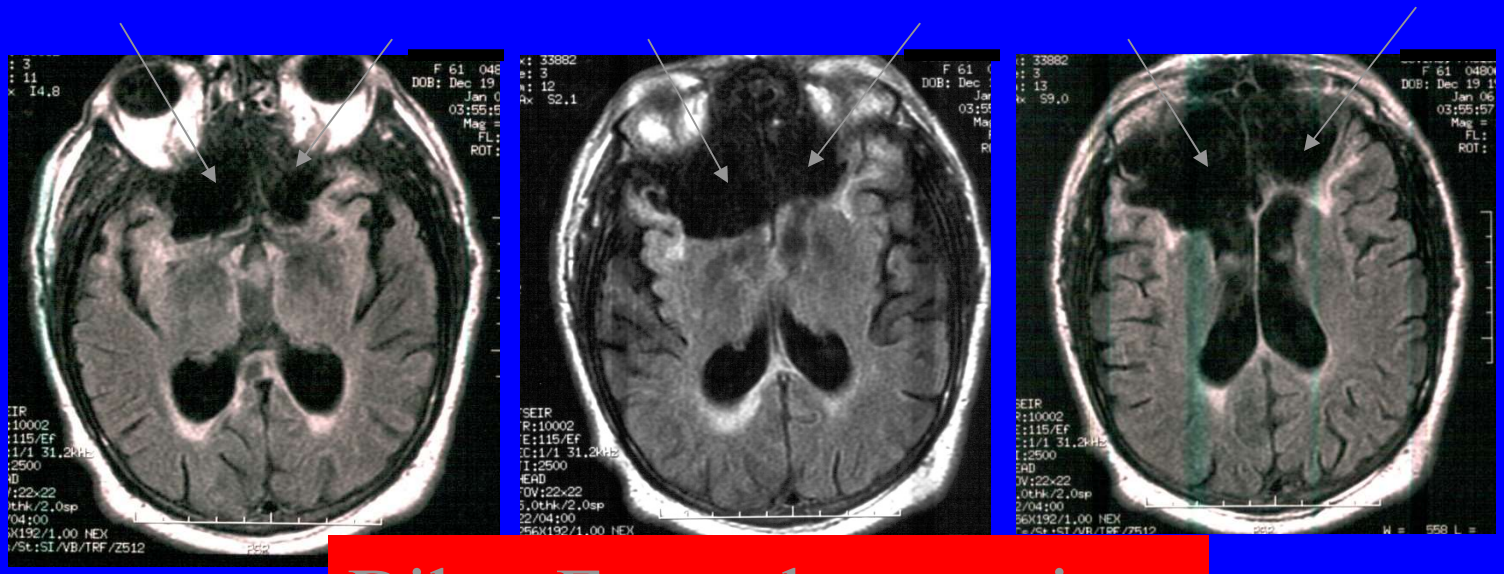
Hippocampus (only partially visible in this view - declarative memory)

Ventral brainstem (arousal, ascending activation of diencephalic, subcortical, and cortical structures)

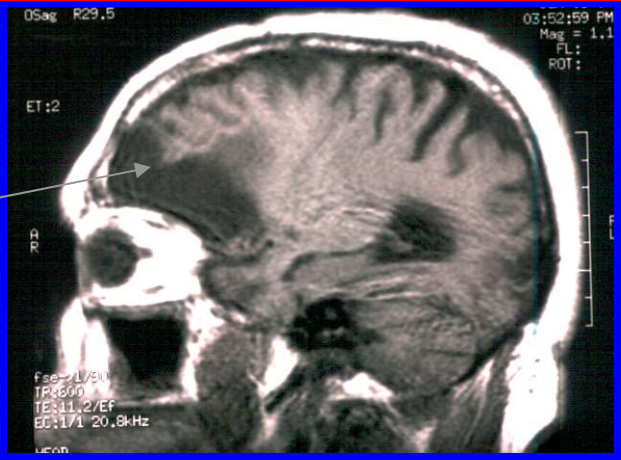
(Figure adapted from Arciniegas and Beresford 2001)

39yoM fell 4 feet off loading dock

brief LOC, then lethargic; PTA 4 wks

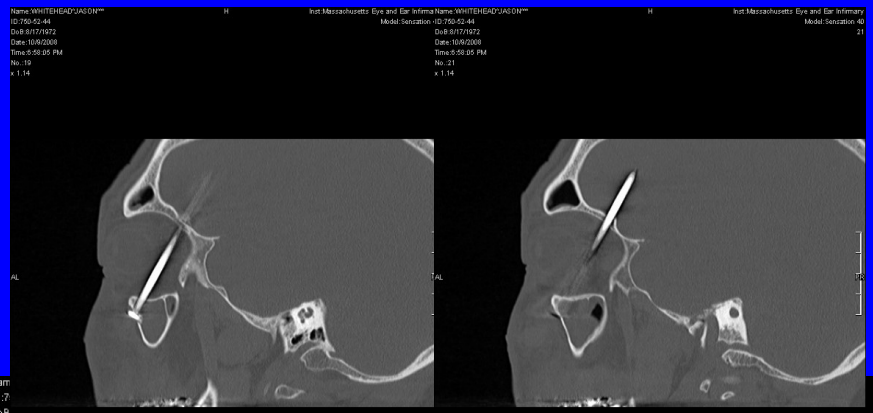


Bilat. Frontal contusions

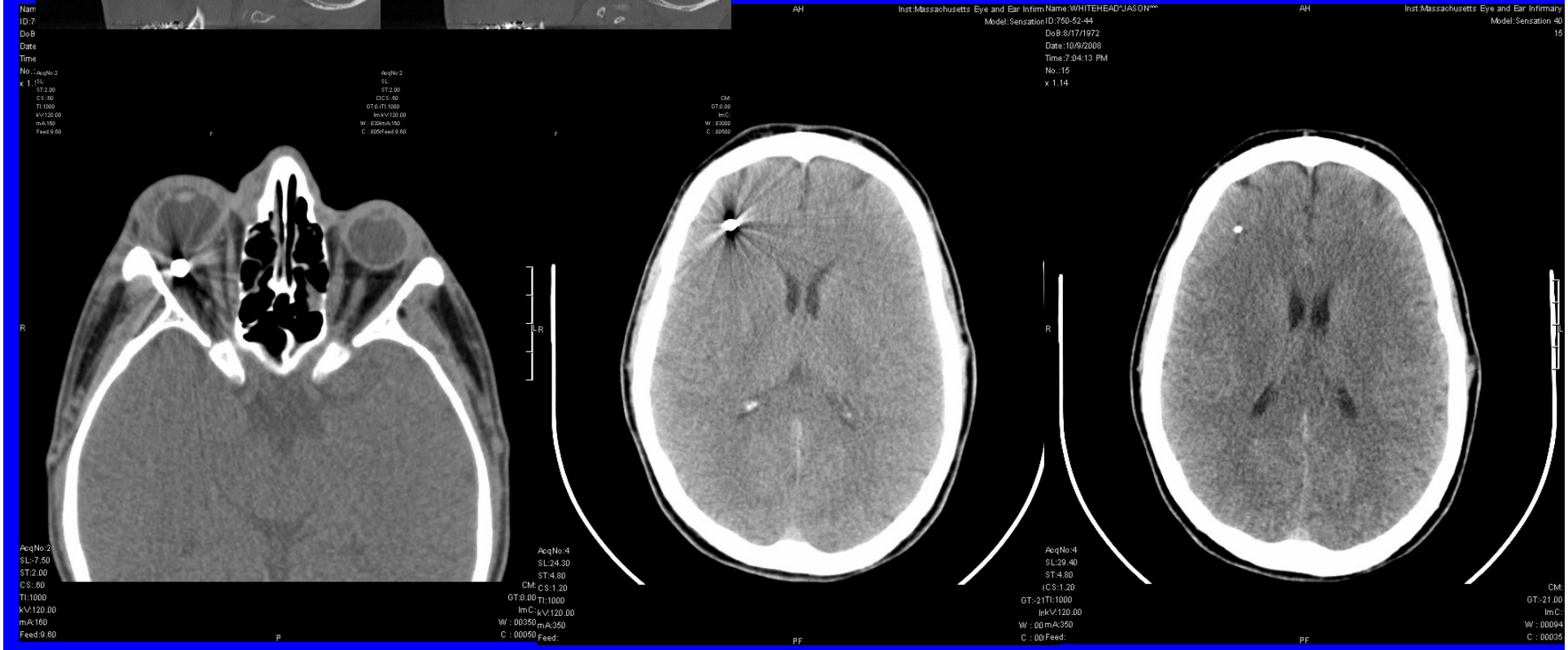


PC

36yoM PBI while working on roof



- No LOC, small maxillary entry wound
- 3 days later, pus from eye & nose
- 1 year later, personality change, less motivated, irritable, decr. working memory



Key points: TBI

- Two major mechanisms of injury are contact and inertial forces.
- Pathophysiology of TBI includes a combination of diffuse, focal and secondary injury processes.
- Diffuse axonal injury (DAI) is the major form of diffuse injury and involves delayed axonal disruptions and a host of associated secondary phenomena.

Key points: TBI (cont.)

- Recovery after diffuse injury involves a recognizable pattern of stages evolving from altered consciousness, to confusion/amnesia, to post-confusional recovery of attention, memory and executive cognitive functions.
- Duration of stages of recovery and severity of impairments at any stage relates to injury severity and other non-injury factors, including age. These variables serve as the main clinical outcome predictors.

Key points: TBI (cont.)

- Focal injuries involve focal cortical contusions, deep hemorrhages, subdural and epidural hematoma and focal ischemic lesions.
- Clinical syndromes relate to areas damaged by focal injury that interrupt distributed neurologic networks.
  - Frontal and temporal networks are most commonly damaged causing impairments in executive functions, attention, memory, behavioral & emotional regulation.
  - Syndromes are similar to those after diffuse injury which affect the same networks.

Key points: TBI (cont.)

- Late secondary complications (chronic subdurals, hydrocephalus, seizures) must be recognized since they may alter the course of recovery.

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