



Dr Rosamund Hill

Neurologist
Auckland

Head Injuries and Blunt Trauma - Main Session (Workshop options scheduled)

Saturday, 22 June 2013

Start 8:15am

Duration: 20mins

Baytrust



Rotorua GP CME 2013
NZMA
New Zealand Medical Association

General Practice Conference & Medical Exhibition

20-23 June 2013 | Energy Events Centre | Rotorua

Head Injury and Blunt Trauma

Rosamund Hill

Neurologist

Auckland Concussion Service

Level 1, 29 Enfield St, Mt Eden, Auckland

Ph 6387501

Fax 6387506

Traumatic Brain Injury

Rosamund Hill

Neurologist

Auckland Concussion Service

Level 1, 29 Enfield St, Mt Eden, Auckland

Ph 6387501

Fax 6387506





Mild Traumatic Brain Injury

- 95 % of injuries are Mild!!!

TBI incidence study (BIONIC) in NZ*



- 170,000 residents (100,000 urban)
- 1 March 2010- 28 February 2012
- 1369 cases
- Largely representative of NZ:
 - NZ Europeans – 63%
 - Maori – 20%
 - Pacific Island – 2%
 - Other – 15%

*Feigin et al. *Lancet Neurology* 2012

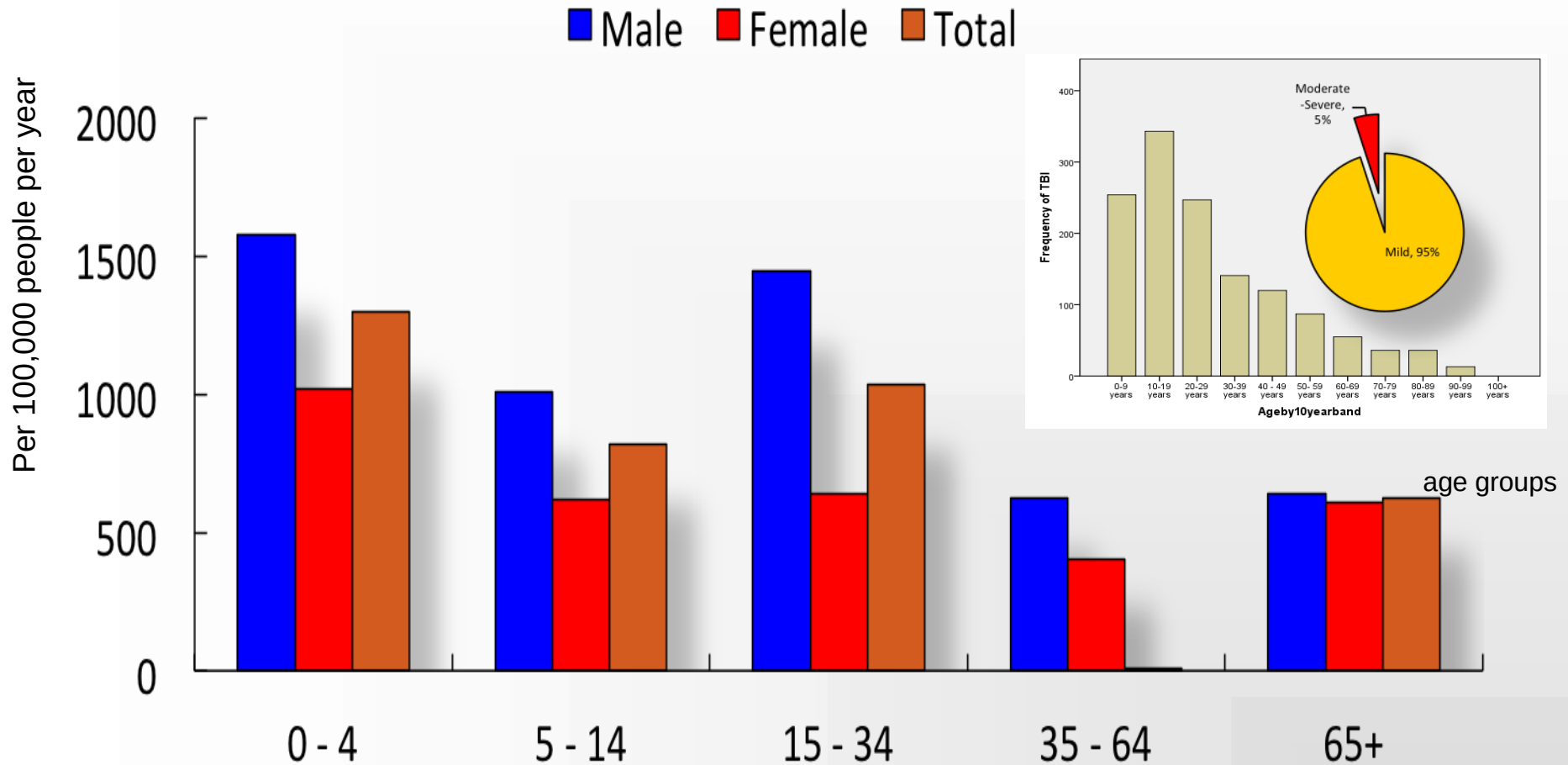
TBI incidence study (BIONIC) in NZ*



- TBI cases across all ages and TBI severity in both rural and urban population, hospitalised and non hospitalised
- follow-up all cases for major outcomes at 1, 6 and 12 months
- The largest and most comprehensive epidemiological study of TBI in the world
- HRC funded, 25 research staff involved

*Feigin et al. *Lancet Neurology* 2012

TBI incidence rates in NZ*



*Feigin et al. *Lancet Neurology* 2012

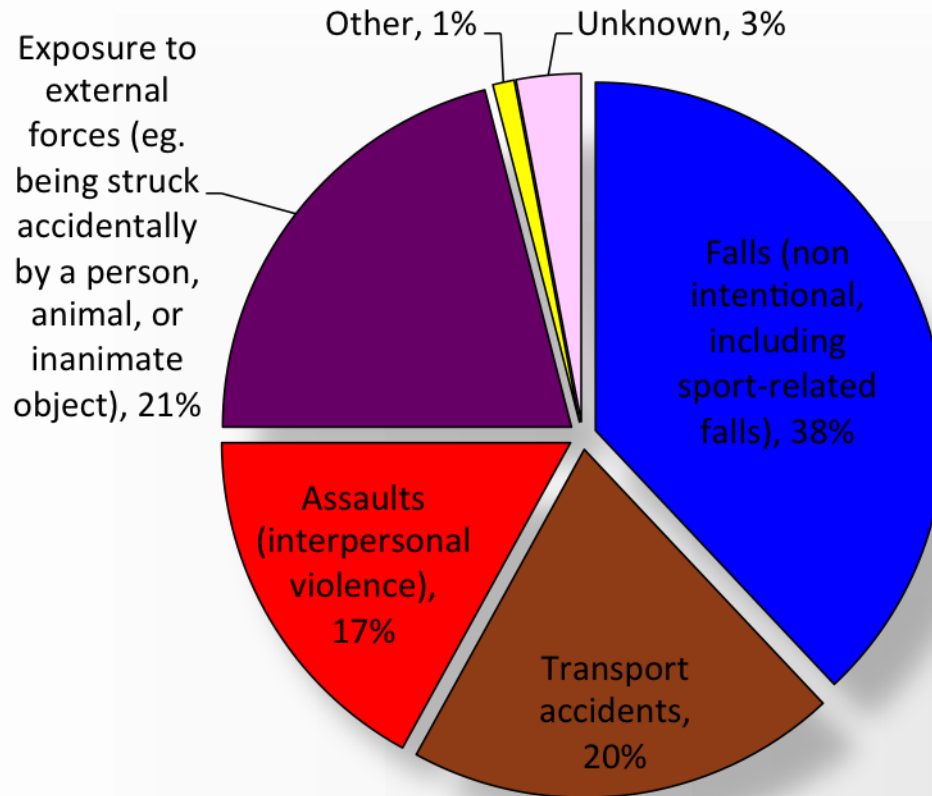
TBI incidence rates in NZ*

- TBI incidence in NZ (790/100,000) is far higher than in other developed countries (47-618/100,000).
- In NZ, there are 36,000 new TBI every year (far more than number of new strokes or heart attack. Likely estimated costs to the country – billions \$)
- The risk of sustaining moderate-severe TBI in the rural population is almost 2 times greater than in the urban population.

TBI incidence rates in NZ*

- Males - 62% of all TBIs
- Children and young adults (0-34 years) – 69% of all TBIs
- **Mild TBI – 95% of all TBIs** (significantly more than previously estimated).
- About 36% of people with mild TBI are never assessed at a hospital.

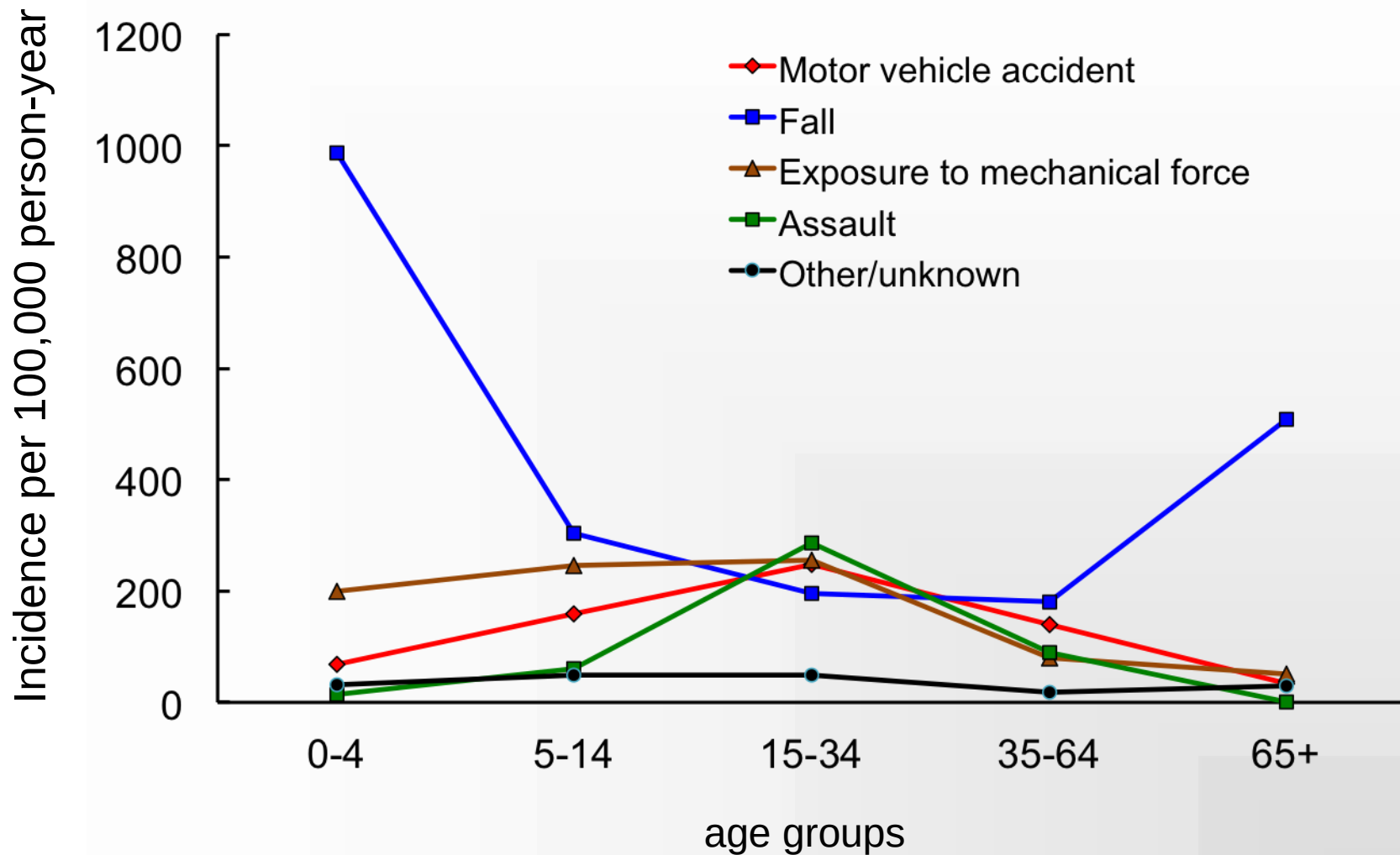
Mechanisms of TBI in NZ*



- Falls are the leading cause of TBI in NZ (not transport accidents as previously thought)
- The proportion of TBI due to assaults in NZ (especially in Maori) is far greater than in other populations

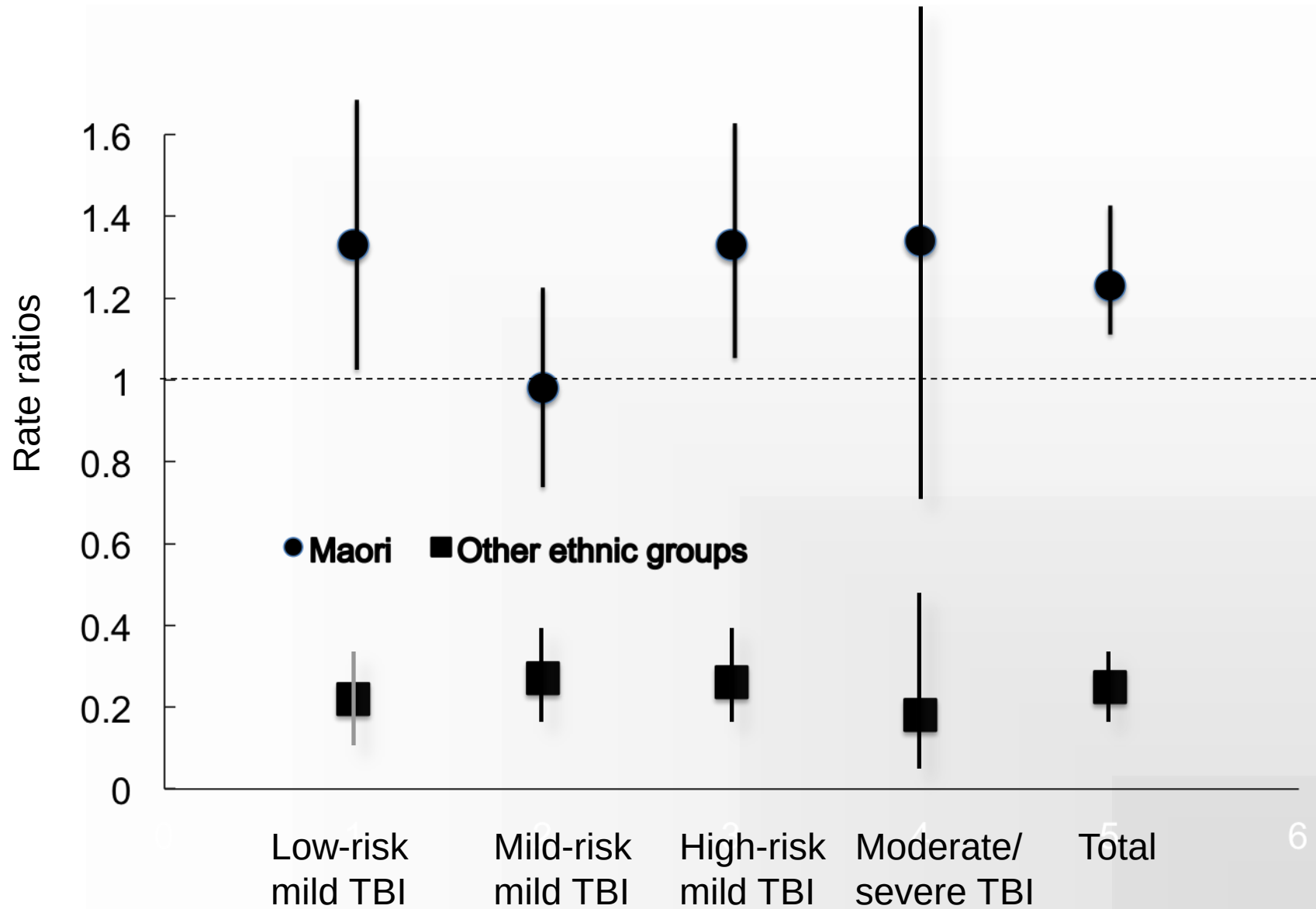
*Feigin et al. *Lancet Neurology* 2012

(A) Age-specific incidence of TBI by mechanism of injury



*Feigin et al. *Lancet Neurology* 2012

Rate ratios of TBI incidence by ethnicity (NZ Europeans as a reference group)



*Feigin et al. *Lancet Neurology* 2012

Direct force – primary injury



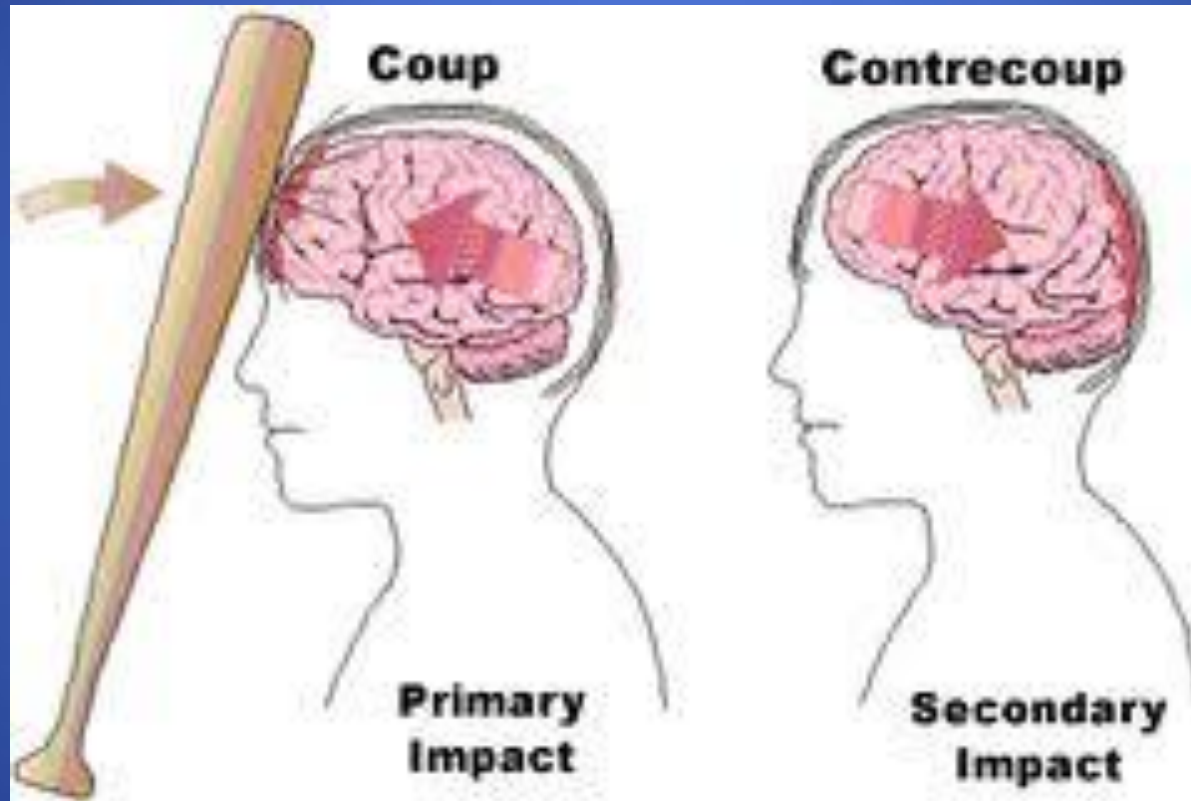
- Translation of kinetic energy and force vectors in either a **linear acceleration-deceleration** mechanism, through a **rotational** mechanism or a combination of both.
- Most injuries involve linear and angular forces.
- “brain slosh”

Direct force – primary injury



- Highest strain forces were imparted to the region corresponding to the deep midbrain level.
- Implanted telemetry devices and football helmet accelerometers provide valuable information.

Coup-contrecoup



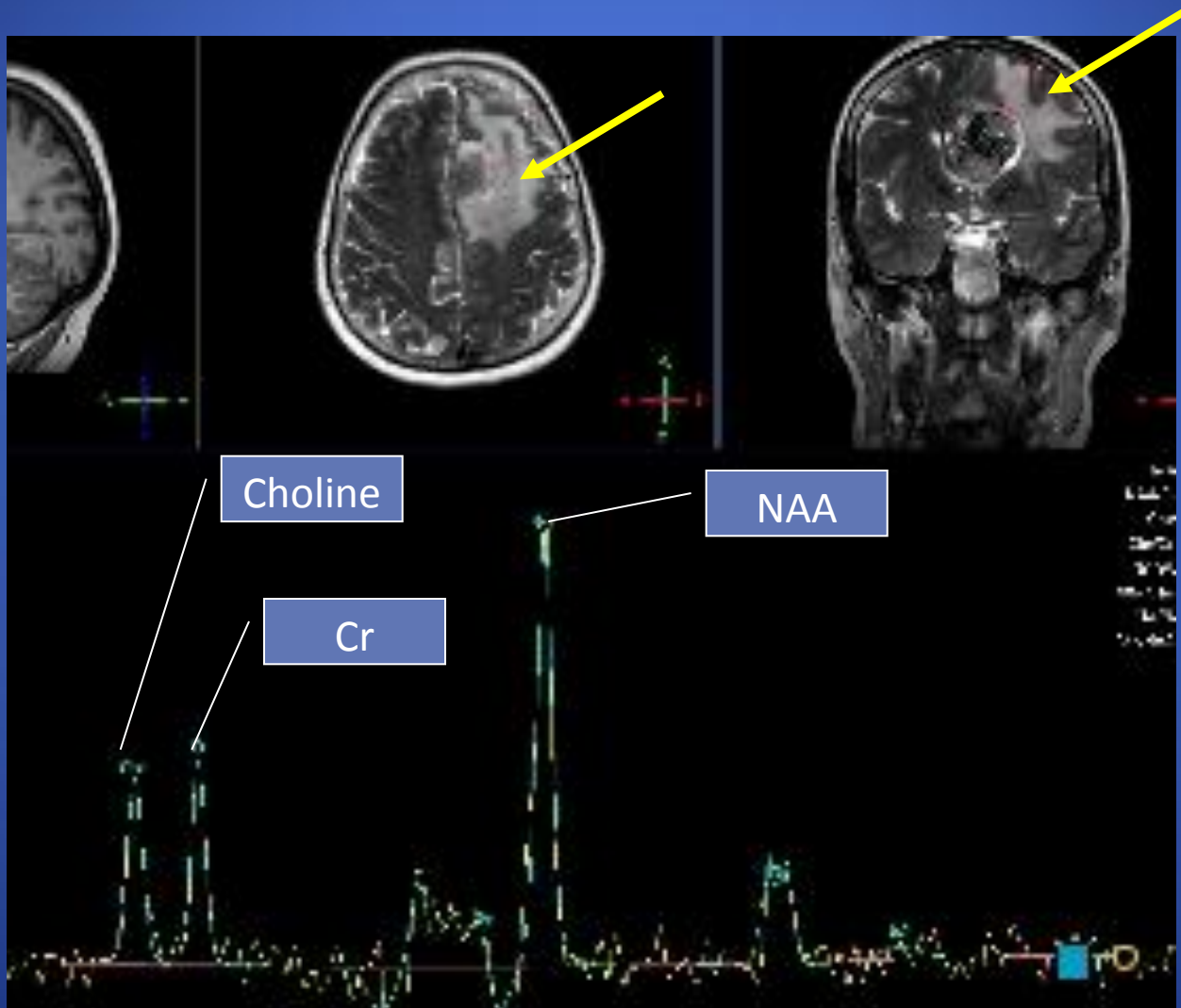
Whiplash



Types of Injuries

- Primary Injury:
 - Fracture
 - Extradural or subdural haematoma
 - Cerebral contusion
 - Diffuse axonal injury
 - Vascular Injury
- Secondary Injury:
 - Oedema, impaired perfusion, impaired autoregulation, impaired cellular homeostasis, delayed tissue injury and neuronal loss

MR Spectroscopy



MR Spectroscopy

In mild to moderate TBI patients there was significant widespread reduction in NAA and NAA/Cr (especially white matter) compared to controls and metabolite alterations correlated with dysfunction on neuropsychiatric testing.

Some studies report recovery of NAA levels at about 1 month post injury whereas others show persistently depressed NAA levels.

- Govind V, Gold S, Kaliannan K. Whole Brain Proton MR Spectroscopic Imaging of Mild to Moderate Traumatic Brain Injury and Correlation with Neuropsychological Deficits. J of Neurotrauma 2010; 27: 483-496
- Vagnozzi R, Signoretti S, Tavazzi B, et al. Temporal window of metabolic brain vulnerability to concussion: a pilot ¹H-magnetic resonance spectroscopic study in concussed athletes—part III. Neurosurgery 2008;62(6):1286–95.
- Henry LC, Tremblay S, Leclerc S et al. Metabolic changes in concussed American football players during the acute and chronic post-injury phases. BMC Neurology 2011; 11: 105-116

Routine MR Findings in DAI

- DAI findings are common in moderate to severe injuries (GCS <13) – 72% in one recent series without SWI

Skandsen T et al. Prevalence and impact of diffuse axonal injury in patients with moderate and severe head injury: a cohort study of early magnetic resonance imaging findings and 1 year outcome. J Neurosurg 2010; 113: 556-563

Susceptibility Weighted Imaging

- Detects approximately 4 times as many haemorrhagic foci as gradient echo T2 in trauma.

Akiyama Y, Miyata K, Harada K, et al. Susceptibility-weighted magnetic resonance imaging for the detection of cerebral microhemorrhage in patients with traumatic brain injury. *Neurol Med Chir (Tokyo)* 2009; 49:97–99

- Showed additional haemorrhagic lesions 30% of the time in paediatric head injuries compared to CT and standard MR sequences

Beauchamp MH et al. Detecting traumatic brain lesions in children: CT versus MRI versus susceptibility weighted imaging. *J Neurotrauma* 2011; 28(6): 915-27

Does the patient need a CT scan?

NICE Guidelines for CT scanning in Mild TBI

- GCS <13 at any point since the injury
- GCS equal to 13 or 14 at 2 hours after the injury
- Suspected open or depressed skull fracture
- Any sign of basal skull fracture (haemotympanum, “raccoon eyes”, CSF otorrhoea or rhinorrhoea, Battle’s sign)
- Post-traumatic seizure
- Focal neurological deficit
- More than one episode of vomiting
- Amnesia for greater than 30 minutes of events before impact

CT versus MRI



- Scanner availability, scan time, patient monitoring, ferromagnetic issues
- MRI wins for brain parenchyma

REMEMBER THE
GOOD OLD DAYS WHEN
WE PLAYED SPORTS
AND NEVER WORRIED
IF WE GOT CONCUSSIONS?



In the Bleachers © 2012 Steve Moore. Dist. by Universal Uclick



www.gocomics.com/inthebleachers

NO.



MoRE
2/21

Severity of Injury

- Glasgow Coma Score (GCS)

	Glasgow Coma Score	E+V+M
Eye Opening (E)	Verbal Response (V)	Motor Response (M)
4=Spontaneous 3=to voice 2=to pain 1=none	5=normal conversation 4=disoriented conversation 3=words but not coherent 2=no words, only sounds 1=none	6=normal 5=localises to pain 4=withdraws to pain 3=decorticate posture 2=decerebrate 1=none

Severity of Injury

- Mild
 - Any loss of consciousness up to 30 mins
 - Any alteration of mental state at the time of the accident (eg. Dazed, confused, disoriented)
 - Any loss of memory for events immediately before or after the accident for up to 24 hours
 - GCS 13 to 15
- Moderate
- Severe

Severity of Injury

- Mild TBI
 - GCS 13 to 15
 - Post traumatic amnesia < 24 hours
- Moderate TBI
 - GCS 9 to 12
 - Post traumatic amnesia 1 to 6 days
- Severe TBI
 - GCS < 8
 - Post traumatic amnesia 7 or more days

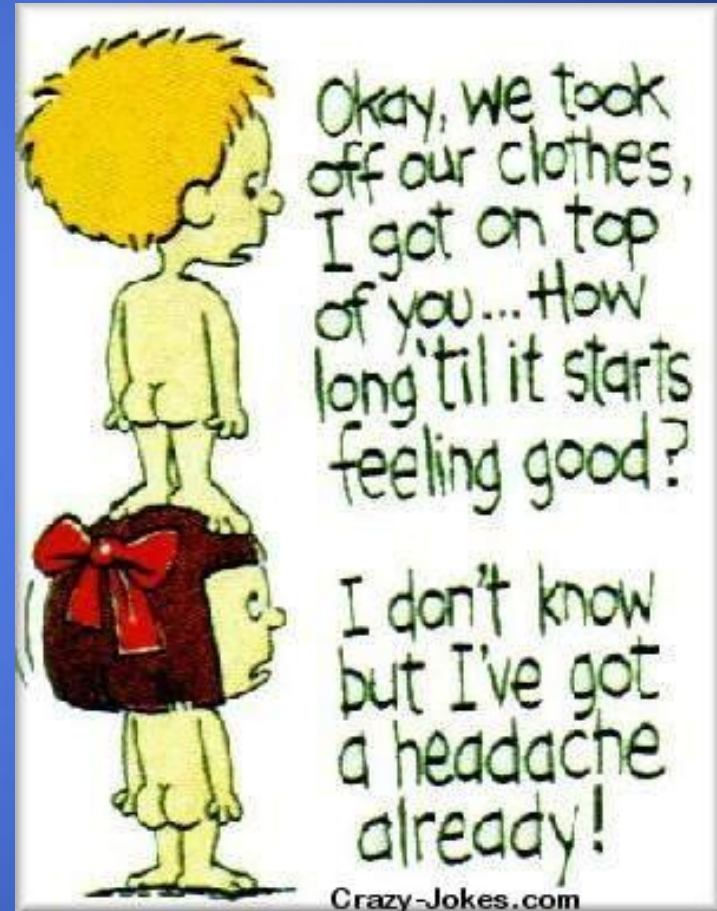
Symptoms – what does the patient complain of?



Common Symptoms of Brain Injury

Physical

Headache
Nausea and vomiting
Blurred or double vision
Balance problems
Dizziness
Light and noise sensitivity
Tinnitus



Common Symptoms of Brain Injury

Behavioural

Drowsiness

Fatigue

Irritability

Depression

Anxiety

Sleeping disturbance

Common Symptoms of Brain Injury

Cognitive

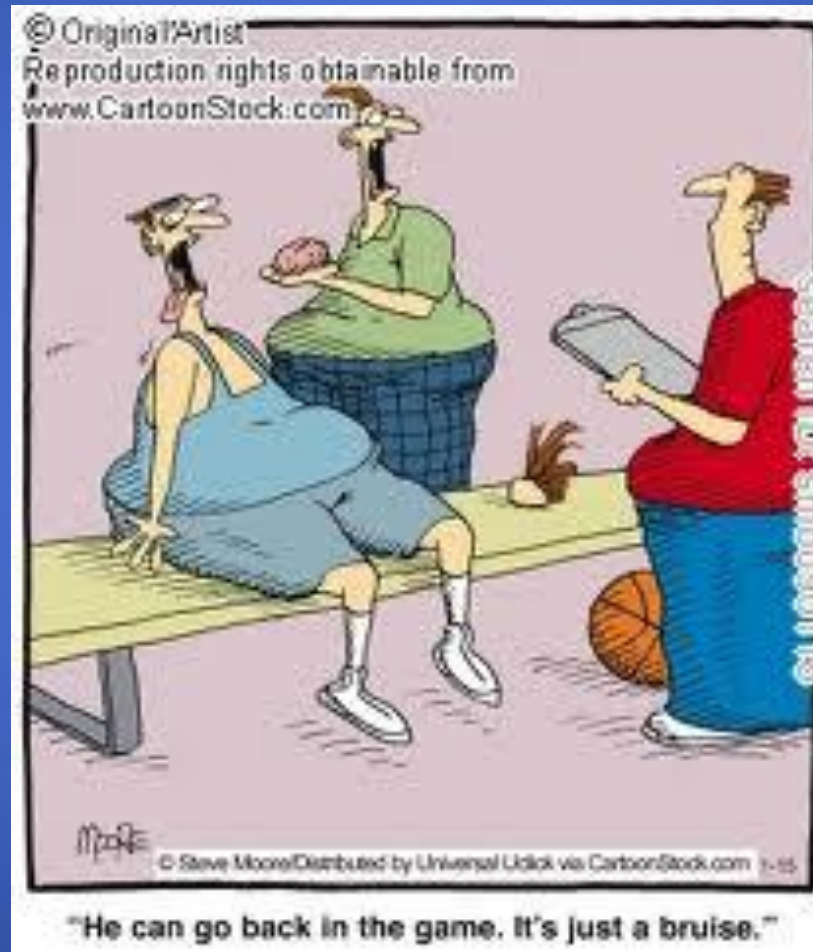
Feeling “slowed down”

Feeling “in a fog”

Difficulty concentrating

Difficulty remembering

Why do some patients with a mild injury do badly, recover slowly or incompletely?



Risk Factors for Persistent Symptoms Post Concussion Syndrome

preinjury

Age (older)

Gender (female)

Lower socio-economic state

Less education/lower levels of intelligence

Pre-existing neurological conditions (migraine)

Mental health disorders (depression, anxiety, traumatic stress or substance abuse)

Risk Factors for Persistent Symptoms

Peri-injury

Lack of support system

Acute symptom presentation (eg. cognitive impairment, headache, dizziness, nausea in the ER)

Context of the injury (stress, traumatic)

Risk Factors for Persistent Symptoms

postinjury

Compensation/litigation

Co-occurrence of psychiatric disorders

Co-occurrence of chronic pain conditions

Lack of support system

Treatment

- Education
- Rest
- Pacing activities
- Gentle exercise
- Simple analgesics
- Treat headache and sleep disturbance
- Education, support and monitoring
- Graduated return to work

Concussion Clinic

- Prompt triage by rehabilitation nurse or occupational therapist
- Selection of therapy options
 - OT – education, monitoring, supervise GRTW
 - PT – balance/dizziness
 - SLT
 - Medical assessment
 - Neuropsychological screening assessment
 - Clinical psychology input

Consequences of Repeated Injuries

- Cumulative effect of repeated injuries.
- Under-recognised effects of multiple mild or moderate injuries.
- Incidence and outcomes of traumatic brain injury and substance abuse in a New Zealand prison population.

Brain Injury 1998 Jun:12(6);455-466

- 86.4% of the 118 respondents had sustained a traumatic brain injury with 56.7% reporting more than one.
- Maori reported 12% more TBI.

Chronic Traumatic Encephalopathy (CTE)

- Seau was an outstanding linebacker for 20 NFL seasons before retiring in 2009.
- He died of a self-inflicted shotgun wound.
- Seau joins a list of several dozen football players who had CTE.
- Boston University's centre for study of the disease reported last month that 34 former pro players and nine who played only college football suffered from CTE.



Chronic Traumatic Encephalopathy (CTE)

- Chronic Traumatic Encephalopathy (CTE) is a progressive degenerative disease of the brain found in athletes (and others) with a history of repetitive brain trauma, including symptomatic concussions as well as asymptomatic subconcussive hits to the head.
- CTE has been known to affect boxers since the 1920s. However, recent reports have been published of neuropathologically confirmed CTE in retired professional football players and other athletes who have a history of repetitive brain trauma.

Chronic Traumatic Encephalopathy (CTE)

- The repetitive trauma triggers progressive degeneration of the brain tissue, including the build-up of an abnormal protein called tau.
- These changes in the brain can begin months, years, or even decades after the last brain trauma or end of active athletic involvement.
- Memory loss, confusion, impaired judgment, impulse control problems, aggression, depression, and, eventually, progressive dementia.

Common Symptoms of Brain Injury

Physical	Behavioural	Cognitive
Headache Nausea Vomiting Blurred or double vision Balance problems Dizziness Light and noise sensitivity tinnitus	Drowsiness Fatigue Irritability Depression Anxiety Sleeping disturbance	Feeling “slowed down” Feeling “in a fog” Difficulty concentrating Difficulty remembering

Outcome - New MR Techniques

- In a series of 43 ICU patients and 15 controls assessed prospectively with MR DTI and spectroscopy at mean of 24 days. Unfavourable outcome (death, PVS or minimally conscious state) was correctly predicted with 86% sensitivity and 97% specificity.

Tollard E, Galanaud D, Vincent Perlberg V, et al. Experience of diffusion tensor imaging and 1H spectroscopy for outcome prediction in severe traumatic brain injury: preliminary results. *Crit Care Med* 2009; 37:1448–1455

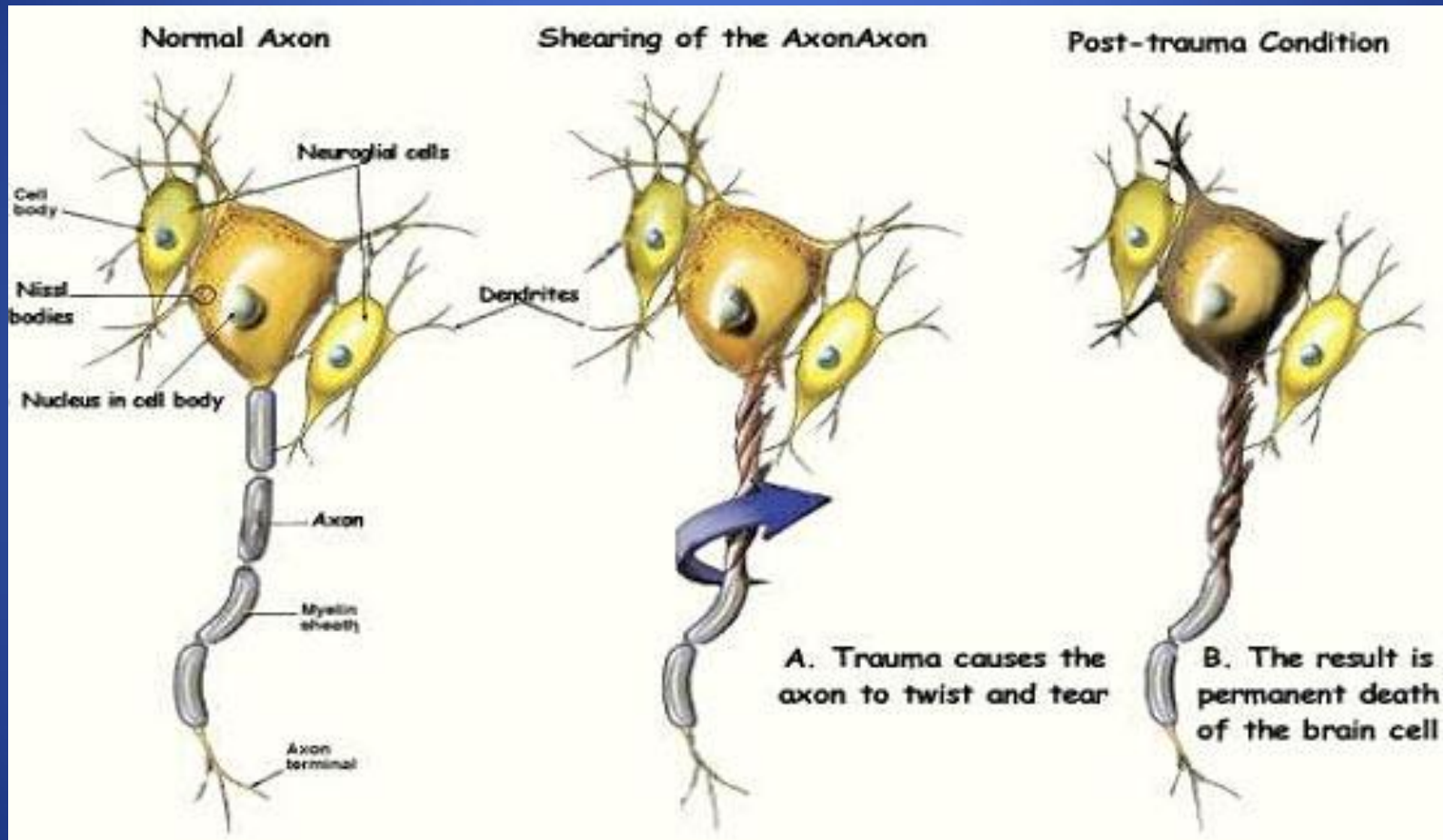
Secondary Injury – subsequent pathophysiological processes

- Forces cause shear stress to neurons causing axonal disruption, injury to neuronal membranes and ion flux and neurotransmitter effects.
- Excitatory amino acid release, potassium efflux, calcium influx.
- ATP pumps in overdrive to restore ionic homeostasis producing a hypermetabolic state.

Secondary Injury – subsequent pathophysiological processes

- Changes in cerebral blood flow.
- Changes in blood brain barrier
 - Damage to endothelium resulting in small vessel permeability and dysregulation
 - vasospasm
- Neuroinflammation and microglia activation

Diffuse Axonal Injury



Chronic Traumatic Encephalopathy

