TBI AND EPILEPSY

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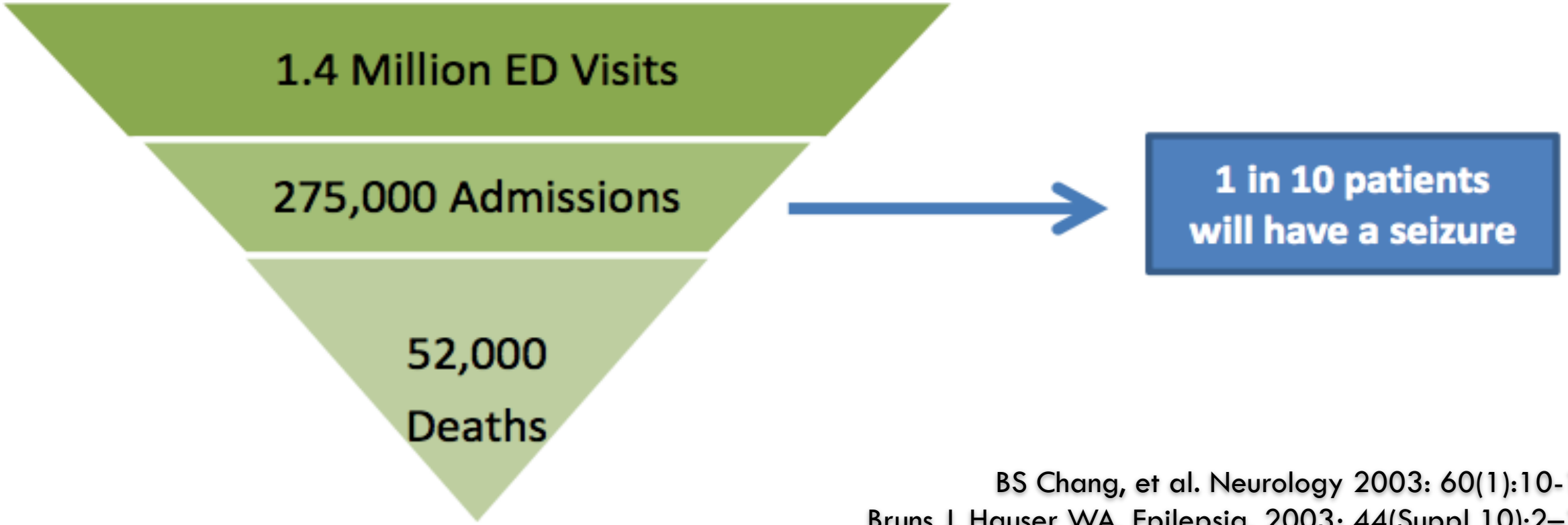
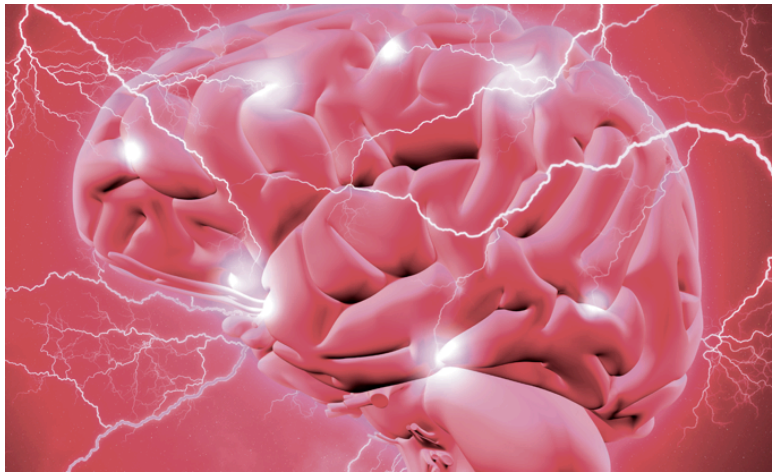
EPIDEMIOLOGY OF TBI

The incidence of TBI is between 180 and 250 per 100,000 per year



BS Chang, et al. Neurology 2003: 60(1):10-16.
Bruns J, Hauser WA. Epilepsia. 2003; 44(Suppl 10):2–10.
Rutland-Brown W, et al. J Head Trauma Rehabil. 2006; 21:544–548.

SEIZURES AFTER TBI



Secondary brain damage

- Increased ICP
- Increased metabolic brain demands post HI
- Excessive release of neurotransmitters

Results in further complicating the existing brain damage

THAILAND

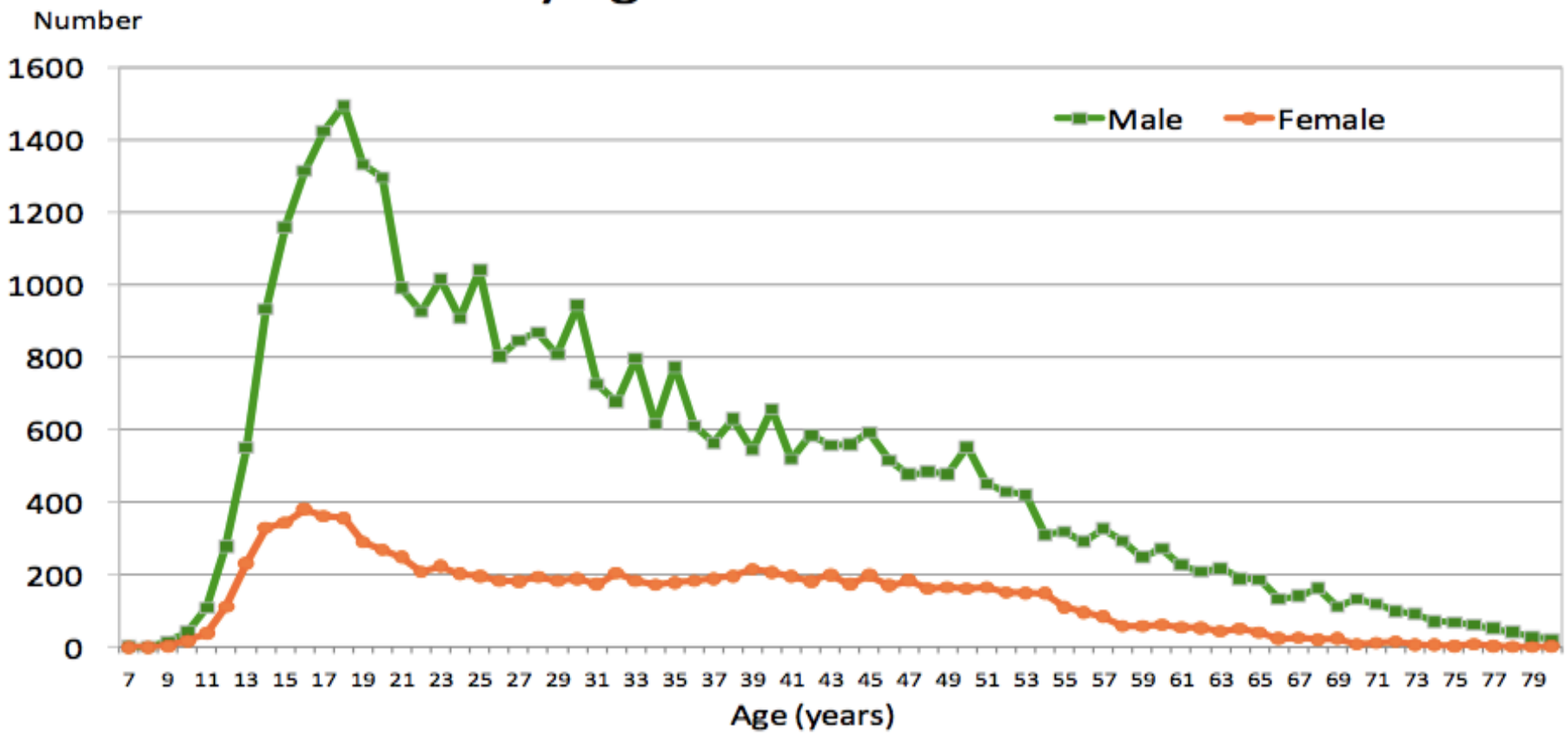
Number of severe injury in Thailand: 155,000 cases / year

Table 2 Number and percentage of severe injuries by 19 external causes (ICD 10 Chapter 20), Thailand 2005 - 2010

CAUSES OF INJURIES	YEAR												Total	
	2005		2006		2007		2008		2009		2010			
	Number	%	Number	%	Number	%	Number	%	Number	%	Number	%	Number	%
Transport Accidents	76,482	48.98	77,910	47.90	75,094	47.36	69,099	46.20	73,775	46.49	72,195	46.90	444,555	47.31
Accidental falls	24,746	15.85	25,977	15.97	25,528	16.10	25,437	17.01	27,225	17.16	26,259	17.05	155,172	16.51
Exposure to inanimate mechanical forces	20,562	13.17	22,492	13.83	22,796	14.38	22,562	15.08	24,822	15.64	24,345	15.81	137,579	14.64
Assaults	15,533	9.95	16,261	10.00	15,424	9.73	13,704	9.16	14,668	9.24	14,002	9.10	89,592	9.54
Accidental exposure to other and unspecified factors	246	0.16	279	0.17	224	0.14	155	0.10	163	0.10	156	0.10	1,223	0.13
Event of undetermined intent	193	0.12	170	0.10	175	0.11	194	0.13	251	0.16	207	0.13	1,190	0.13
Legal intervention and operation of wars	90	0.06	81	0.05	255	0.16	183	0.12	41	0.03	21	0.01	671	0.07
Unknown causes and intent	835	0.53	904	0.56	919	0.58	1,053	0.70	726	0.46	486	0.31	4,923	0.52
Total	156,157	100.00	162,660	100.00	158,571	100.00	149,573	100.00	158,685	100.00	153,939	100	939,585	100.00

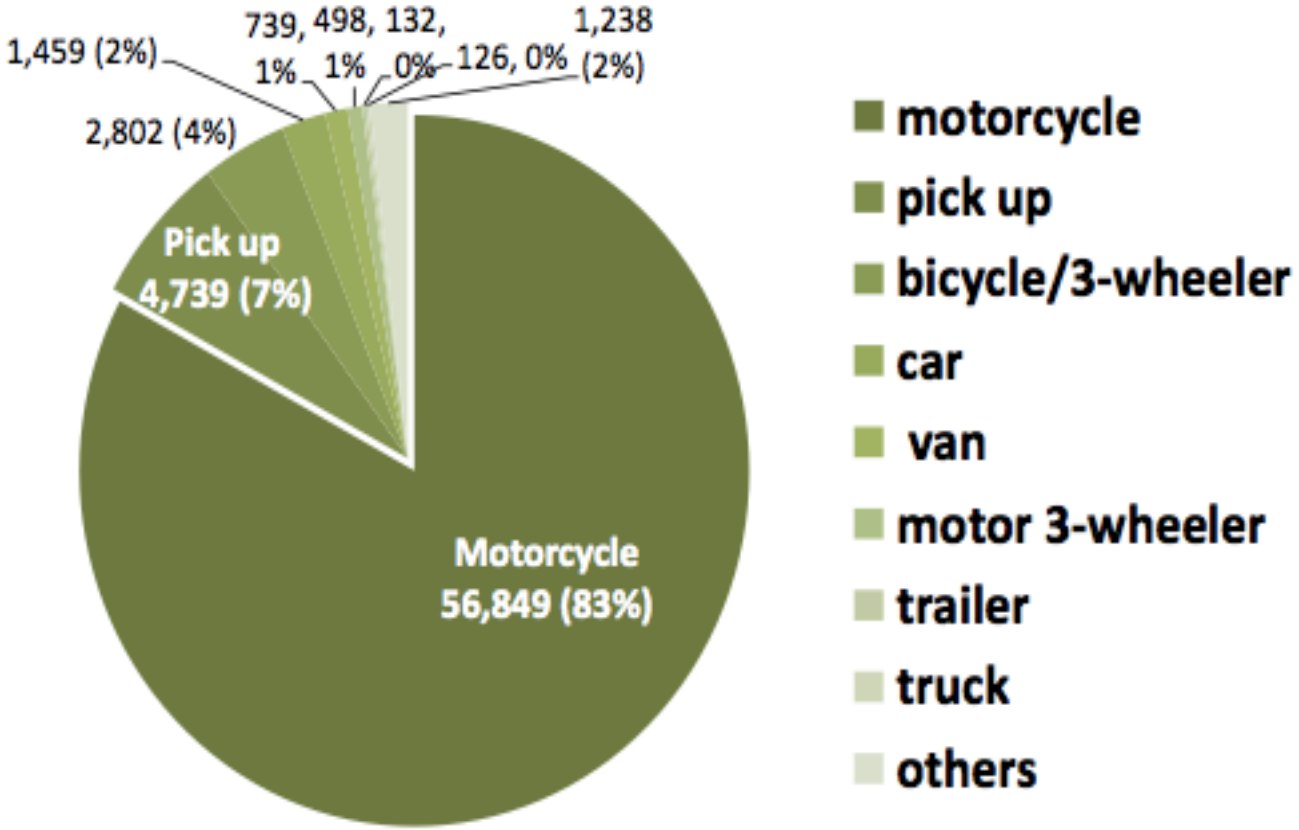
Source: 28 Sentinel hospitals, National Injury Surveillance (IS) System, Bureau of Epidemiology, Department of Disease Control, Ministry of Public Health, Thailand 2005-2010

by age and sex 2010



Source: 28 Sentinel hospitals, National Injury Surveillance System, Bureau of Epidemiology, Ministry of Public Health, Thailand

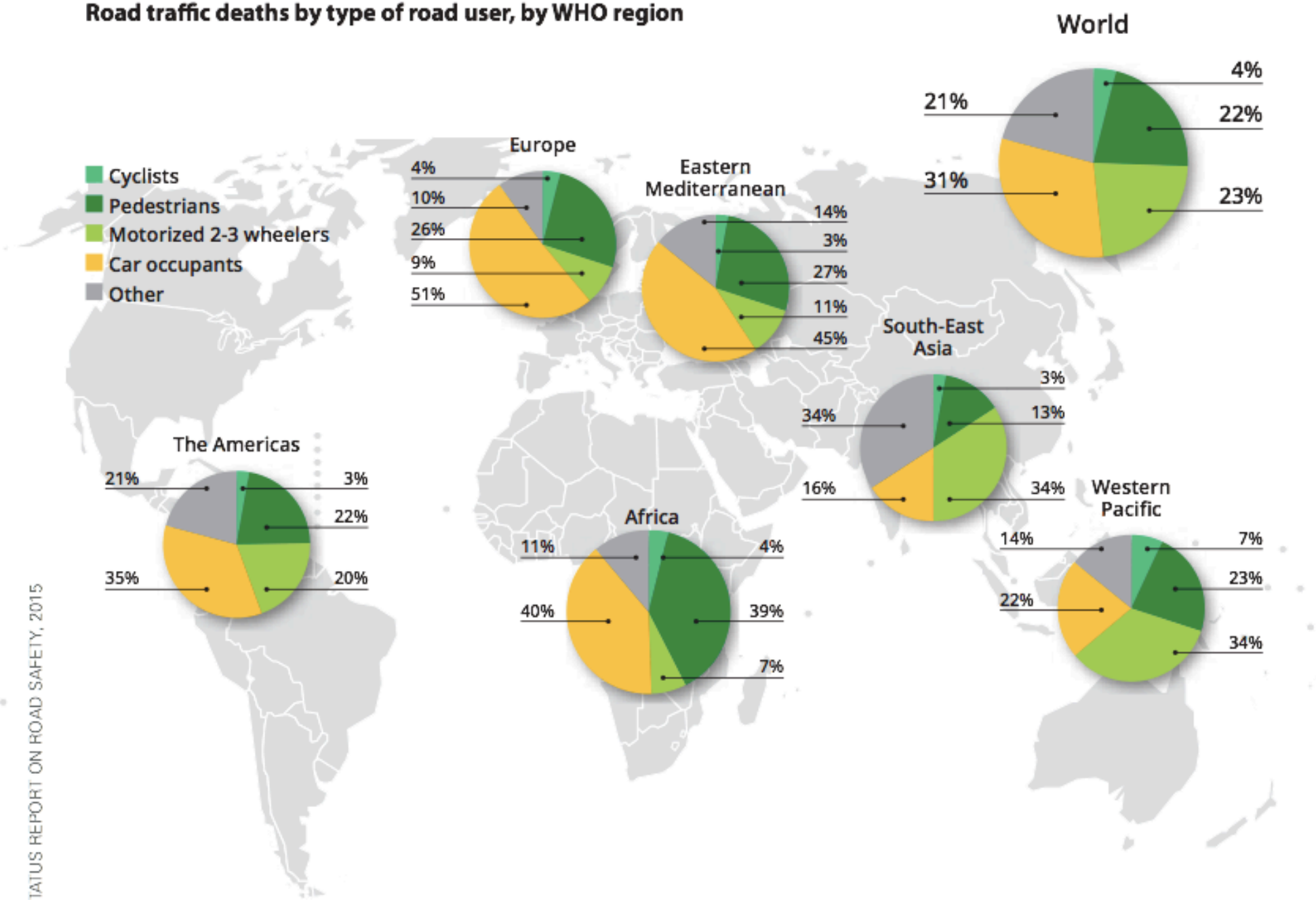
Figure 2 Number and proportion of transport injuries by type of vehicles, 2010



Source: 28 Sentinel hospitals, National Injury Surveillance System, Bureau of Epidemiology, Ministry of Public Health, Thailand

FIGURE 7

Road traffic deaths by type of road user, by WHO region



World Health Organization

GLOBAL STATUS REPORT ON ROAD SAFETY 2015

SCOPE OF PTE

Definition & Epidemiology

Risk factors

Epileptogensis & pathology

Clinical features

Diagnosis

Guideline of management

POST TRAUMATIC EPILEPSY (PTE)
POST TRAUMATIC SEIZURES (PTS)

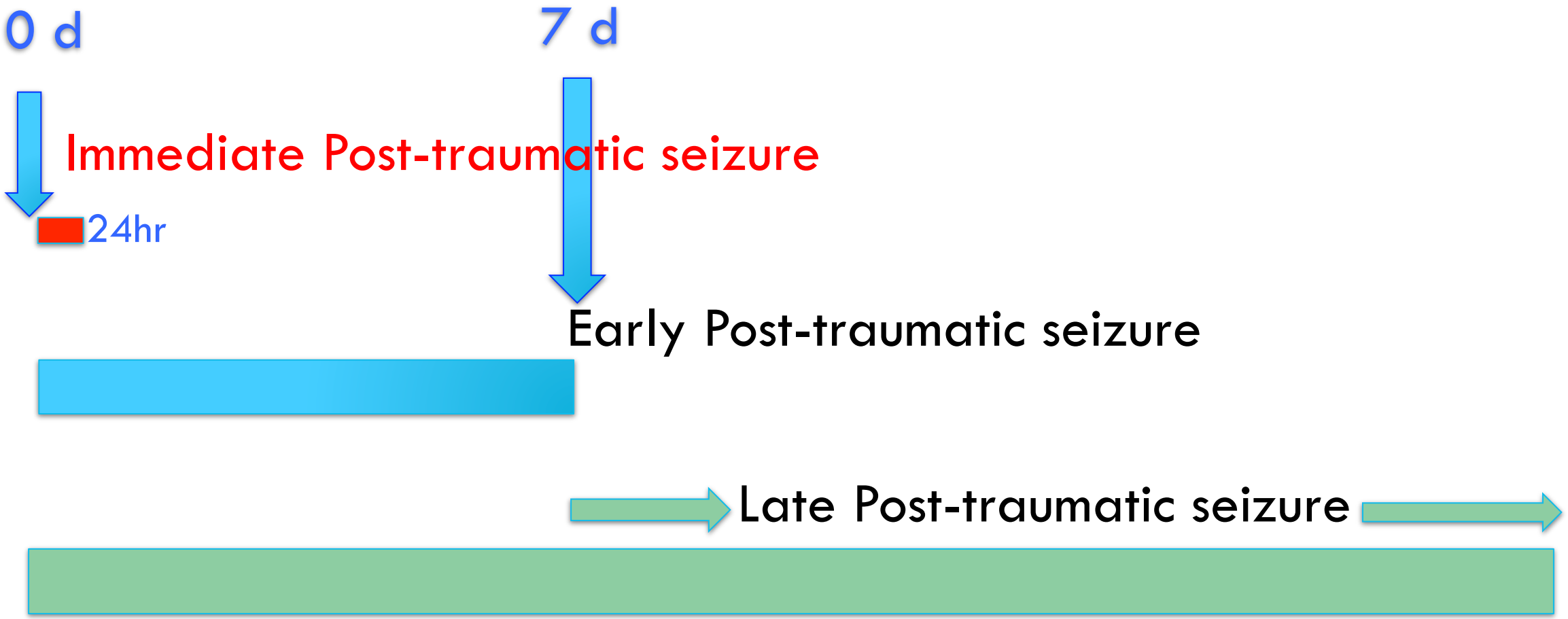
Seizures are caused related to trauma

PTS: provoked seizure by traumatic brain injury

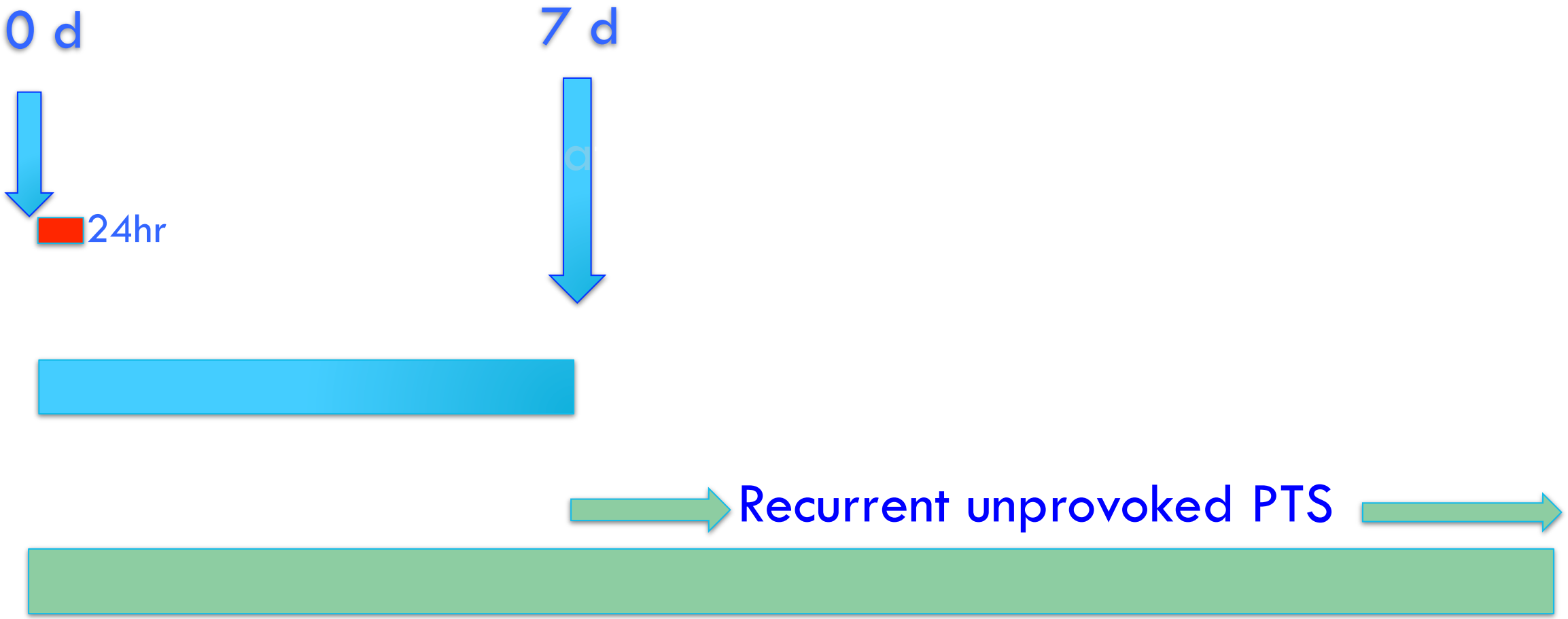
PTE: recurrent and unprovoked PTS and occur at least a week after injury

Frey LC. Epilepsia. 2003; 44(Suppl 10):11–17.
Jennett B. Arch Neurol. 1974; 30(5):394–398.]

POST-TRAUMATIC SEIZURE



POST-TRAUMATIC EPILEPSY



EPIDEMIOLOGY

Epilepsy related to TBI ≈ 2-16% in European countries

Forsgren L, et al 2005. Eur J Neurol 12:245-253

Severe TBI:

An increased risk of developing epilepsy risk 7.02 X in children

Cansu A et al 2007.Seizure 16:338-344

An increased risk of developing early and late PTS

Frey LC, et al 2003. Epilepsia 44 Suppl 10: 11-17

Post-traumatic seizure

PTS

Incidence

i. Early PTS 6-10%
   i. 50% within the first 24hrs
   ii. 25% within the first 1hr
   iii. 29-fold increased risk of developing epilepsy

Immediate PTS

ii. Late PTS 9-25%
   i. 25% of patients with early PTS will have another seizure within 6 mths
   ii. 13% of patients without early PTS

Brain Trauma Foundation. J Neurotrauma.2007;24(suppl 1):S1-106
J Englander, et al. Arch Phys Med Rehabil 2003;84:365-73.
Pagni Ca. Acta Neurochir Suppl.1990;50:38

EARLY PTS

2.6-16.9%, depending on the severity of TBI

Hahn YS, et al. 1988 Neurosurg;22: 864-867

In infant population,

Incidence of early PTSs esp.<24 hrs is more common than late PTSs

Annegers JF, et al. 1988 N Engl J Med;338: 20-24
Asikainen I, et al. 1999 Epilepsia; 40: 584-589

In general,

Severe TBIs are associated with high frequency of early PTSs.

Annegers JF, et al. 1980 Neurology;30: 683-689

SEVERITY OF TBI

	LOC	Amnesia	GCS	remarks
Mild	Y<24 hrs	< 30 mins	13-15	No skull fx
Moderate	Y<24 hrs	30mins-24hrs	9-12	Skull fx
Severe	Y>24 hrs	> 24 hrs	3-8	Contusion, intracranial hematoma

Annegers JF, et al. 2009 Lancet;373:1105-1110

RISK FOR PTS

	Standardized incidence ratio
Mild	1.5
Moderate	2.9
Severe	17.0

Annegers JF, et al. 2009 Lancet;373:1105-1110

CLINICAL FEATURES OF PTS

GTC:

- 70-80% of seizures within the 1st day

Simple Partial Sz or Focal with 2nd GTC:

- 50% of SZ, after the 1st day

Status Epilepticus : 10% of Pt with acute TBI
(more common in children & usually with other underlying complications)

Jennett B, et al. 1975 William Heinemann Med Book, England

LATE PTS

The higher risk period to develop late PTSs: the 1st 18 months

Incidence 1.9-30%, variable in different studies

An average PTS frequency of 2.1% in TBI and 12% in severe TBI

Annegers JF, et al. 1988 N Engl J Med;338: 20-24

LATE PTS

Incidence ratio: a risk of unprovoked seizure during 1st year

Incidence ratio	TBIs : Gen Pop
	x11
After 4 Ys	X 3.5
After 5Ys	X 2.39
After 10 Ys	X1.56
After 10Ys in Severe TBI	X10

Annegers JF, et al. 1988 N Engl J Med;338: 20-24

RISK FACTORS

Early Post-traumatic seizure

- Acute Intracranial Hematoma esp. subdural hematoma, intraparenchymal hemorrhage need of surgery
- Younger age
- Higher injury severity
  - PTA /LOC > 30 min
- Chronic Alcoholism

Late post-traumatic seizure

- Acute Intracranial Hematoma esp. subdural hematoma
- Brain contusions
- Age older>65 Y @ time of injury
- Alcohol withdrawal
- Higher injury severity
  - PTA/LOC > 30 min

Yeh CC, et al. 2013,J Neurol Neurosurg Psychiatry 84: 441-445

RISK FACTORS

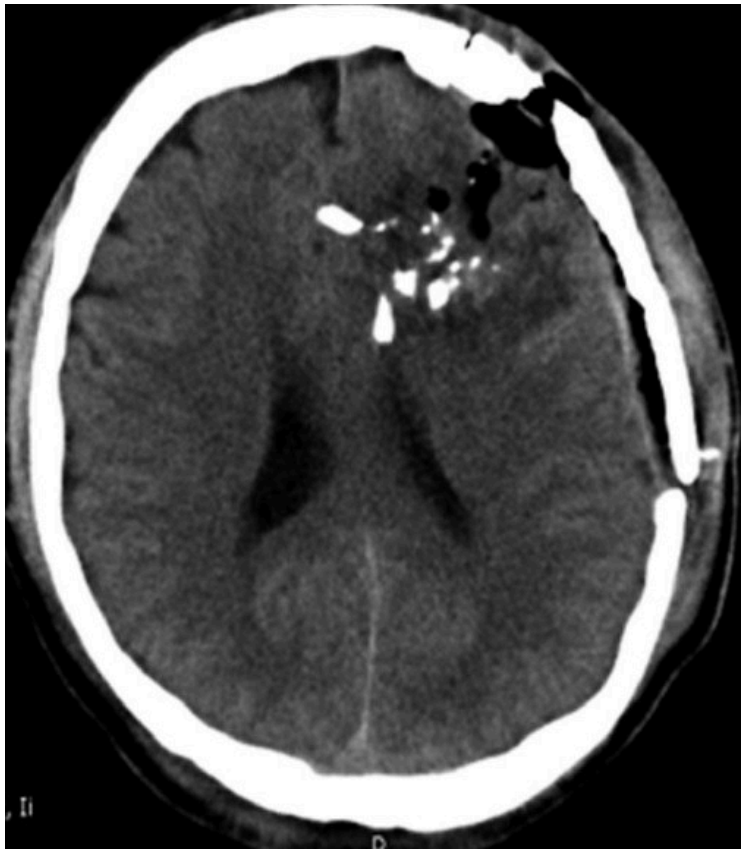
Controversial

The presence of early PTS as risk factor for late PTSs in adults

Pro: Jennett B, et al. 1975 William Heinemann Med Book, England
Angeleri F, et al. 1999; Epilepsia 40: 1222-1230
Temkin NR. 2003 Epilepsia; 44 suppl 10:18

Cons: Annegers JF, et al. 1998 N Engl J Med;338: 20-24

POST TRAUMATIC SEIZURE



VIETNAM HEAD INJURY STUDY

Raymont V, et al. 2010 Neurology,.75(3):224-9

POST-TRAUMATIC EPILEPSY

A form of symptomatic epilepsy

defined by the presence of recurrent seizures secondary to traumatic brain injury

Agrawal A, et al.2006 Clin Neurol Neurosurg ,108:433–439
Da Silva AM, st al. 2012 Handbook Clin Neurol, 108:585–599
Cesnik E, Casetta L, et al. 2013 J Neurol Neurophysiol , S2:009

PTE

10-Y incidence of PTE of any severity ≈ 2%

Annegers JF, et al. 1980 Neurology;30: 683-689

Severity of TBI correlates with risk

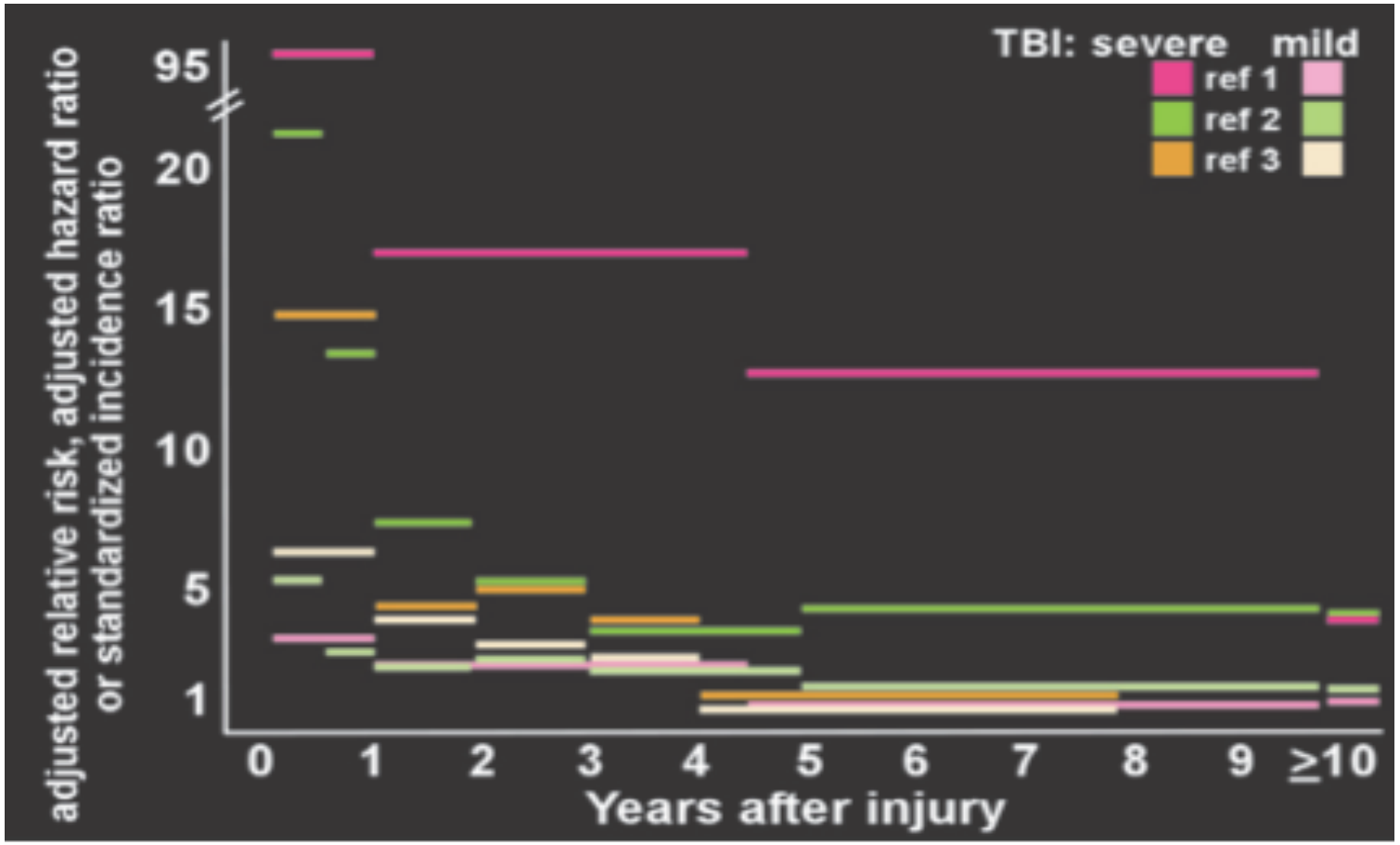
	Cumulative 5-Y probability of seizure
Mild	0.5%
Moderate	1.2%
Severe	10%

Annegers JF, et al. 1998 N Engl J Med;338: 20-24

POPULATION BASED STUDIES

Study	RISK of PTE	Mild	Moderate	Severe	
Denmark	Accum 1-Y	3.5		12.2	Christensen J, et al.2003 Lancet 2009; 373:1105–1110
N=1,605,216 (78,572)	Accum 10-Y	1.5		3.5	
USA	Accum 5-Y	0.7	1.2	10	Annegers JF, et al. 1998 N Engl J Med, 338:20–24
N=4,541	Accum 30-Y	2.1	4.2	16.7	
Taiwan N=559,658 (19,336)	15 Y	3.9		7.8	Yeh CC, et al. 2013 J Neurol Neurosurg Psychiatry ,84:441–445
USA		9.1/100 persons			Ferguson PL, et al. 2010 Epilepsia, 51:891–898
N=2,118 (>15 Y)	Accum 3-Y	4.4	7.6	13.6	
China	Accum 3-Y	5/100 persons			Zhao Y, et al.2012 Seizure ,21:322–326
N=2,826 (4-79 Y)		3.6	6.9	17%	

POPULATION BASED STUDIES



Christensen J, et al.2003 Lancet 2009; 373:1105–1110

NATURAL HISTORY

80% pts with PTE tend to have the 1st unprovoked sz within the 1st year

Annegers JF, et al. 1988 N Engl J Med;338: 20-24

90% PTEs start within two years from trauma

In Military population,

PTE frequency (34-53%) show higher rate than in general pop.

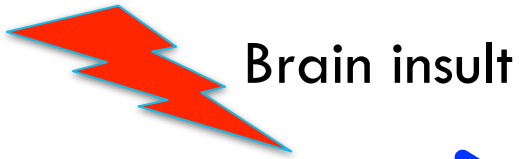
Caveness WF, et al. 1962 J Neurosurg;19: 122-129
Sa;azar AM, et al. 1985 Neurology; 35: 1406-1414

Remission rate: 25-40%

Haltiner AM, et al. 1997; Arch Phys Med Rehabil 78: 835-840

HUMAN EPILEPTOGENESIS

Agrawal A, et al.2006 Clin Neurol Neurosurg ,108:433–439
Da Silva AM, st al. 2012 Handbook Clin Neurol, 108:585–599
Cesnik E, Casetta L, et al. 2013 J Neurol Neurophysiol , S2:009
Willmore LJ, 2009. Neurochem Res,34:688–697



Brain insult

A nonepileptogenic brain → An epileptogenic brain

Molecular & Cellular alternation

Latent period
(days to weeks)

Axonal sprouting & dendritic modificaion

Increasing cell excitability

Cicatrix (scar) in cortex

Ischemia and hemorrhage

Toxic effect of hemoglobin breakdown

Diffuse axonal injury

Secondary cellular injury (excitatory

Hyperexcitability, Hypersynchronization, and Development of recurrent seizures

PATHOGENESIS

2 main pathogenic pathway caused late seizures

Oxidative stress mechanisms

- Lipid peroxidation of neuronal mb and mitochondria, alt Na+/K+ pump
- A reduction of the chronic threshold of a group of neuronal cells

Excitotoxic-mechanisms-neuronal hyper excitability

- Extra cellular immediately increase of excitatory amino acid after injury, increased levels of glutamate and aspartatic acid.
- Increased expression of SNAT1 and SNAT2, form axonal sprouting with high immumoreactivity to GAP 43

Katayama Y, et al. 1990; J Neurosurg 73: 889-900
Tani H, et al. 2007; Neurobiol Dis 25: 230-238
McKinney RA, et al. 1997; Nat Med 3: 990-996

PATHOLOGY

Trauma can induce hippocampal epilepsy and injury processes can be progressive

Hippocampal degeneration

- to be more severe in patients surviving more than 6 months compared with patients surviving less than 1week

Maxwell WL, et al.2003 J Neuropathol Exp Neurol. 62:272-279.

Hippocampal atrophy

- a common finding among chronic TBI survivors and in various experimental models

Bramlett HM, et al.2002 Acta Neuropathol. 103:607-614

Histopathological examination

- neocortical gliosis was present in all specimens and hippocampal neuronal loss occurred in 94% of the cases

Bramlett HM, et al.2002 Acta Neuropathol. 103:607-614

CLINICAL FEATURE OF PTE (1)

FOCAL EPILEPSY /

FOCAL EPILEPSY WITH A SECONDARY GENERALIZATION

MESIAL TEMPORAL LOBE EPILEPSY (Trauma induced MTS in patient with history trauma in childhood< 5 Y)

Diaz-Arrastia R, et al. 2000 Arch Neurol, 57: 1611-1616
Marthern GW, et al. 1994 Epilepsy Res, 19: 129-139

Status Epilepticus:

10% of PTSs, infant population

Jennett B, et al. 1975 William Heinemann Med Book, England

CLINICAL FEATURES OF PTE (2)

Clinical manifestation: variable & depend on the cortical areas involved.

Medically intractable epilepsy

- 78%–91% of focal onset seizures
- Temporal lobe in 15%–56%, Frontal lobe in 23%–36%, Occipital lobe in 2%–6%, and parietal lobe in 5%–38%.

Kazemi H,, et al. 2012 Injury, 43:2132–2135
Hudak AM, et al. 2004 J Head Trauma Rehabil, 19:290–295

CLINICAL FEATURE OF PTE (3)

Parietal semiology found more frequently in patients with penetrating trauma,

whereas patients with blunt trauma showed higher temporal and frontal semiologies

Kazemi H, et al. 2012 Injury, 43:2132–2135

PTE manifested as medial TLE in patients with Hx of TBI younger than age of 5 Y

Recent study, also found in patient at older age (30-35%)

Diaz-Arrastia R, et al. 2000 Arch Neurol, 57:1611– 1616
Hudak AM, et al. 2004 J Head Trauma Rehabil, 19:290–295
Marks DA, et al:. 1995 Neurology, 45:2051–2057

RISK FACTORS OF PTE

Advanced age,

Penetrating injuries,

Injury severity (e.g. neurosurgical procedure, intracranial hemorrhage, greater than 5 mm midline shift, duration of coma 24 hours, loss of consciousness 24 hours, prolonged length of post- traumatic amnesia),

Biparietal or multiple contusions,

Frontal or temporal locations of the lesion.

Agrawal A, et al. 2006 Clin Neurol Neurosurg,108:433–439
Da Silva AM, et al. 2012 Clin Neurol,108:585–599
Cesnik E, Casetta L, et al. 2013.J Neurol Neurophysiol,S2:009
Lowenstein DH.2009 Epilepsia ,50(Suppl 2):4–9
Christensen J. 2012 Epilepsia , 53 (Suppl 4):43–47

DIAGNOSIS

Electroencephalogram (EEG)

- Useful for the localization of the lesion focus and measurements of the extent of damage
- Unable to define the probability to develop epilepsy;
  - >20% of pts with PTE have negative EEG during the 1st three months after injury

Jennett B, et al. 1975; Epilepsia 16: 251-256

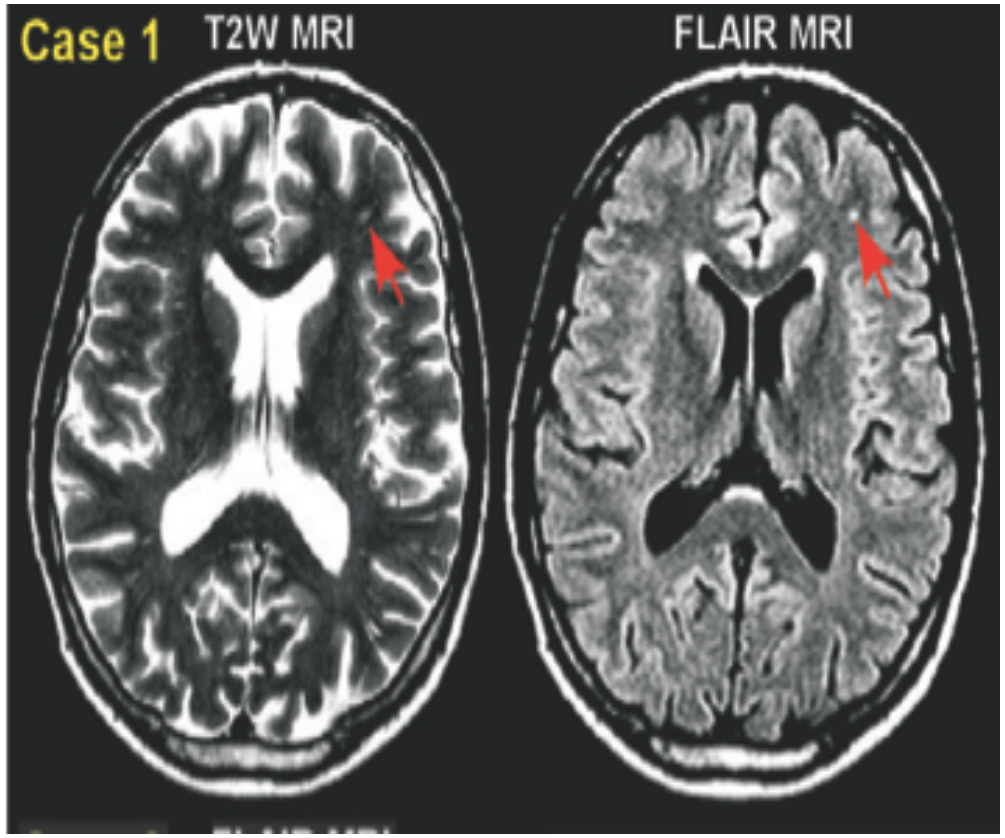
Neuroimaging

- CT scan of brain; immediately trauma
- MRI brain; study of choice in late seizures

A 58-Y-man who had a mild TBI as a teenager

He developed GTC within 5 years after injury.

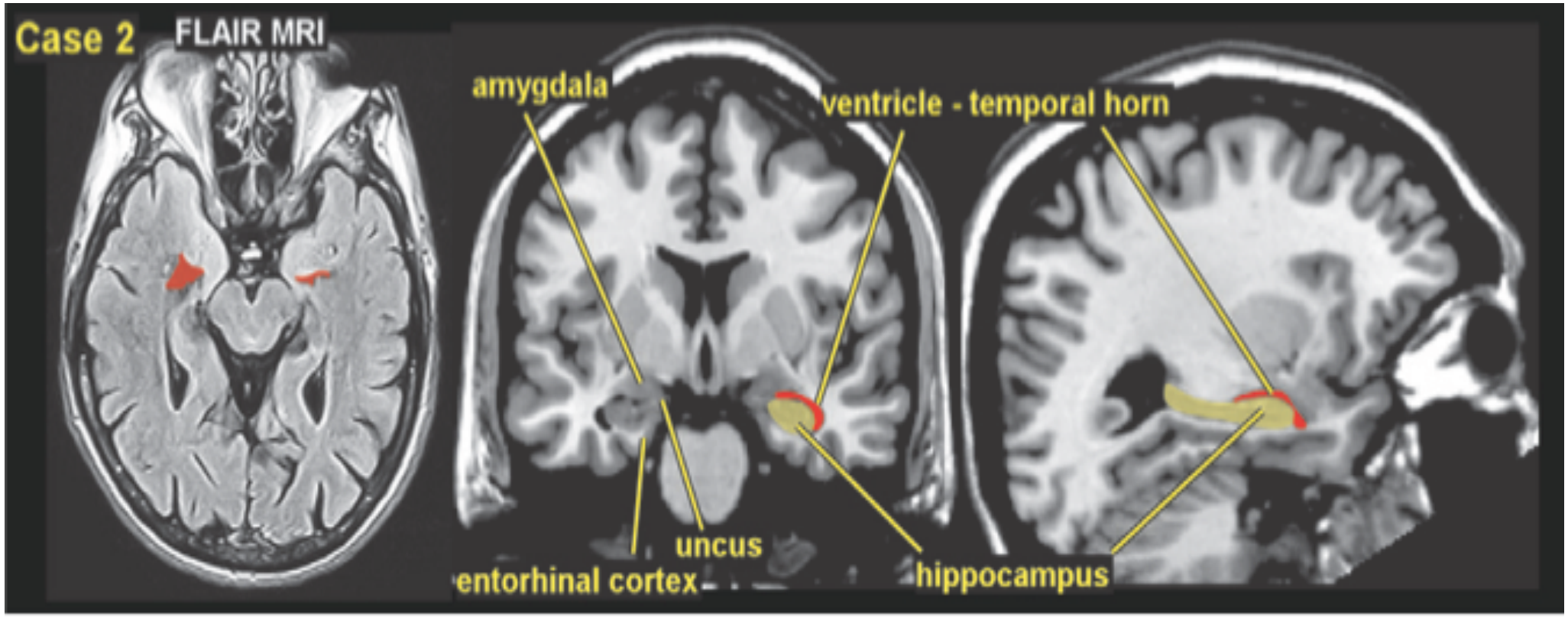
MRI brain: a single 3mm non-specific gliotic lesion in the left frontal lobe.



Lamar CD, et al.2014 J Neuropsychiatry Clin Neurosci 26:2, 108-113

A 49-Y man with previous history of TBI as a fall from a ladder.

MRI brain: dilatation of the tip of the right temporal horn due to volume loss in the mesial temporal structure.

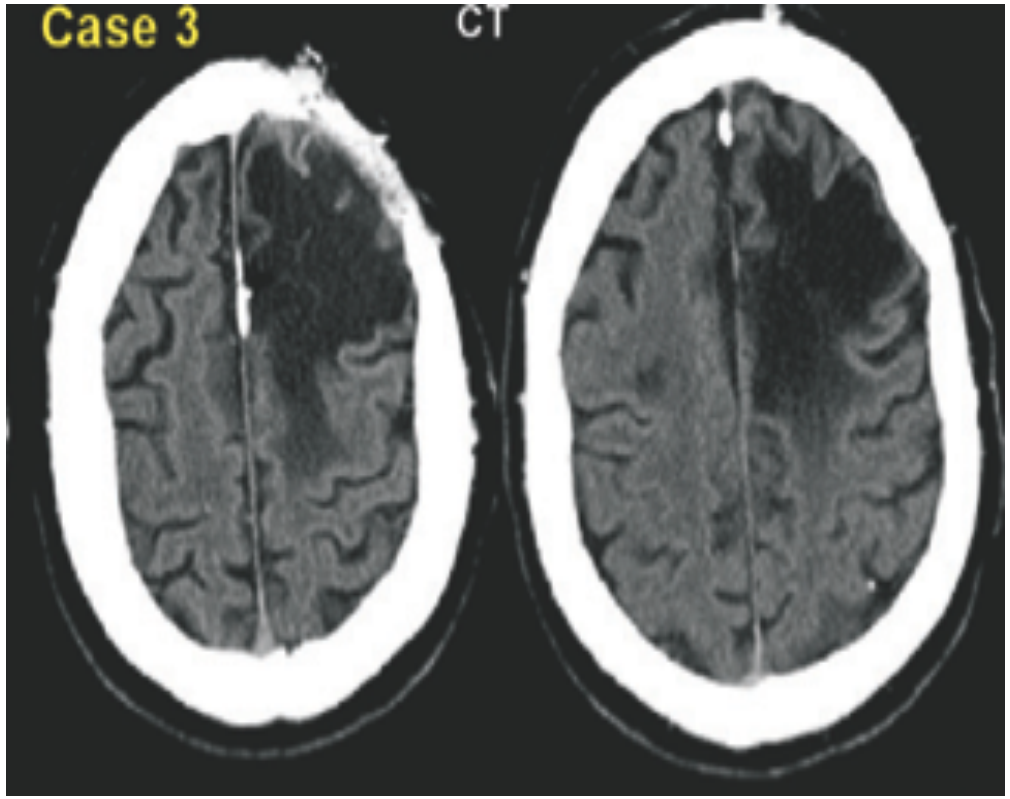


Lamar CD, et al.2014 J Neuropsychiatry Clin Neurosci 26:2, 108-113

A 66-Y-man suffered from severe TBI caused by GSW at the age of 19.

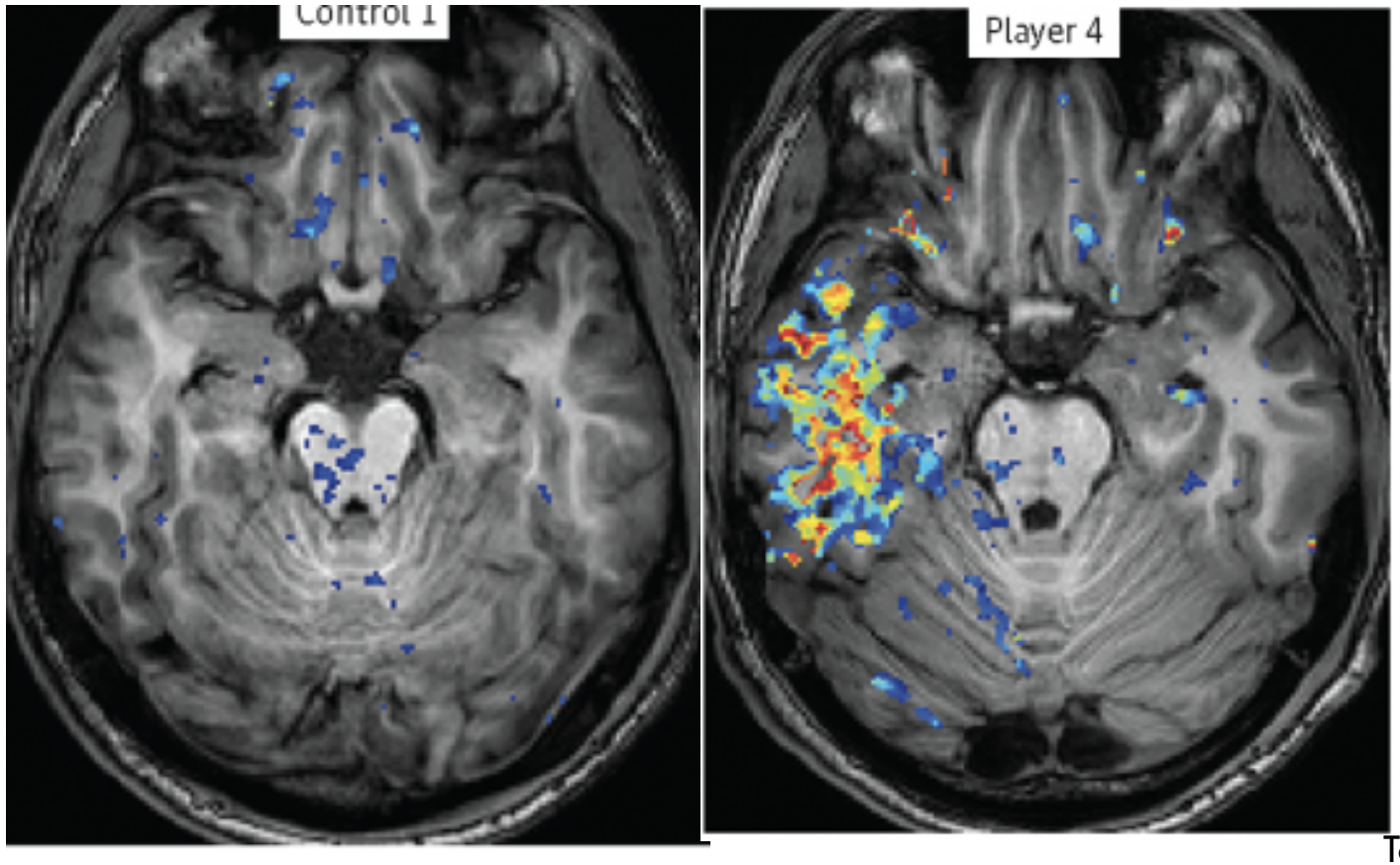
He developed sz within 2 years following this injury.

CT brain: Chronic postoperative changes with a large area of encephalomalacia at the left frontal lobe.



Lamar CD, et al.2014 J Neuropsychiatry Clin Neurosci 26:2, 108-113

POST TRAUMATIC MRI FINDING



Blood-Brain Barrier (BBB) Permeability in Football Players vs. a control group
Weissberg Itai, et al 2014 .JAMA Neurol, 71(11);1453-1455

Microstructure changes e.g. microhemorrhage or white matter injury (SWI/DTI)

Gliosis and/or residual hemosiderin (T1W & T2W MRI)

BBB disruption (contrast-enhanced MRI) more frequent in PTE group (82.4% VS 25%

Tomkins O, et al. 2011Cardiovasc Psychiatry Neurol,:765923.

ANTICONVULSANTS

Goal:

To minimize the brain damage by preventing early seizures

Schierhout G, et al. Neurol Neurosurg Psychiatry,1998.64:108-12

To create neuroprotective effect

Vartanian MG, et al.Brain Res Dev Brain Res , 1996.95(2):169-75
Tasker RC, et al. J Neurosci,1992.12(11):4298-308.

KEY FACTS

Anticonvulsants:

to reduce early PTS occurring within the first 7 days

But little to no benefits to reduce late PTS occurring after 7 days

Guidelines for the management of severe traumatic brain injury
XIII. Anti-seizure prophylaxis. 2007

PROPHYLAXIS

Use of AEDs at early stage

- to prevent PTE; Controversial
- to prevent of early PTS; Good efficacy

Epilepsia, 50(Suppl. 2): 10–13, 2009
doi: 10.1111/j.1528-1167.2008.02005.x

POSTTRAUMATIC EPILEPSY

Preventing and treating posttraumatic seizures: The human experience

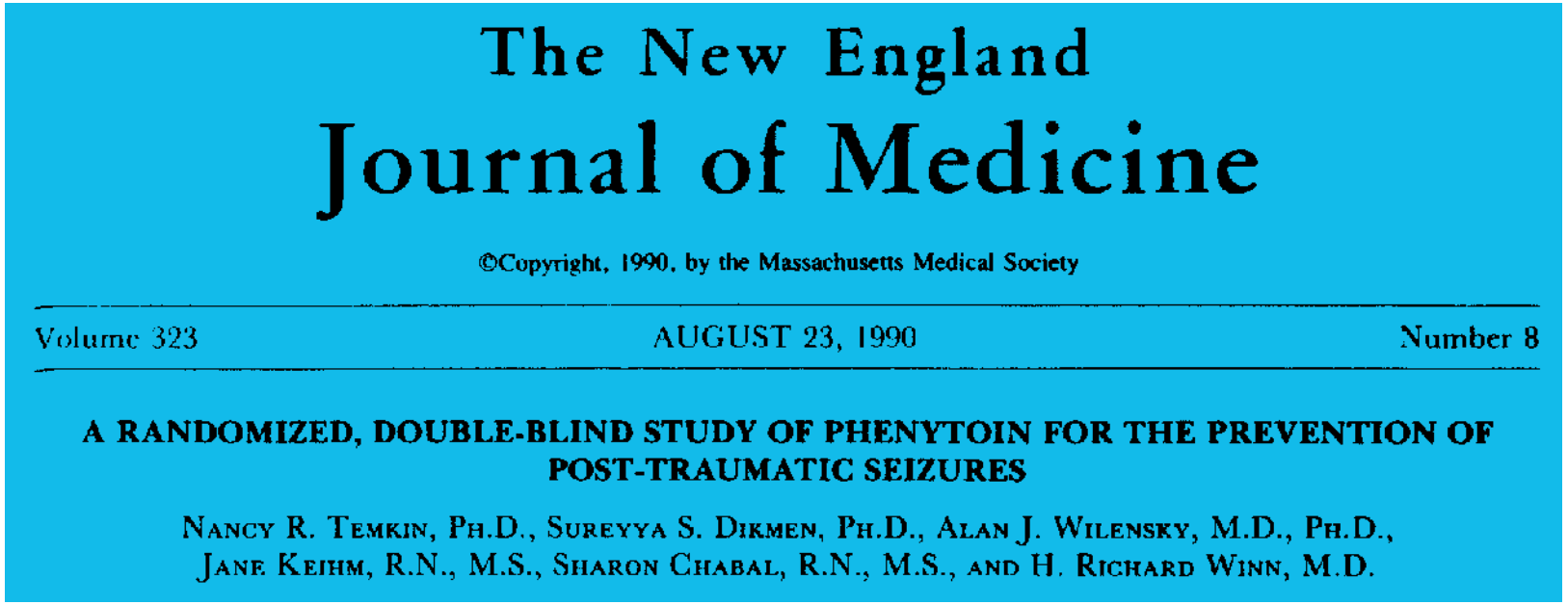
Nancy R. Temkin

Departments of Neurological Surgery and Biostatistics, University of Washington, Seattle, Washington, U.S.A.

SUMMARY

Posttraumatic epilepsy presents an ideal target for prevention efforts. Traumatic brain injury (TBI) is common, characteristics that put people at high risk such as penetrating injury or subdural hematoma or provoked seizures are easily identified, and the latency between the injury and the onset of epileptic seizures is frequently short. Several drugs have been tested for their ability to prevent provoked seizures and epilepsy after TBI. We describe the design of those studies and their results. Phenytoin and carbamazepine significantly reduce the incidence of provoked seizures. Phenobarbital and the combination of phenobarbital and phenytoin also look promising for reducing provoked seizures, but small sample sizes in the studies evaluating these drugs do not allow definitive conclusions. None of the drugs studied (phenytoin, phenobarbital, their combination, carbamazepine, valproate, or magnesium) have shown reliable evidence that they prevent, or even suppress, epileptic seizures after TBI. For most of the regimens tested (the phenytoin/phenobarbital combination being the exception), the best estimate of effect is under a 25% reduction in posttraumatic seizures, well less than the 50% reduction most studies were designed to detect. The evaluation of the tested drugs has serious limitations, however, and antiepileptic drugs (AEDs) developed since 1980 and other compounds have barely been tested at all. Better understanding the process of epileptogenesis, testing treatments that demonstrate antiepileptogenic effects in the laboratory, and performing thorough preclinical and phase II evaluations before attempting definitive trials should greatly improve the chance of identifying ways to prevent posttraumatic epilepsy, providing the ultimate cure for this condition.

KEY WORDS: Antiepileptogenesis, Prophylaxis, Posttraumatic epilepsy, Meta-analysis, Antiepileptic drugs.



The New England
Journal of Medicine

Volume 323 AUGUST 23, 1990 Number 8

A RANDOMIZED, DOUBLE-BLIND STUDY OF PHENYTOIN FOR THE PREVENTION OF POST-TRAUMATIC SEIZURES

NANCY R. TEMKIN, PH.D., SUREYYA S. DIKMEN, PH.D., ALAN J. WILENSKY, M.D., PH.D.,
JANE KEIHM, R.N., M.S., SHARON CHABAL, R.N., M.S., AND H. RICHARD WINN, M.D.

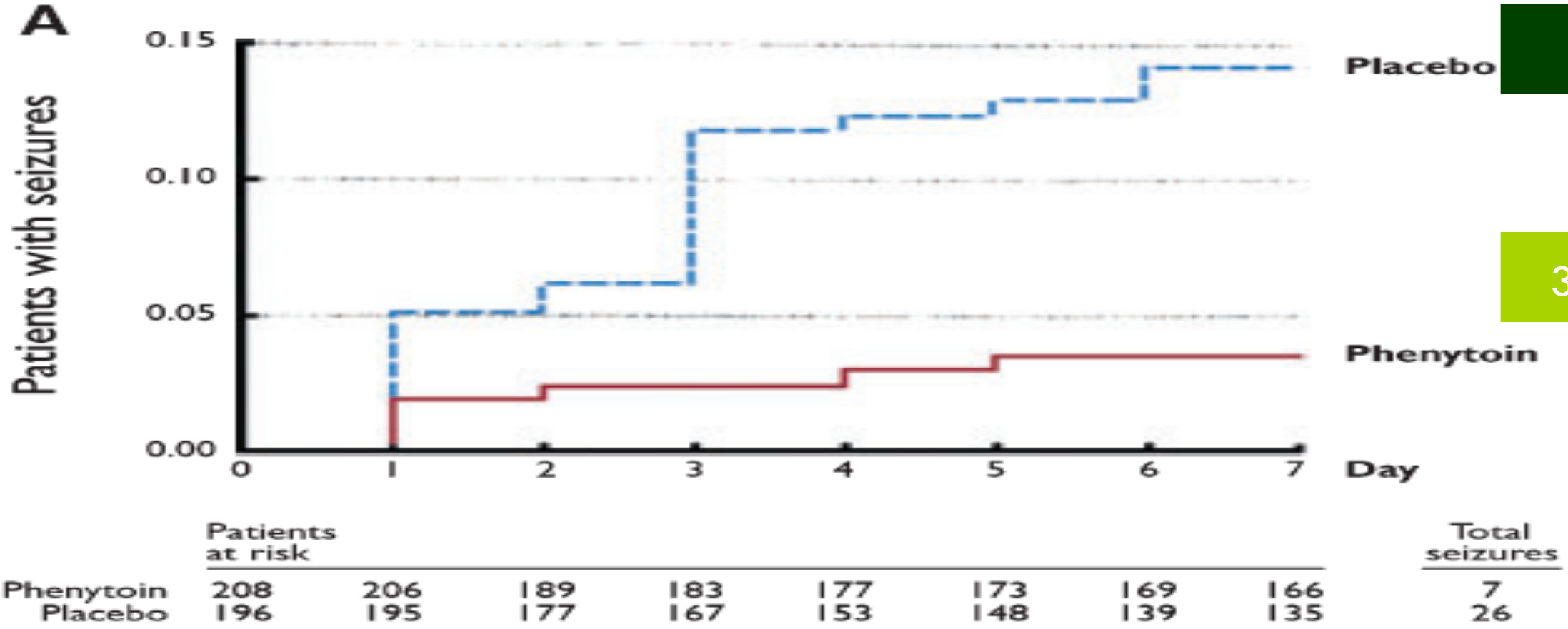
404 post traumatic head injury patients (GCS 3-10 and abnormal head CT) randomized to treatment with phenytoin or placebo for one year with a two year follow up.

In the first week after injury 4% of the patients receiving phenytoin had seizures compared to 14% taking placebo.

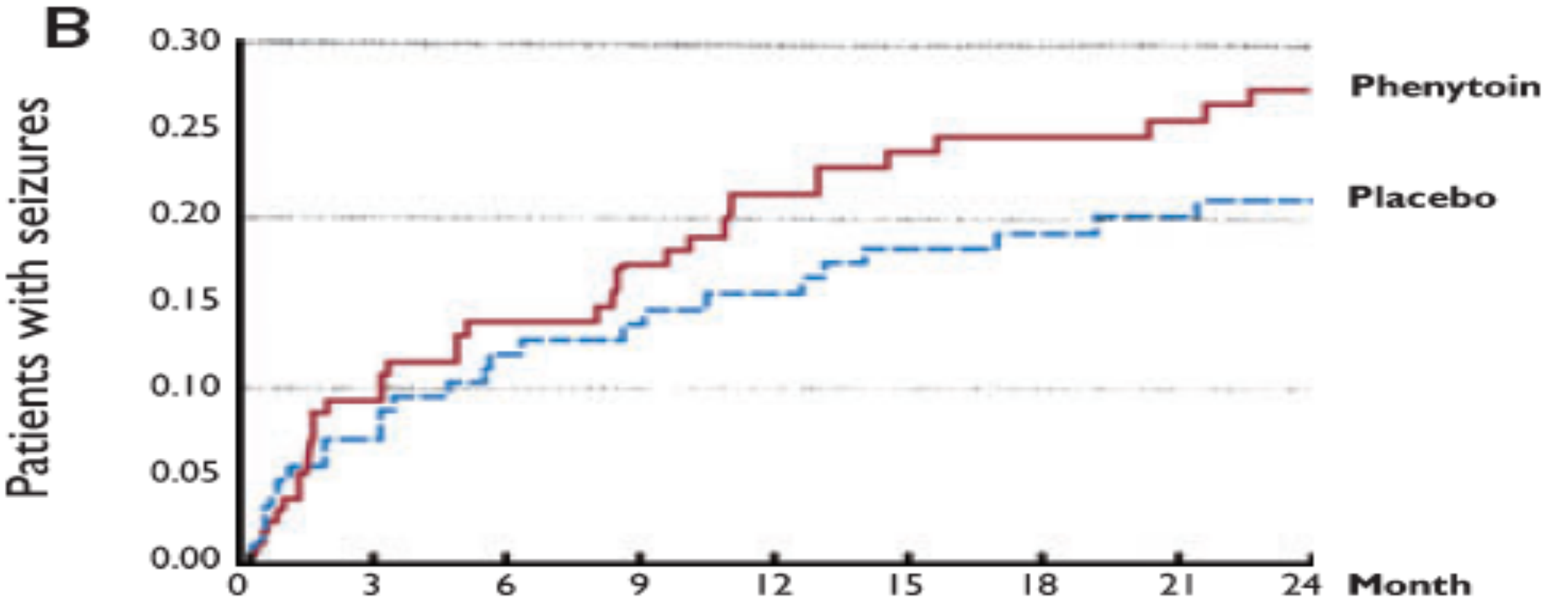
After the first week there was no significant difference between the rate of seizures in the two groups.

Temkin NR, Dikmen SS. *N Eng J Med* 1990; 323:497-502

A Randomized,double-blind study of phenytoin for the prevention of post-traumatic seizures.

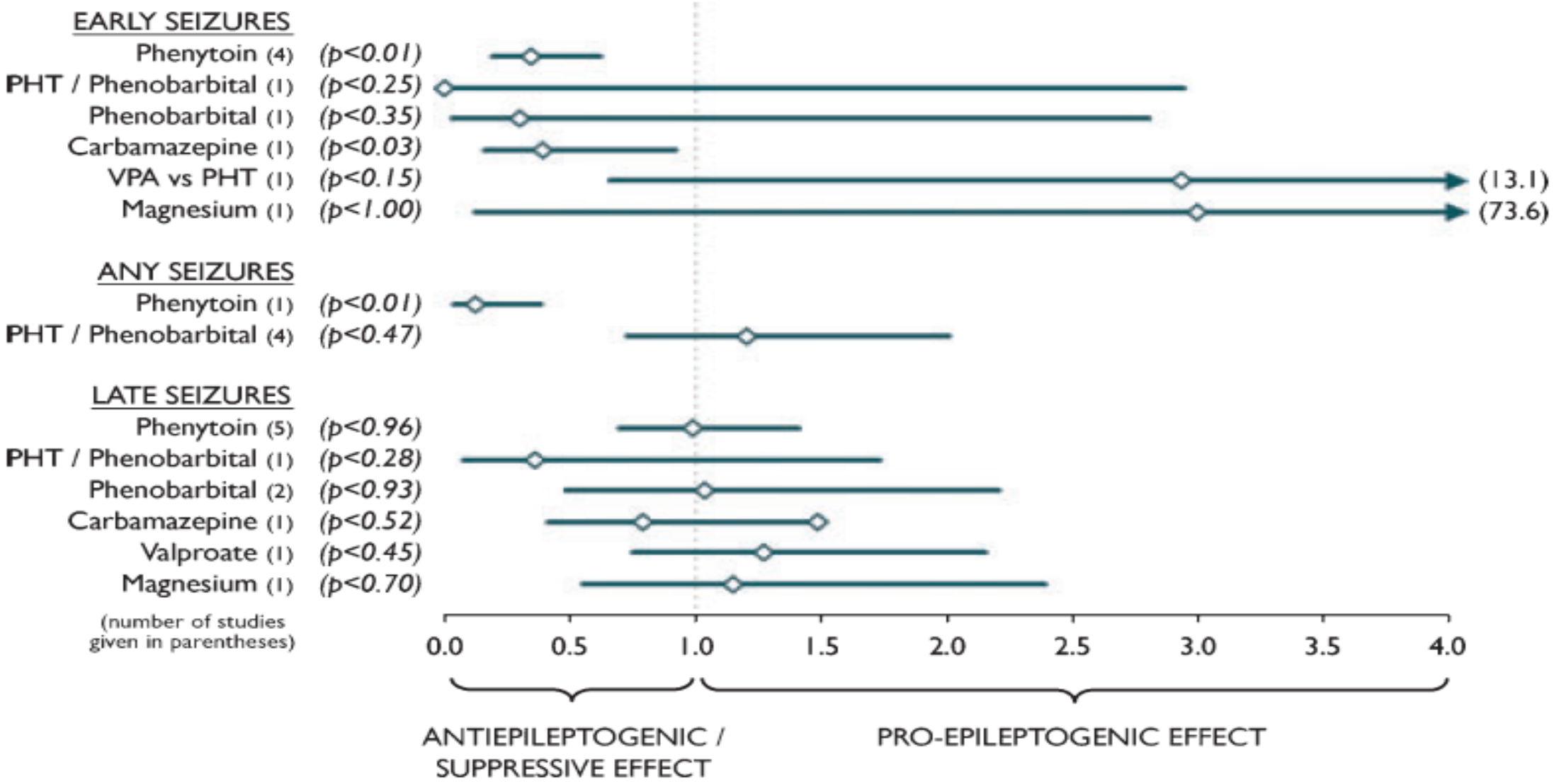


Temkin NR, Dikmen SS. *N Eng J Med* 1990; 323:497-502



	Patients at risk									Total seizures
Phenytoin	170	123	108	101	95	88	86	85	74	36
Placebo	153	113	106	100	94	90	87	86	77	26

Temkin NR, Dikmen SS. *N Eng J Med* 1990; 323:497-502



Temkin NR. *Epilepsia* 2001; 42:515-524

Chang BS, Lowenstein HD. *Neurology* 2003;60:10-16

AMERICAN ACADEMY OF NEUROLOGY

Special Article

CME

Practice parameter: Antiepileptic drug prophylaxis in severe traumatic brain injury

Report of the Quality Standards Subcommittee of the American Academy of Neurology

Bernard S. Chang, MD; and Daniel H. Lowenstein, MD

Abstract—*Objective*: To review the evidence regarding antiepileptic drug (AED) prophylaxis in patients with severe traumatic brain injury (TBI) in order to make practice recommendations. *Methods:* The authors identified relevant studies by searching multiple databases and reviewing reference lists of other sources. They included studies that prospectively compared post-traumatic seizure rates in patients given AED prophylaxis vs controls. Each study was graded (class I to IV) according to a standard classification-of-evidence scheme and results were analyzed and pooled. *Results:* Pooled class I studies demonstrated a significantly lower risk of early post-traumatic seizures (those occurring within 7 days after injury) in patients given phenytoin prophylaxis compared to controls (relative risk 0.37, 95% CI 0.18 to 0.74). Pooled class I and class II studies demonstrated no significant difference in the risk of late post-traumatic seizures (those occurring beyond 7 days after injury) in patients given AED prophylaxis compared to controls (relative risk 1.05, 95% CI 0.82 to 1.35). Serum AED levels were suboptimal in these studies and adverse effects were mild but frequent. *Conclusions:* For adult patients with severe TBI, prophylaxis with phenytoin is effective in decreasing the risk of early post-traumatic seizures. AED prophylaxis is probably not effective in decreasing the risk of late post-traumatic seizures. Further studies addressing milder forms of TBI, the use of newer AEDs, the utility of EEG, and the applicability of these findings to children are recommended.

NEUROLOGY 2003;60:10–16

The Quality Standards Subcommittee (QSS) of the American Academy of Neurology (AAN) is charged with developing practice parameters for neurologists for diagnostic procedures, treatment modalities, and clinical disorders. Practice parameters are strategies for patient management that assist physicians in clinical decision-making. They comprise one or more recommendations based on analysis of evidence on a specific clinical problem. This report addresses the prophylactic use of antiepileptic drugs (AEDs) in patients with severe traumatic brain injury (TBI).

the United States.[1,2] Among all patients with head trauma who seek medical attention, about 2% develop post-traumatic seizures, although the number varies widely depending primarily on injury severity. About 12% of patients with severe TBI develop post-traumatic seizures, and the rate may be more than 50% for those with penetrating missile injuries.[3-5]

The use of AEDs to treat patients who have developed post-traumatic epilepsy is standard. However, the important question of whether to use AEDs prophylactically after TBI to prevent the development of

PTS

- "Prophylactic treatment with PHT should be initiated as soon as possible after injury to decrease the risk of Post traumatic seizures occurring within the first 7 days"

(evidence level A)

Chang BS, Lowenstein HD. *Neurology* 2003;60:10-16

PTE

- "PHT, CBZ or VPA should not routinely be used beyond the first 7 days after injury to decrease the risk of Post traumatic seizures occurring beyond that time"

(evidence level A)

Chang BS, Lowenstein HD. *Neurology* 2003;60:10-16

CURRENT GUIDELINES

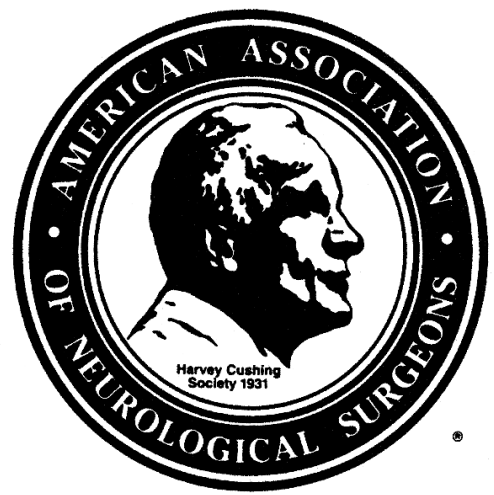
American Academic of Neurology (AAN) 2003

Brain Trauma Foundation 2007

Clinical Practice Guidelines for Traumatic Brain Injury 2013 (Thai version)

GUIDELINES *for the Management of*

SEVERE HEAD INJURY



A Joint Initiative of:

The Brain Trauma Foundation
The American Association of Neurological Surgeons
The Joint Section on Neurotrauma and Critical Care

แนวทางเวชปฏิบัติกรณีสมองบาดเจ็บ

(Clinical Practice Guidelines for Traumatic Brain Injury)

โดย ● วิทยาลัยประสาทศัลยแพทย์แห่งประเทศไทย ● ราชวิทยาลัยศัลยแพทย์แห่งประเทศไทย
● กรมการแพทย์ กระทรวงสาธารณสุข ● สถาบันประสาทวิทยา กรมการแพทย์ กระทรวงสาธารณสุข
● กรมแพทย์ทหารบก ● กรมแพทย์ทหารอากาศ ● สำนักงานคณะกรรมการการอุดมศึกษา

CURRENT GUIDELINES

American Academic of Neurology (AAN) 2003

i. Prophylactic treatment with PHT as soon as possible
ii. Prophylaxis NOT routinely used after 7 d

Brain Trauma Foundation 2007

i. PHT shown to reduce incidence of early PTS
ii. VPA has comparable effects but higher mortality
iii. Seizure prophylaxis > 1 wk after TBI not recommended

Clinical Practice Guidelines for Traumatic Brain Injury 2013 (Thai version)

CURRENT GUIDELINES

American Academic of Neurology (AAN) 2003

Brain Trauma Foundation 2007

Clinical Practice Guidelines for Traumatic Brain Injury 2013 (Thai version)

- Consider AEDs for highly risk patients
  - Immediate PTS/ known epilepsy patients
  - Prophylaxis:
    - Intracranial hemorrhage -Penetrating head injury
    - GCS<10 -Depressed skull fracture

ANTI-SEIZURE PROPHYLAXIS

Early Post Traumatic Seizures (PTS) prophylaxis

Drug of Choice:

- Phenytoin as frontline drug
- Carbamazepine and Levetiracetam as second choice drugs

Phenobarbital and Valproate; not indicated

*Zonisamide; AED with antioxidant efficacy

Late Post Traumatic Seizures prophylaxis

NOT recommended AEDs

CONCLUSION

Most PTE start within two years from trauma

Severe TBI tend to correlate with increased risk of developing early and late PTSs

Prophylactic AEDs prevent early PTSs, but not Late PTSs

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