Management of Traumatic Brain Injury

COLLEGE OF MEDICINE
The UNIVERSITY *of* OKLAHOMA HEALTH SCIENCES

OU Health

Acute Care Surgery

Management of Traumatic Brain Injury (TBI)

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Disclosure of Financial Relationships

Emily E Switzer

I have no financial relationships or affiliations with ineligible companies to disclose.

Learning Objectives

1. Discuss Stats on TBI
2. Work-up of TBI, including BIG guidelines
3. Identify the different types of traumatic brain injury
4. TBI pathophysiology
5. Management strategies for TBI
6. Indications for ICP monitoring and Craniectomy
7. Anticoagulation after TBI
8. Prognostication

Definition of TBI

Position statement: definition of traumatic brain injury.

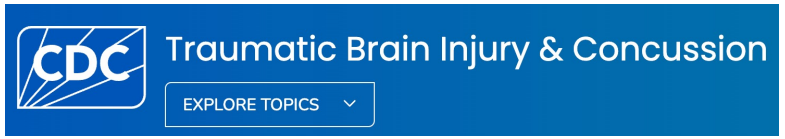
Menon DK, Schwab K, Wright DW, Maas AI, Demographics and Clinical Assessment Working Group of the International and Interagency Initiative toward Common Data Elements for Research on Traumatic Brain Injury and Psychological Health

Arch Phys Med Rehabil. 2010;91(11):1637.

- **“an alteration in brain function, or other evidence of brain pathology, caused by an external force”**

EPIDEMIOLOGY OF TBI

Epidemiology of TBI



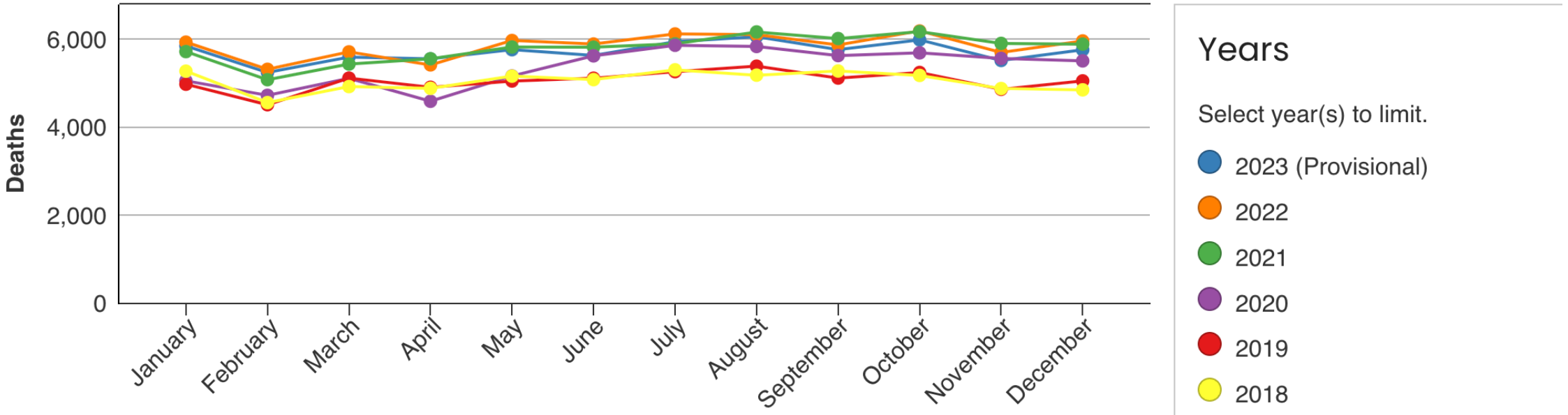
- Leading cause of death in trauma patients in US
  - 30% of ALL deaths from trauma
- Major cause of death and disability worldwide
  - 70 million people/year
- Socioeconomic burden
  - $$$$ ($76Billion/year)
  - 1% of US population lives with TBI

TBI – US fatalities/month

Traumatic Brain Injury & Concussion

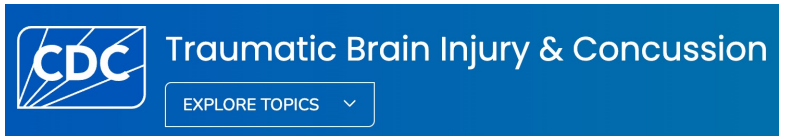
EXPLORE TOPICS

2023 = 68,666 deaths



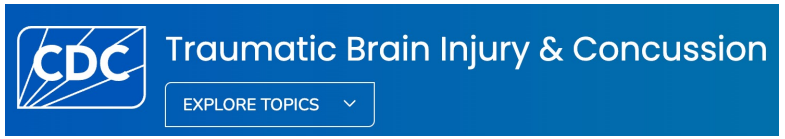
2018 = 60,565 deaths

TBI Stats (US)



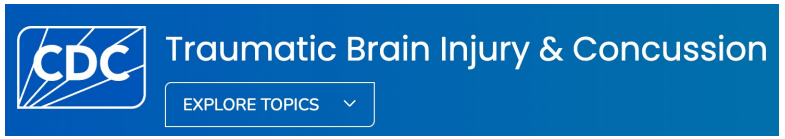
- 214,110 TBI-related hospitalizations in 2020
- 586 TBI-related hospitalizations per day(2020)
- 190 TBI-related deaths per day (2020)
  - Does not include TBIs that are not admitted or treated in outpatient settings

TBI Stats (US)



- A leading COD/disability in children/teens
  - Age 0-4 2nd highest TBI diagnoses and hospitalization rates
- >75 years highest rates of TBI diagnoses, hospitalizations, and deaths
  - With increasing age/ falls--> increase mortality
- M >>> F

Epidemiology of TBI



- MVC > Falls > Suicide > Homicide cause of TBI
  - (some quote falls now #1)
  - Suicide/firearms most common cause of TBI related death
    - Highest rates in patients > 75 years

Risk factors for TBI

- Low socio-economic status
- Substance abuse
- Psychiatric/cognitive disorders

Prevalence and correlates of traumatic brain injuries among adolescents.
Ilie G, Boak A, Adlaf EM, Asbridge M, Cusimano MD
JAMA. 2013;309(24):2550.

Risk and outcomes for traumatic brain injury in patients with mental disorders.
Liao CC, Chiu WT, Yeh CC, Chang HC, Chen TL
J Neurol Neurosurg Psychiatry. 2012 Dec;83(12):1186-92. Epub 2012 Jul 8.

PATHOPHYSIOLOGY OF TBI

Primary vs Secondary TBI

- Two distinct phases
  - Primary
    - Injury that occurs at the time of the trauma
  - Secondary
    - Cascade of injury that starts at the time of the trauma and continues
    - Treatment goal to prevent elevated ICP and herniation

Primary vs Secondary TBI

Traumatic Brain Injury
An Overview of Epidemiology, Pathophysiology, and Medical Management

Allison Capizzi, MD[a], Jean Woo, MD[b], Monica Verduzco-Gutierrez, MD[c,*]

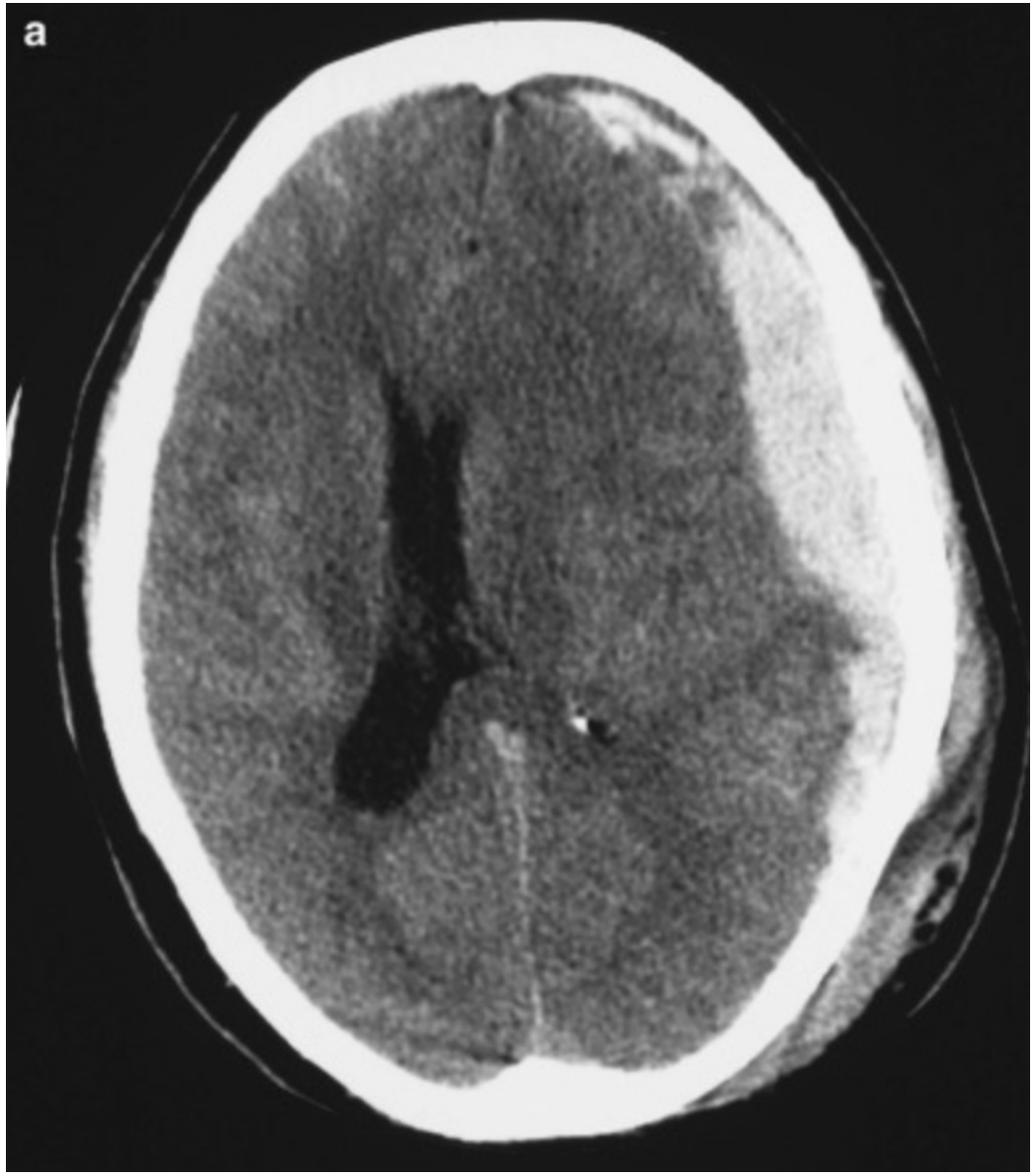
Primary Injury	Secondary Injury
• Direct hit can produce indirect damage through acceleration-deceleration (coup contrecoup) mechanism • Penetrating (open) vs nonpenetrating (closed) • Considered the period of focal injury, which can progress to become diffuse through secondary injury mechanisms	• Damage at the cellular/molecular level • Ischemia causes cell death • Vasogenic edema = extracellular edema, associated with cerebral contusion • Cytogenic edema = intracellular edema, associated with hypoxic and ischemic injury

Types of TBI

- Extra-axial
  - SDH
  - EDH
  - SAH
- Intra-axial
  - IPH
- Shearing
  - DAI

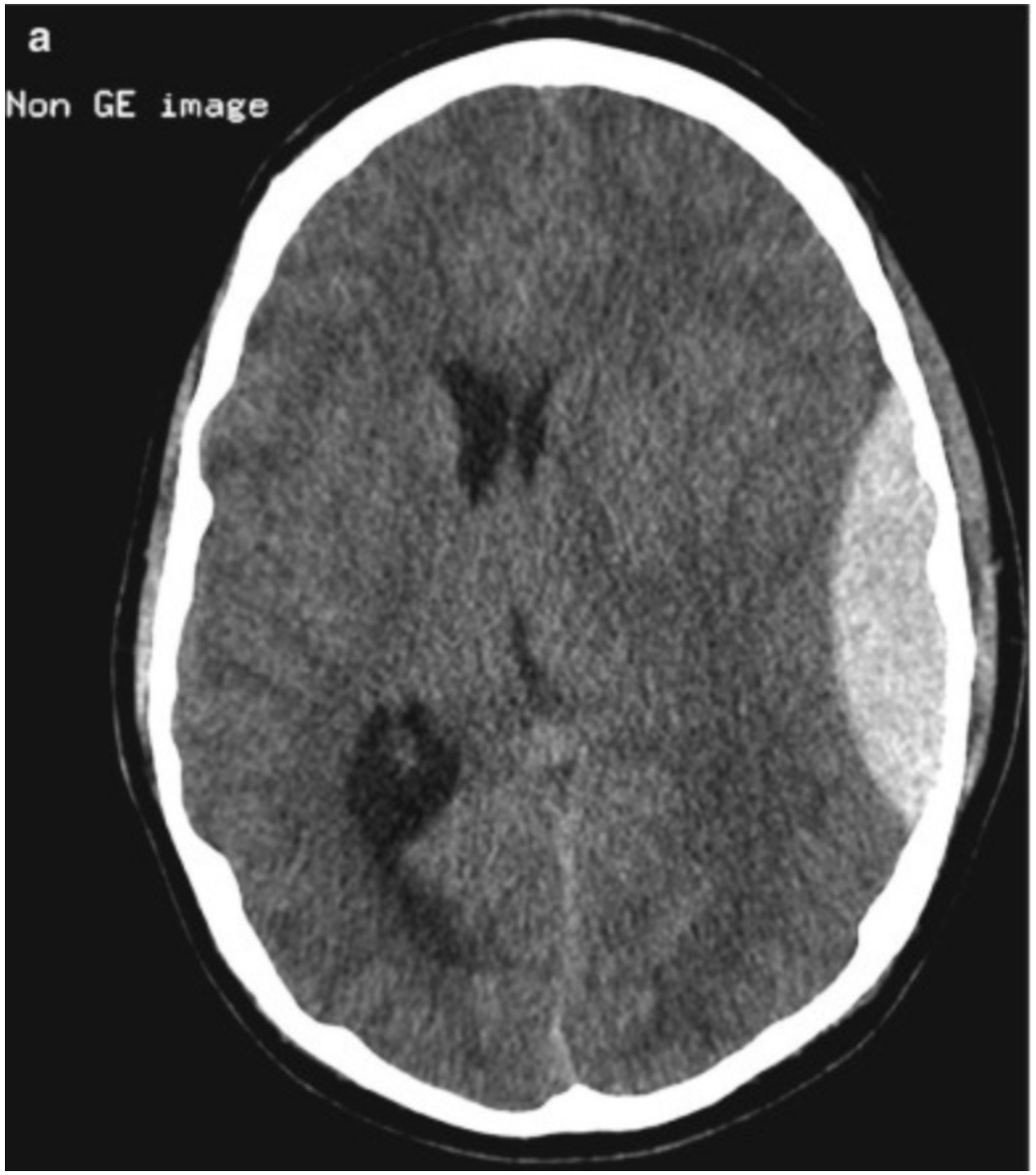
SDH

- Blood between the dura and the arachnoid space
- “contre coup”
- More common than EDH
- Injury to bridging veins
- Can cross suture lines
- “Crescent” shaped
- acute, acute on chronic, or chronic



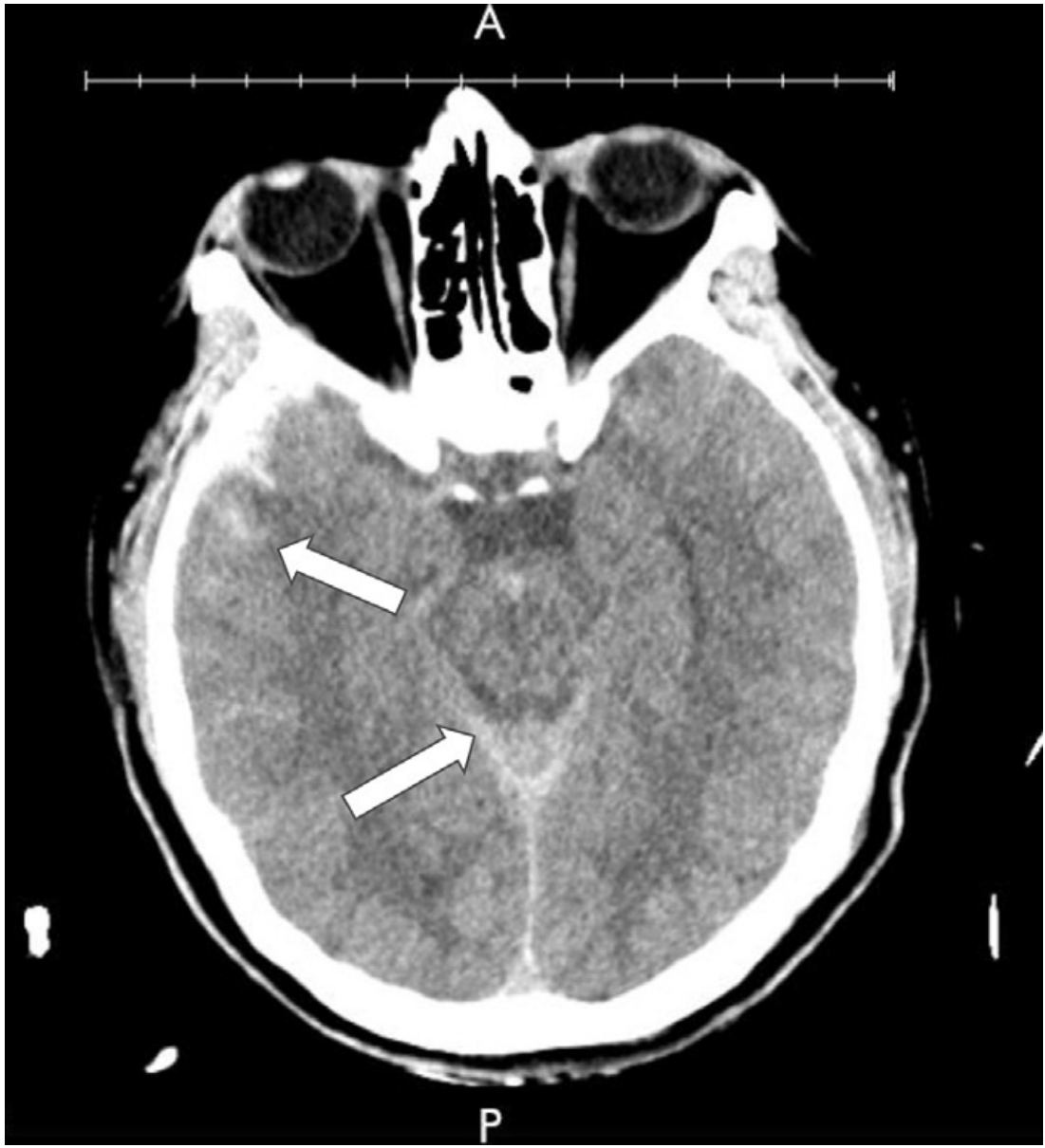
EDH

- Blood between skull and the dura
- "coup" side
- Commonly associated with skull fx
- Arterial injury >>> venous injury
  - Middle meningeal a.
- Do not cross suture lines
- "Lentiform" shaped
- + LOC → Lucid interval → decline



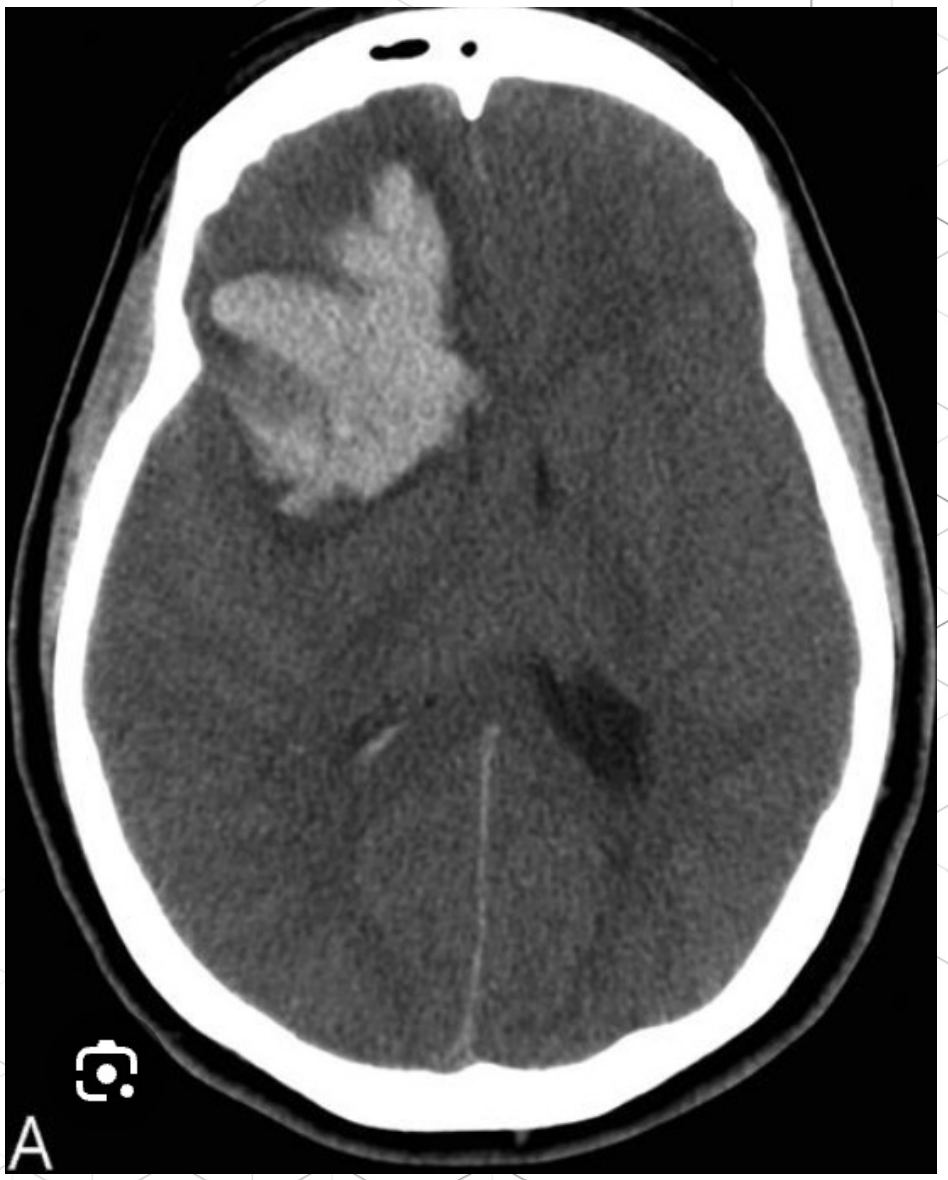
SAH

- Traumatic or non-traumatic
  - Most commonly aneurysmal rupture



IPH

- Multiple etiologies
  - AVM
  - HTN
  - Tumor
  - Aneurysm rupture
  - Trauma



Chapter 7 Traumatic Neuroemergency: Imaging Patients with Traumatic Brain Injury—An Introduction

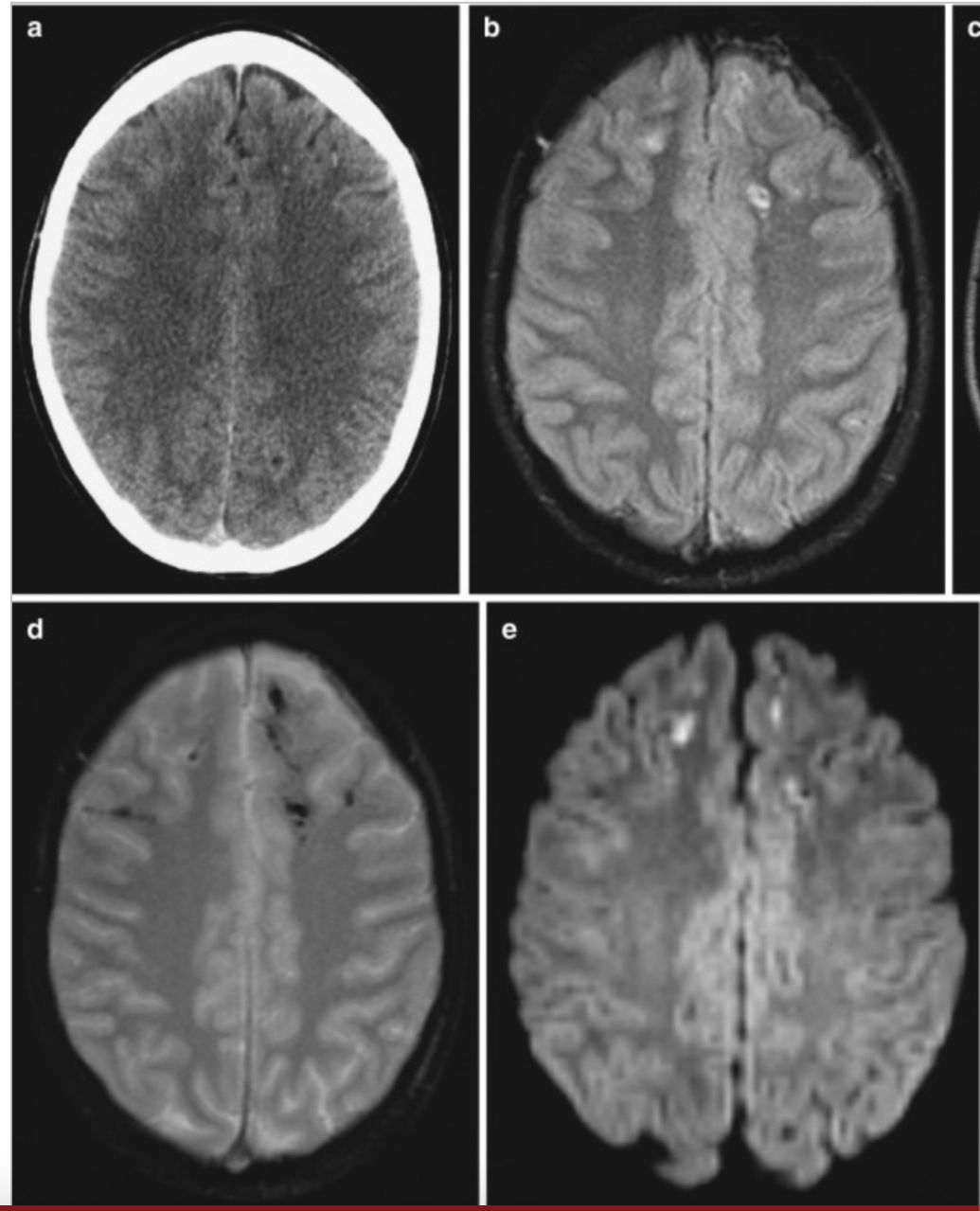
Paul M. Parizel and C. Douglas Philips.

DAI

- Neuronal injury → Shearing force
- MRI >>> CT

Grading of diffuse axonal injury

Grade I	Grade II	Grade III
Affects gray-white matter interface Frontal/ temporal > parietal/occipital	Involves frontal, temporal, parietal, occipital lobes, and corpus callosum	Includes damage to the brainstem as well as damage to structures mentioned in grade I and II



Chapter 7 Traumatic Neuroemergency: Imaging Patients with Traumatic Brain Injury—An Introduction

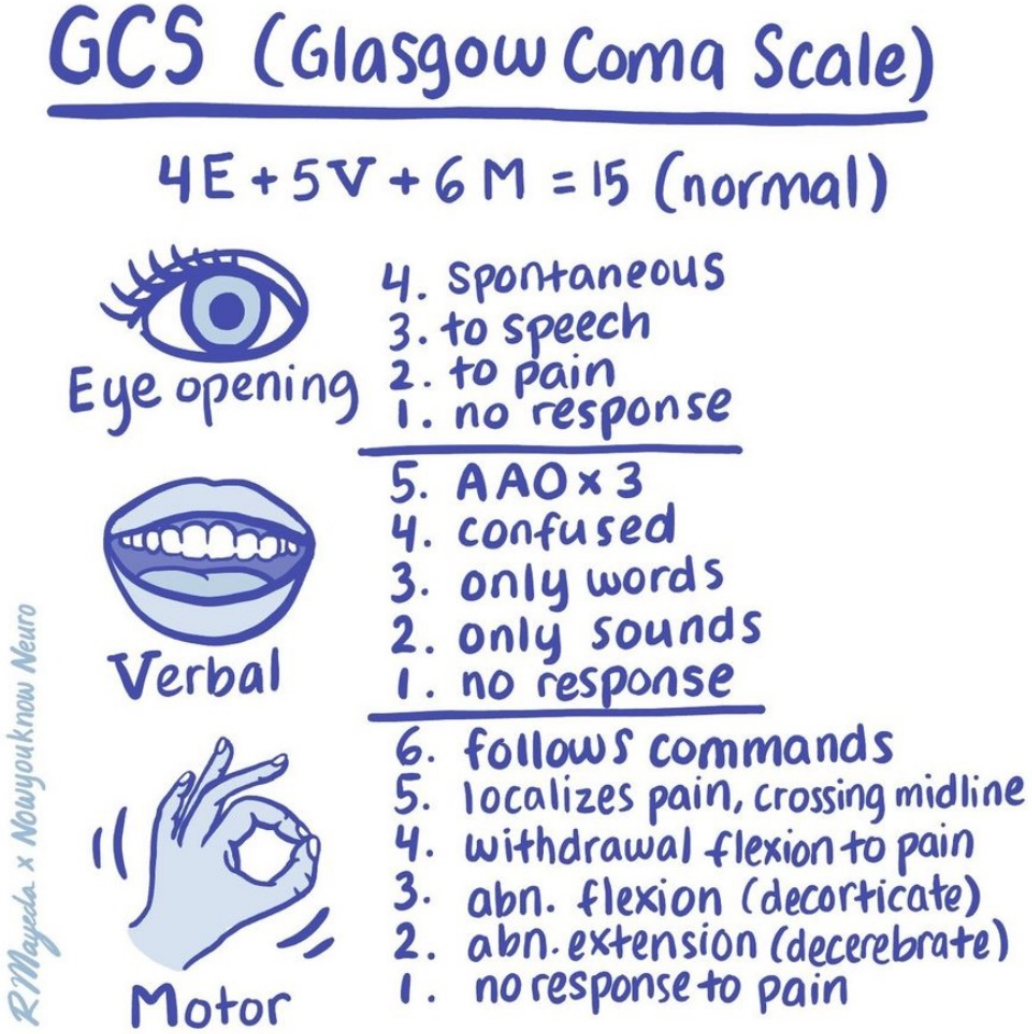
Paul M. Parizel and C. Douglas Philips.

Work-up of suspected TBI

- Primary Survey
  - A,B,C, E
  - D
    - **GCS, Pupils**
- Secondary Survey
- Imaging
  - **Who should get it?**

GCS

- Systematic method to assess the severity of neurotrauma
- Motor component has prognostic value
- Mild/Mod/Severe



The 20th century: the dawn of modern neurotrauma treatment

Stefana-Andrada Dobran [1] and Dafin Fior Muresanu [1, 2]

J Med Life. 2024 May; 17(5): 459–461.
doi: 10.25122/jml-2024-1008

Work–up of suspected TBI Nexus Head CT instrument

Evidence of significant skull fracture
e.g. periorbital or periauricular ecchymoses, hemotympanum, drainage of clear fluid from ears or nose, palpable step-off, stellate laceration (see Evidence for more detail)

No | Yes

Scalp hematoma
Injuries not involving calvarium (e.g. hematomas limited to the face/neck), are not considered scalp hematomas

No | Yes

Neurologic deficit
Any abnormal neurologic finding revealed by detailed exam (see Evidence for more detail)

No | Yes

Altered level of alertness
e.g. Glasgow Coma Scale (GCS) ≤14; delayed or inappropriate response to external stimuli; excessive somnolence; disorientation to person, place, time, or events; inability to remember three objects at 5 mins; perseverating speech

No | Yes

Abnormal behavior
Any inappropriate action, e.g. excessive agitation, inconsolability, refusal to cooperate, lack of affective response to questions or events, violent activity

No | Yes

Coagulopathy
Any clotting impairment, e.g. hemophilia, secondary to medications (Coumadin, heparin, aspirin, etc), hepatic insufficiency

No | Yes

Persistent vomiting
Recurrent (≥1 episode) projectile or forceful emesis, either observed or by history, after trauma

No | Yes

Age ≥65 years

No | Yes

Criteria Present	Risk of Significant Intracranial Injuries	Recommendation
0	Low	CT not necessary
>0	High	CT necessary

LITERATURE

ORIGINAL/PRIMARY REFERENCE

Mower WR, Hoffman JR, Herbert M, et al. Developing a decision instrument to guide computed tomographic imaging of blunt head injury patients. J Trauma. 2005;59(4):954-959.

VALIDATION

Mower WR, Gupta M, Rodriguez R, Hendey GW. Validation of the sensitivity of the National Emergency X-Radiography Utilization Study (Nexus) Head computed tomographic (Ct) decision instrument for selective imaging of blunt head injury patients: An observational study. PLoS Med. 2017;14(7):e1002313.

CT findings

- Type of bleed
- Skull fracture
- Size of bleed
  - Diameter, volume
- Midline shift
- Edema
- Herniation

Brain Injury Guidelines (BIG)

- 2014, single institution
- AAST validated (2022)
- Risk of progression
  - BIG 1= 0% (0%)
  - BIG 2= 2.6% (0%)
- Reduced CT, admission,

And NES consultation

	BIG 1	BIG 2	BIG 3
Neuro Exam (Abnormal = Best GCS <15 at time of classification, or focal deficit)	Normal	Normal	Abnormal
Intoxication (EtOH > 80 mg/dl, Suspicion of any non-EtOH substance abuse)	No	No/Yes	No/Yes
Coagulopathy [Pharmacological (anticoagulation, antiplatelets), Non-pharmacological (Abnormal TEG, cirrhosis, INR>1.4, thrombocytopenia <100)]	No	No	Yes
Skull fracture	No	Non-displaced	Displaced
Subdural Hematoma	≤4mm	5-7mm	≥8mm
Epidural Hematoma	≤4mm	5-7mm	≥8mm
Intraparenchymal Hemorrhage	≤4mm and 1 location	5-7mm and/or 2 locations	≥8mm and/or multiple locations
Subarachnoid Hemorrhage	"Trace" = ≤3 sulci	"Localized" =Single hemisphere	"Scattered" Bi-hemispheric
Intraventricular Hemorrhage	No	No	Yes
Midline Shift	No	No	Yes

All measurements/exact verbiage according to FINAL HCT

Patients must meet all criteria for categorization into BIG 1 or BIG 2. Failure to meet even 1 criterion (in BIG 1 or BIG 2) categorizes the patient into the BIG 3 category

Repeat HCT	No	No	Yes @6 Hours
Neurosurgical Consultation	No	No	Yes
Hospitalization	6 hour observation	Yes, 24 hour observation	Yes
Neurocheck Frequency	Q2	Q4	Q1 (until otherwise specified by NSG)
Discharge Criteria	GCS 15 (or baseline), Neuro Intact	GCS 15 (or baseline), Neuro Intact	NA
Discharge Criteria	Intact	Intact	NA

WTA 2013 PLENARY PAPER

The BIG (brain injury guidelines) project

Defining the management of traumatic brain injury by acute care surgeons

Joseph, Bellal MD; Friese, Randall S. MD; Sadoun, Moutamn MD; Aziz, Hassan MD; Kulvatunyou, Narong MD; Pandit, Viraj MD; Wynne, Julie MD; Tang, Andrew MD; O'Keeffe, Terence MB, ChB; Rhee, Peter MD

Author Information

Journal of Trauma and Acute Care Surgery 76(4):p 965-969, April 2014. | **DOI:** 10.1097/TA.0000000000000161

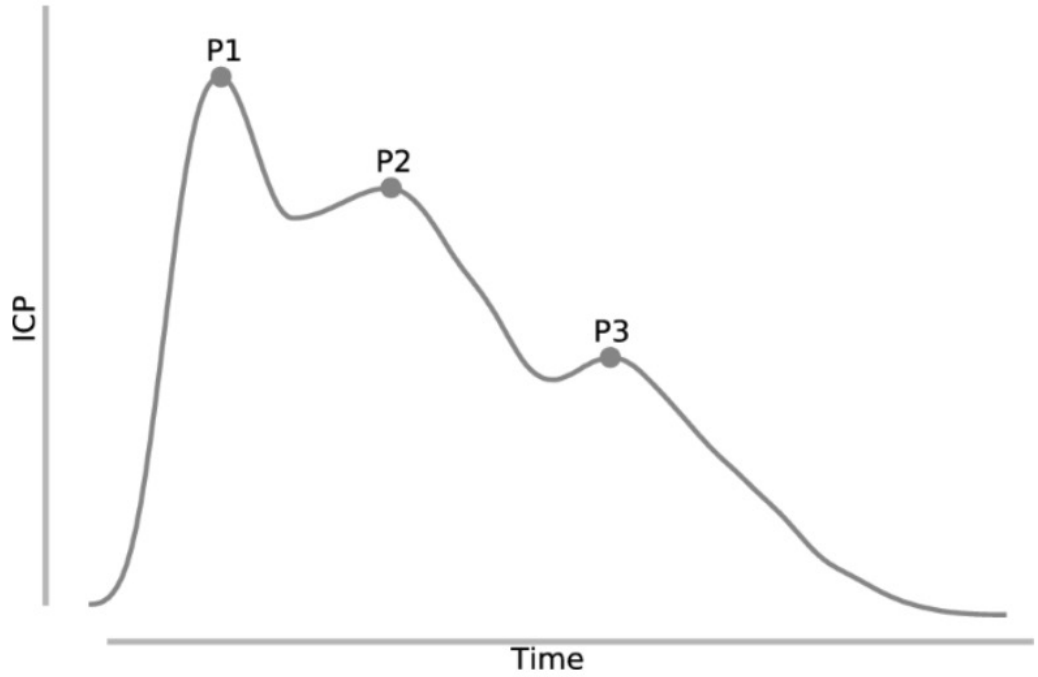
MEDICINE
OMA HEALTH SCIENCES

Intracranial pressure (ICP)

Image Credit: peterschreiber.media/Shutterstock.com

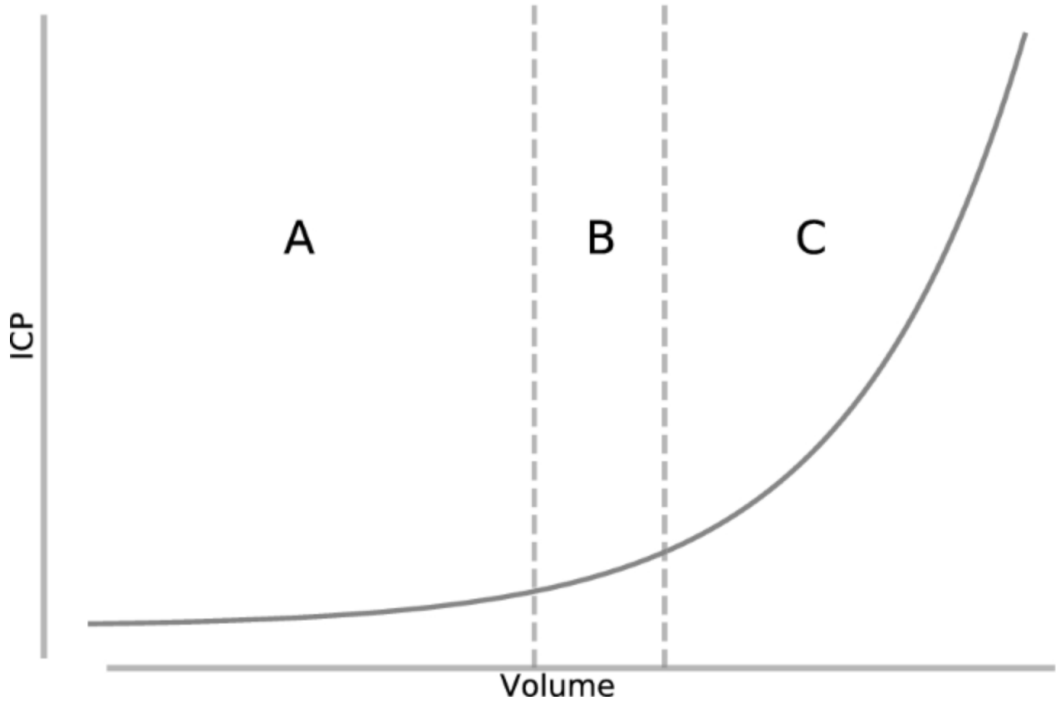
Intracranial Pressure

- Pressure inside the skull = CSF Pressure
- Pulsatile signal, driven by cardiac cycle (3 peaks)
- ICP = 7–15 mm HG
  - normal adults
- Goals in ICU < 22 mm HG

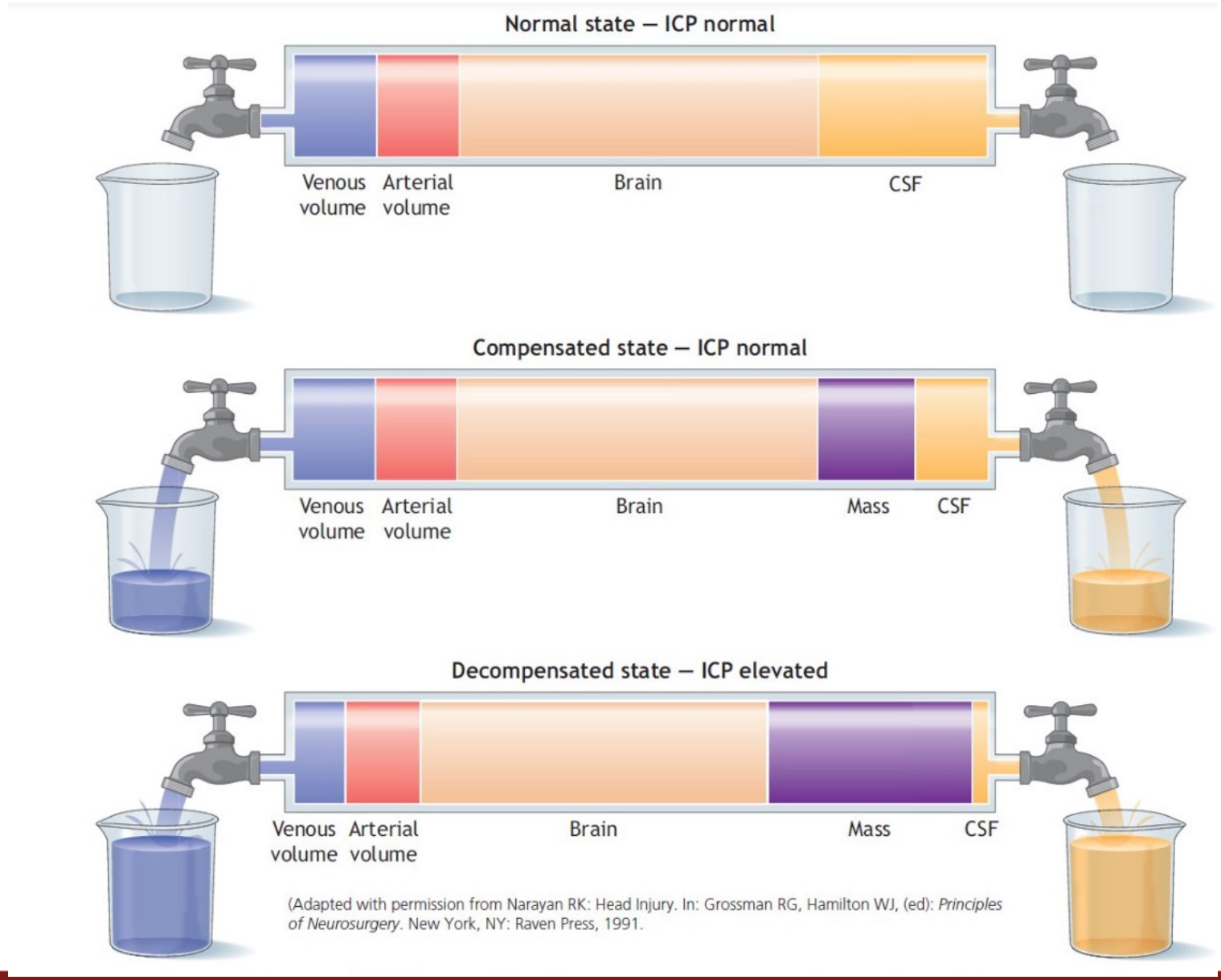


ICP

- 3 components of intra-cranial space = CSF, Blood, and Brain
- Enclosed in a rigid skull/closed system
- Pressure/Volume are related
  - Any change in volume → increases your pressure



ICP



(Adapted with permission from Narayan RK: Head Injury. In: Grossman RG, Hamilton WJ, (ed): *Principles of Neurosurgery*. New York, NY: Raven Press, 1991.

ICP

- Autoregulation
  - Vasodilation of arterioles (decrease CVR)
  - Increase arterial blood pressure

* All work to increase CBF → ultimately increase ICP too

$$CBF = \frac{MAP - ICP}{CVR}.$$

CBF= Cerebral blood flow
MAP= Mean arterial Pressure
ICP= Intracranial Pressure
CVR= Cerebrovascular resistance

ICP

- Simplified

$$CPP = MAP - ICP$$

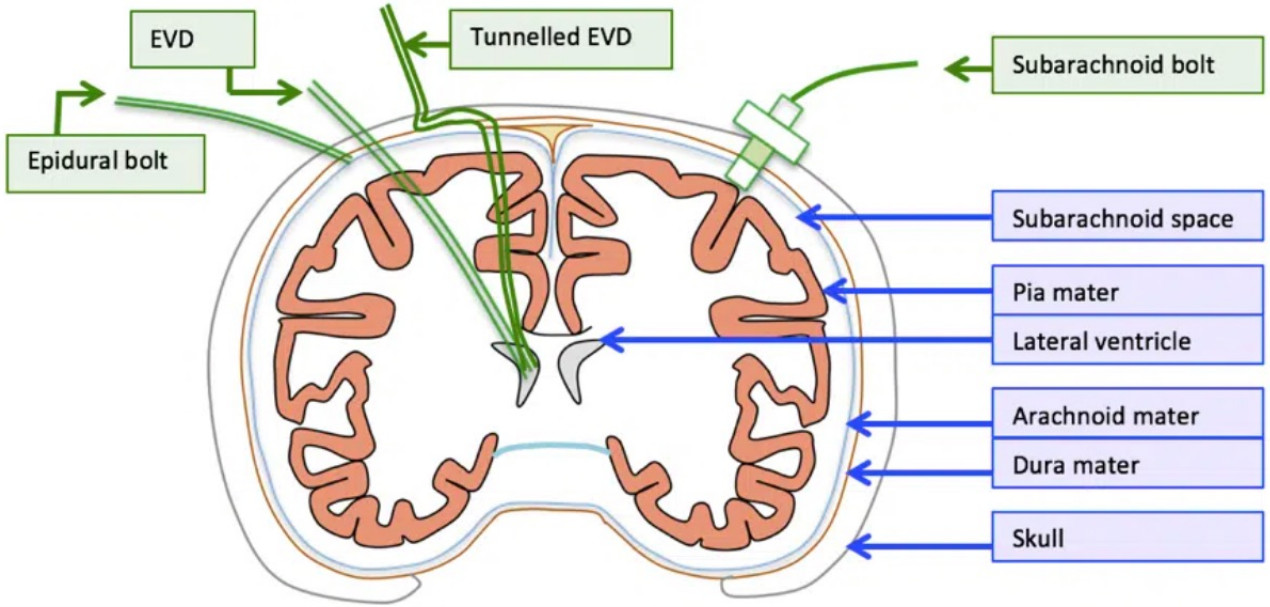
- Goal CPP 60–70

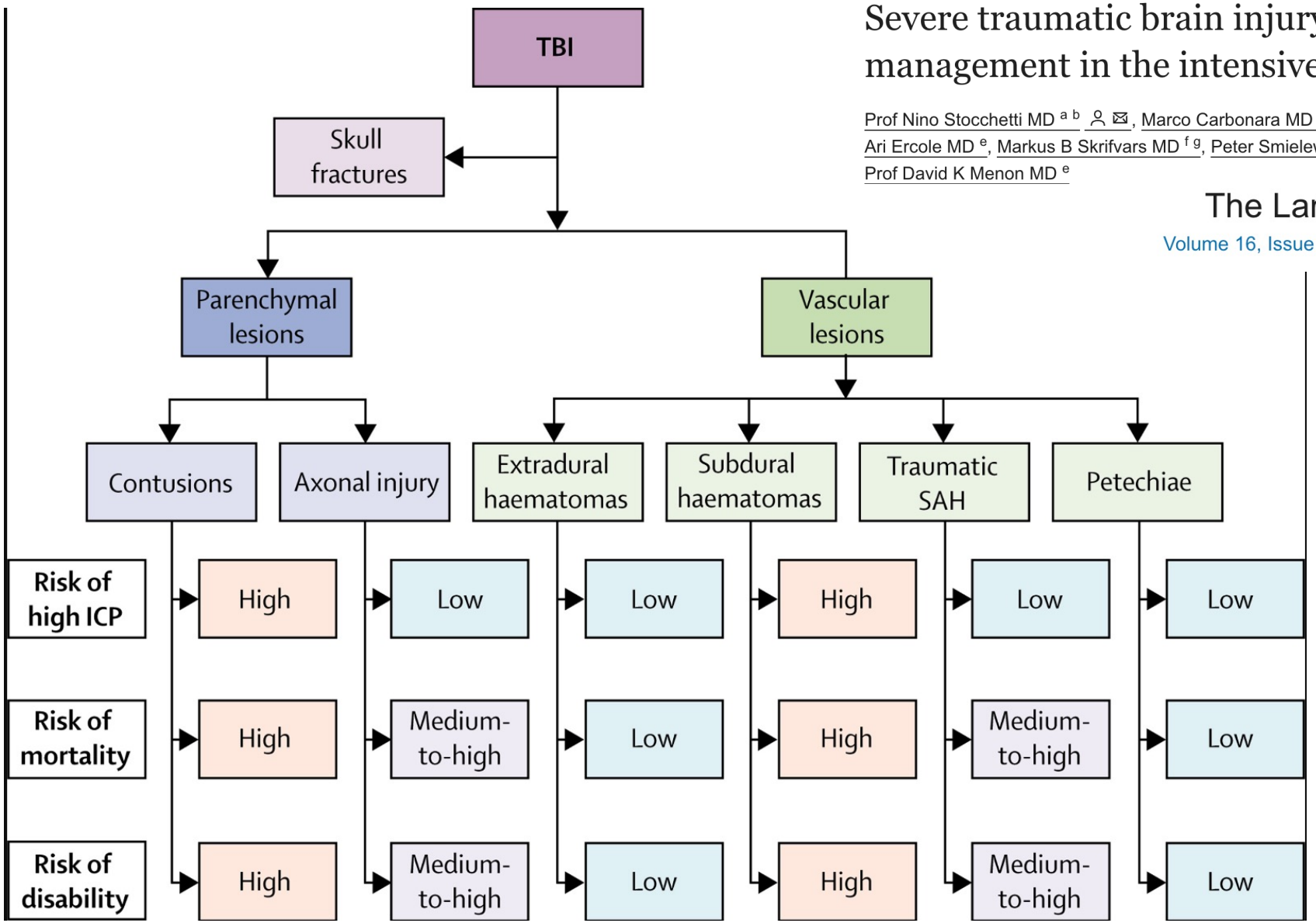
- Elevated ICP → Decreased CPP → Ischemia/Herniation → Disability or Death

- Elevated ICP → Increase mortality and poor outcomes

Ways to monitor ICP

- EVD
  - Catheter placed into the ventricle
  - Monitor and drain CSF
  - Gold standard *
- Fiberoptic probe = bolt
  - Varying locations
  - Only monitors ICP
  - Lower risk that EVD



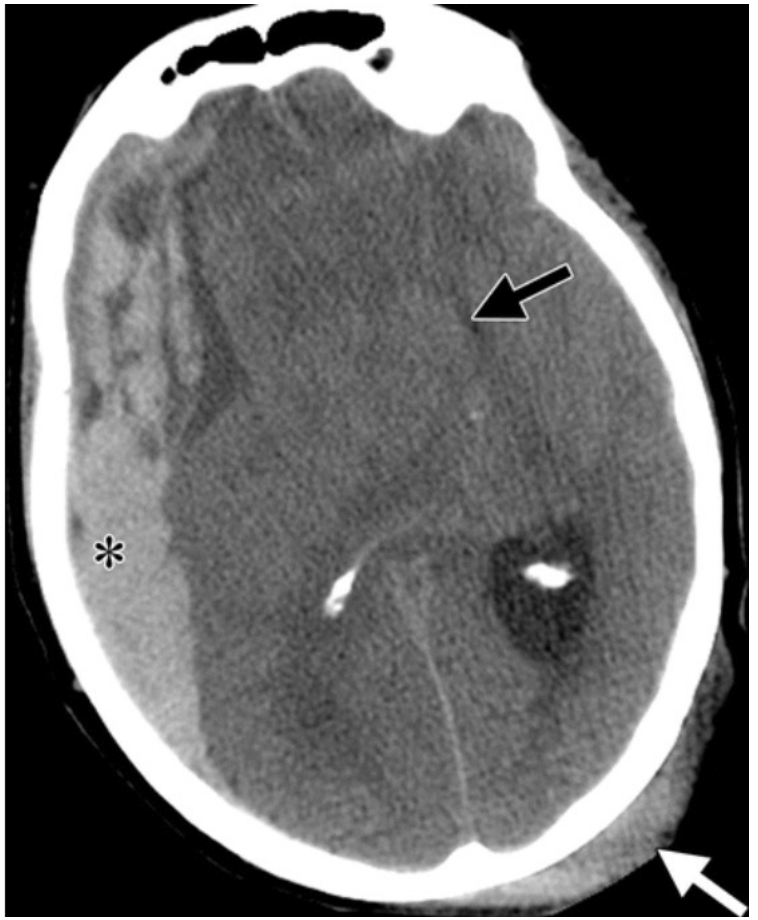


Severe traumatic brain injury: targeted management in the intensive care unit

Prof Nino Stocchetti MD [a b], Marco Carbonara MD [a], Giuseppe Citerio MD [c d], Ari Ercole MD [e], Markus B Skrifvars MD [f g], Peter Smielewski PhD [h], Tommaso Zoerle MD [a], Prof David K Menon MD [e]

The Lancet Neurology
Volume 16, Issue 6, June 2017, Pages 452-464

Special populations
Elderly patients



- Increased comorbidities
- Anticoagulation
- Poly-pharmacy → Increases falls
- Less brain reserve
- Baseline neurocognitive issues
- Increased risk of SDH
- Low velocity injuries (differ from younger patients)
- <u>Poorer outcomes – higher mortality</u>

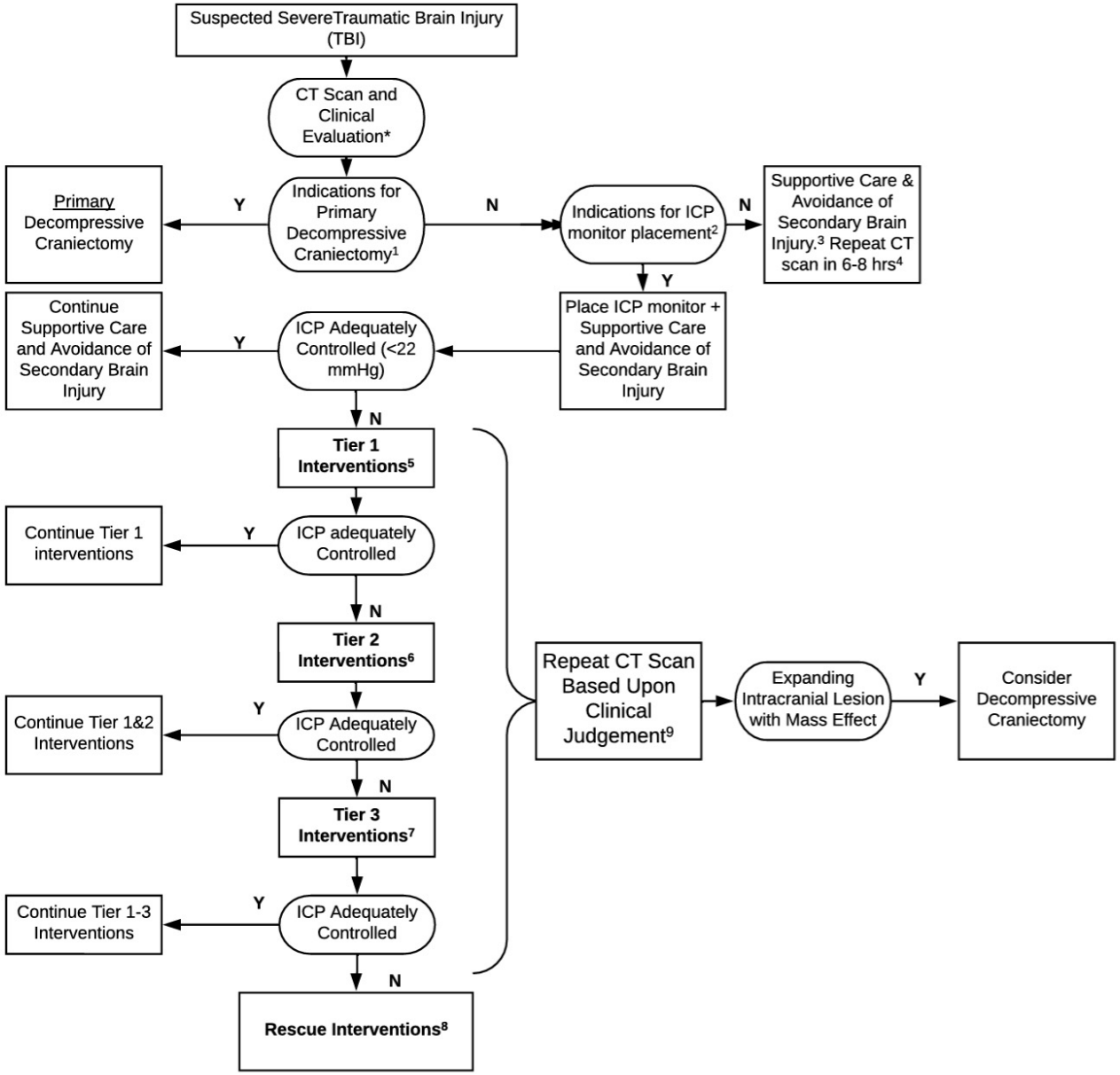
GOALS OF TREATMENT

Guidelines for TBI Management

- Brain Trauma Foundation
- Western Trauma Association

Many recommendations low-quality evidence

Goals of TBI Management



Goals of TBI Management
Supportive Care

Western Trauma Association

Standard trauma care	Control of hemorrhage	Correction of coagulopathy	Resuscitation, electrolyte management
Elevate HOB	Normotension	Sats > 90%	Normo-carbia
Avoid hyperpyrexia	Prevention of hyponatremia	Prevent hypoglycemia and treat hyperglycemia	Early nutrition
Prevention of infections	Sedation/Pain control	Seizure prophylaxis	

TBI management algorithm
Tier 1

Western Trauma Association

Additional sedation

Drainage of CSF

TBI management algorithm
Tier 2

Western Trauma Association

Hyperosmolar therapy

Paralysis

TBI management algorithm
Tier 3

Western Trauma Association

Barbituates?

TBI management algorithm
Rescue Strategies

Western Trauma Association

Decompressive Craniectomy

Experimental therapy

Additional therapies

Propranolol

Beta-Blocker Therapy in Severe Traumatic Brain Injury: A Prospective Randomised Controlled trial

Hosseinali Khalili. World J Surgery 2020; 44:1844-1853. https://doi.org/10.1007/s00268-020-05391-8

TXA

Effects of tranexamic acid on death, disability, vascular occlusive events and other morbidities in patients with acute traumatic brain injury (CRASH – 3): a randomised, placebo-controlled trial

The CRASH-3 trial collaborators. The Lancet, 2019; doi.org/10.1016/S1040-6736(19)32233-0

ICP monitors – who?

- Current guidelines– BTF (level 2 evidence)
  - GCS < 8
  - Abnormal head CT
  - Two of more: age >40 years, posturing, SBP < 90

ICP monitors – who?

- Current guidelines– BTF (level 3 evidence)
  - Progression on repeat CT scan
  - Brain swelling or absence of basal cisterns
  - Bifrontal contusions
  - Unable to follow neuro exam (pause sedation)

ICP monitors – complications

- Bleeding
- Infection
- Technical
- Over-drainage
- False readings

ICP monitors – Do we need them?

- What we know-- Elevated ICPs → lead to worse outcomes
  - We can treat elevated ICPs

- What we need to know -- Does treating elevated ICPs improve outcomes?

* Some Experts equate ICP monitors to PA catheters

September 27, 2023

Intracranial Pressure Monitoring in Traumatic Brain Injury—A Tool of the Trade or One That Betrays Us?

Ruchira M. Jha, MD, MSc[1]

ICP monitors – Do we need them?

- BEST-TRIP (S. American trial)
  - RCT
  - No difference in outcomes if ICP monitors were used versus clinical exam/repeat CT scans
  - No change in rates of craniectomy
  - Increased vent strategies, hyperosmolar therapy, and barbituates

A Trial of Intracranial-Pressure Monitoring in Traumatic Brain Injury

This article has been corrected. VIEW THE CORRECTION

Authors: Randall M. Chesnut, M.D., Nancy Temkin, Ph.D., Nancy Carney, Ph.D., Sureyya Dikmen, Ph.D., Carlos Rondina, M.D., Walter Videtta, M.D., Gustavo Petroni, M.D., +8, for the Global Neurotrauma Research Group* Author Info & Affiliations

Published December 27, 2012 | N Engl J Med 2012;367:2471-2481 | DOI: 10.1056/NEJMoa1207363

ICP monitors – the FUTURE

- Noninvasive methods

Craniectomy – who?

- Considered for refractory elevated ICPs
- SDH
  - Diameter > 10 mm, Shift > 5mm (any GCS)
  - Diameter > 10 mm, Shift < 5mm, AND Worsening GCS
- EDH
  - > 30 cm^3 (any GCS)
- IPH
  - GCS 6–8, midline shift > 5mm and > 20 cm^3
  - Controversial *
- Rarely – SAH, DAI

Craniectomy – who?

- RESCUE-ICP
  - MCT, RCT
  - Severe TBI, refractory elevated ICP
  - Decreased mortality
  - Increased survival to vegetative state
  - No difference in time to death or discharge between groups

Trial of Decompressive Craniectomy for Traumatic Intracranial Hypertension

Authors: Peter J. Hutchinson, Ph.D., F.R.C.S. (SN), Angelos G. Kolias, Ph.D., M.R.C.S., Ivan S. Timofeev, Ph.D., F.R.C.S. (SN), Elizabeth A. Corteen, M.Sc., Marek Czosnyka, Ph.D., Jake Timothy, M.D., F.R.C.S. (SN), Ian Anderson, F.R.C.S. (SN) , +16 , for the RESCUEicp Trial Collaborators* Author Info & Affiliations

Published September 22, 2016 | N Engl J Med 2016;375:1119-1130 | DOI: 10.1056/NEJMoa1605215

Craniectomy – who?

- DECRA
  - MCT, RCT
  - Similar mortality
  - Increased "unfavorable outcomes"
  - Decreased ICU LOS
  - No difference in HLOS
  - Decrease elevated ICP

Decompressive Craniectomy in Diffuse Traumatic Brain Injury

Authors: D. James Cooper, M.D., Jeffrey V. Rosenfeld, M.D., Lynnette Murray, B.App.Sci., Yaseen M. Arabi, M.D., Andrew R. Davies, M.B., B.S., Paul D'Urso, Ph.D., Thomas Kossmann, M.D., Jennie Ponsford, Ph.D., Ian Seppelt, M.B., B.S., Peter Reilly, M.D., and Rory Wolfe, Ph.D., for the DECRA Trial Investigators and the Australian and New Zealand Intensive Care Society Clinical Trials Group* Author Info & Affiliations

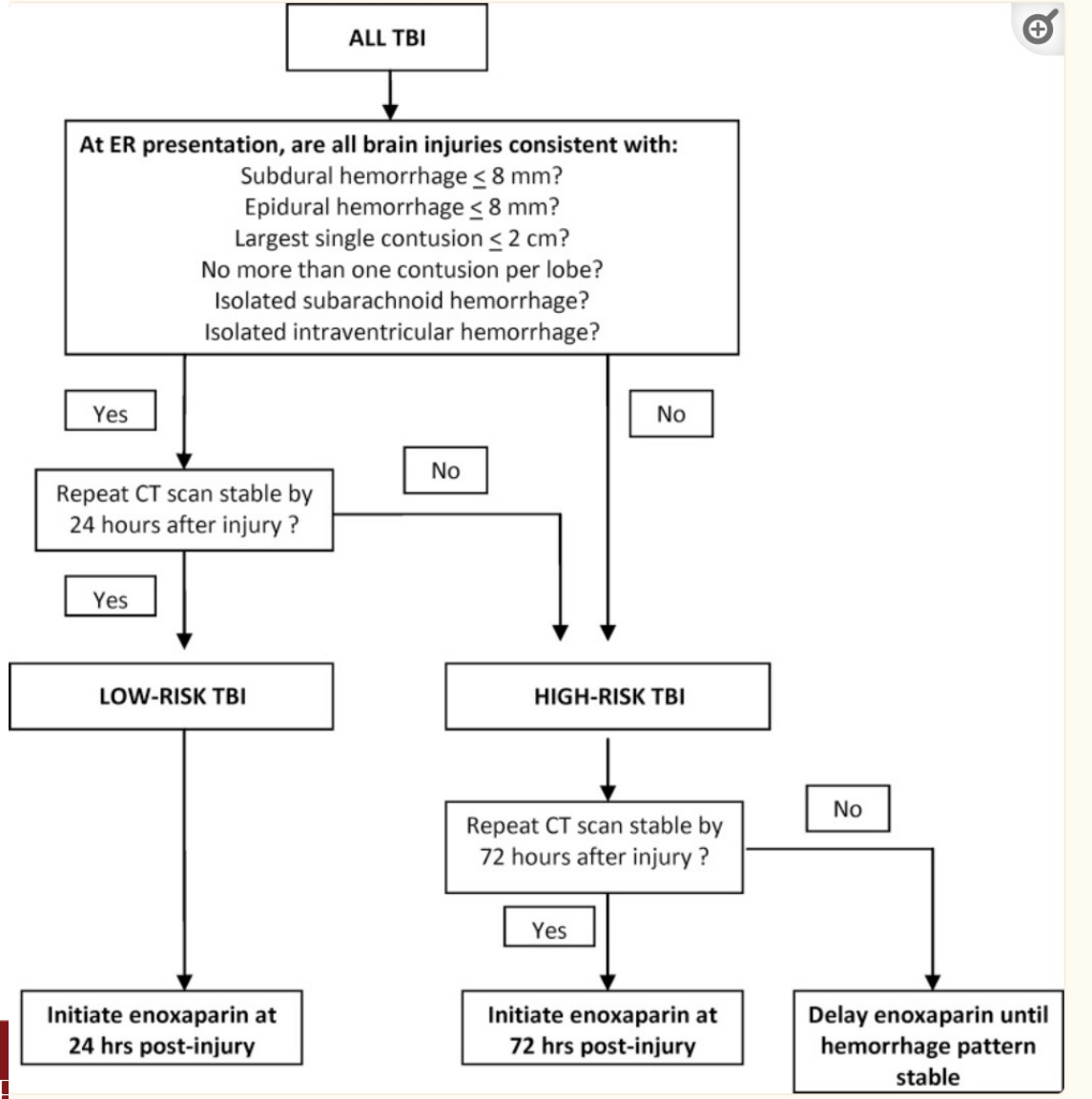
Published April 21, 2011 | N Engl J Med 2011;364:1493-1502 | DOI: 10.1056/NEJMoa1102077 | VOL. 364 NO. 16

Craniectomy – who?

- Sometimes we have good outcomes
- Patients who get craniectomy for refractory ICPs – may not have good outcomes

When to anti-coagulate?

Parkland



J Neurotrauma. 2014 Oct 15; 31(20): 1737–1743.
doi: 10.1089/neu.2014.3366
PMCID: PMC4180120
PMID: 24945196

The Parkland Protocol's Modified Berne-Norwood Criteria Predict Two Tiers of Risk for Traumatic Brain Injury Progression

Rachel A. Pastorek,[1] Michael W. Cripps,[2] Ira H. Bernstein,[3] William W. Scott,[4] Christopher J. Madden,[4] Kim L. Rickert,[4] Steven E. Wolf,[2] and Herb A. Phelan[#2]

Anticoagulation – reversal
OUH Trauma Clinical guideline

- All patients on therapeutic anticoagulation should be considered for reversal in the setting of life-threatening bleeding

Anticoagulation – reversal
OUH Trauma Clinical guideline

Oral Anticoagulants		
Vitamin K Antagonist	**Reversal**	**Monitoring**
1. Warfarin 2. Tecarfarin	First Line: Plasma Prothrombin Complex Concentrate (PCC) • See chart below (pg.2) • **Target:** INR <1.6 within 4 hours of arrival 1. Stop Warfarin 2. Vitamin K PO or IV 3. 4 Factor PCC (KCentra) from pharmacy Second Line: • Fresh Frozen Plasma (low in Factor IX) **consideration only, if need volume** variable effects, slower reversal	☐ INR obtained 30m after infusion/reversal • Consider TEG

Anticoagulation – reversal
OUH Trauma Clinical guideline

Warfarin Reversal Chart		
INR > 1.5	Clinical Scenario	Management
CNS Bleeding	Stable	Stop Warfarin until INR therapeutic
	Rapid Reversal Needed	Stop Warfarin Vitamin K 10 mg Oral or 10 mg IV not to exceed 1 mg/min. X1 4 Factor PCC (Kcentra): 1500U IVP x1 If use FFP 10 – 15 ml/kg
No CNS Bleeding	Stable	Stop Warfarin until INR therapeutic
	Rapid Reversal Needed	Stop Warfarin Vitamin K 10 mg oral or 10 mg IV not to exceed 1 mg/min. X1 4 Factor PCC: 1000U IVP x1 If using FFP 15-30 ml/kg

Anticoagulation – reversal
OUH Trauma Clinical guideline

Oral Anticoagulants		
Direct Thrombin Inhibitor	**Reversal**	**Monitoring**
1. Dabigtran (Pradaxa)	First Line: • Idaracuizamab (Praxbind) from pharmacy • Dose: 2.5g IV bolus x2 (total dose 5g) • No more than 15 minutes apart for medication administration Second line: FEIBA or rFVIIa (from pharmacy) □ FEIBA 50 units/kg IV (maximum dose 5000 units)	□ Qualitative-aPTT, thrombin time obtained 30m post-infusion

Anticoagulation – reversal
OUH Trauma Clinical guideline

Factor Xa Inhibitors-Direct	Reversal	Monitoring
1. Ribaroxaban (Xarelto)) 2. Apixaban (Eliquis) 3. Edoxaban (Lixiana, Sayvasa) 4. Betrixaban 5. Darexaban	CNS Bleed: • 4 – Factor PCC (KCentra): 25-50U/kg IVP x1 (maximum dose 5000units) No CNS Bleed: ○ 4-Factor PCC : 2000U IVP x1 • If 4F-PPC not available: □ aPCC (FEIBA) 50 units/kg IV (maximum dose 5000 units) □ Contact pharmacy to discuss use of coagulation factor Xa (recombinant), inactivated-zhzo (*Andexxa)* • Nephrology Consult	□ Anti - Xa Assay (obtain prior to reversal attempts, and 30 minutes after administration)

Anticoagulation – reversal
OUH Trauma Clinical guideline

Antiplatelet Agents	Reversal	Monitoring
1. ASA 325 mg	▯ DDAVP 0.3mcg/kg IV x1 ▯ Platelet transfusion	▯ TEG w/ Platelet Mapping ▯ If DDAVP/platelets transfused, recheck TEG w/ Platelet Mapping after each intervention If platelet dysfunction is suspected, transfuse platelets and consider cryoprecipitate
2. Adenosine diphosphate receptor antagonist: a. Clopidogrel (Plavix) b. Ticlopidine (Ticlid) c. Prasugrel (Effient) d. Ticagrelor (Brilinta) e. Dipyridamole (Persantine/Aggr enox)	Standard: 1. Platelet transfusion Consider: 1. Severe cases of ICH: If platelet count < 100k or decreased MA on TEG w/ Platelet Mapping 2. Cryoprecipitate (1 unit) Consider addition in patient with platelet dysfunction (Renal failure, etc.)	

Prognostication

- TRACK–TBI
- Largest prospective observational study
- GCS/DRS assessed @ 2 weeks, 3/6/12 months
- Ca

Functional Outcomes Over the First Year After Moderate to Severe Traumatic Brain Injury in the Prospective, Longitudinal TRACK-TBI Study

Michael A. McCrea, PhD[1]; Joseph T. Giacino, PhD[2,3,4]; Jason Barber, MS[5]; et al

Figure 2. Glasgow Outcome Scale-Extended (GOSE) Total Score Distribution for Patients With Moderate Traumatic Brain Injury at 2 Weeks and 3, 6, and 12 Months Postinjury

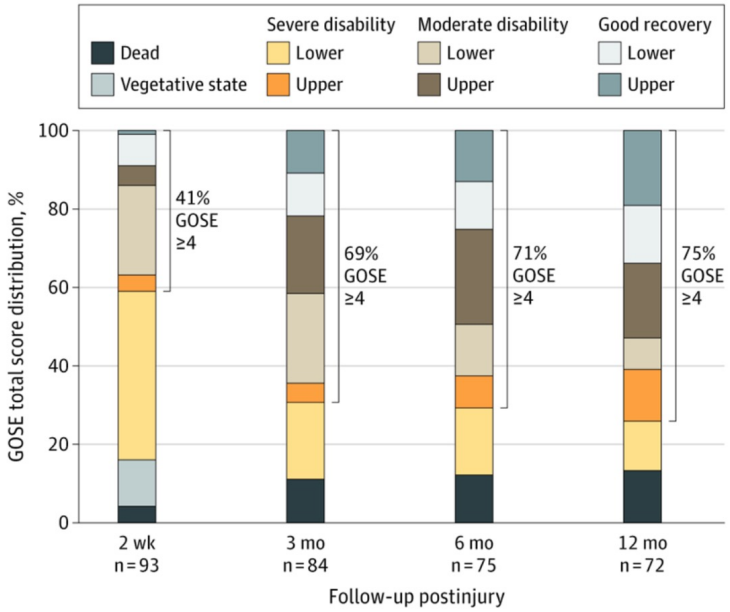
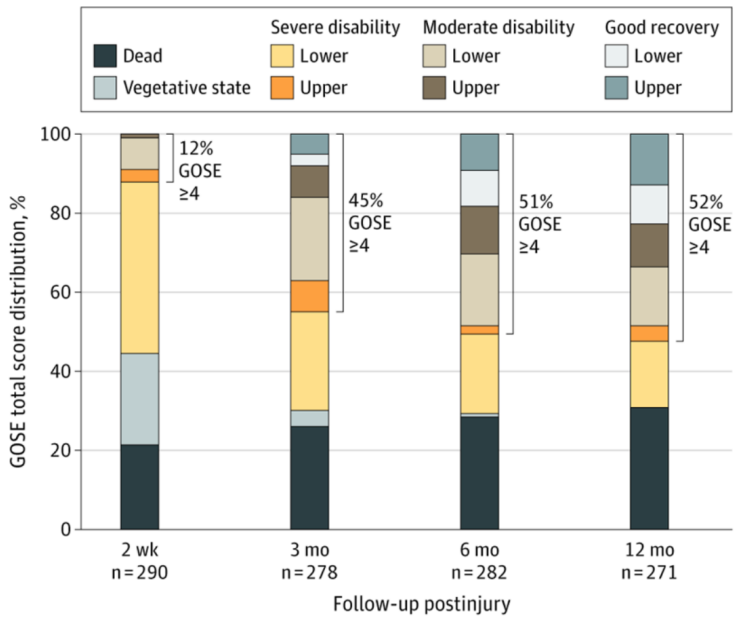


Figure 1. Glasgow Outcome Scale-Extended (GOSE) Total Score Distribution for Patients With Severe Traumatic Brain Injury at 2 Weeks and 3, 6, and 12 Months Postinjury



Prognostication

Functional Outcomes Over the First Year After Moderate to Severe Traumatic Brain Injury in the Prospective, Longitudinal TRACK-TBI Study

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- Careful making early, definitive statements

Take Home Points

- TBI – most common cause of death/disability following injury
- Standard approach to assessing patients in ED
- Consider BIG guidelines
- Algorithm for TBI management (vary and institutional)
- Communication between ICU and NES team critical
- ICP monitors/Craniectomy remains controversial
- Anti–coagulate per Parkland
- Reverse anticoagulation for life threatening bleeding
- Careful with early prognostication

QUESTIONS?