Management Acute Brain Injury

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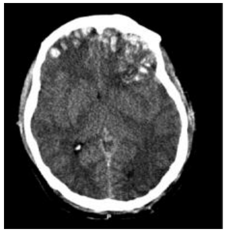
Disclosures

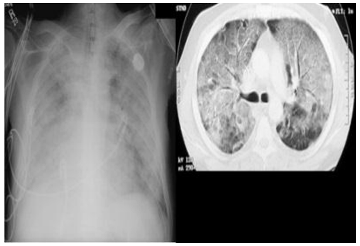
I have no relevant financial interests to disclose

Case

- 46-year-old male (70 kg IBW) with no significant PMH transferred to the ED from OSH post motor vehicle injury
- Prior to arrival by helicopter:
  - GCS 8 (E2,V2,M4), intubated without complications



- Trauma work-up extensive, including CT brain
- Ventilator Settings:
  - AC/VC mode, VT 650 mL, PEEP 5 cm H_2O, FiO_2 0.50, peak IP 29 cm H_20 and Insp. plateau pressures 25 cm H_2O



- Chest X-ray and CT
  - Aspiration pneumonia, bilateral alveolar infiltrates
- ED Course:
  - airway pressure and decreased O_2 saturation on FiO_2 1.0B
  - BP 103/76, HR 120

Case

<u>What are the issues?</u>

<u>What are the treatment options?</u>

Basic Brain Injury Facts

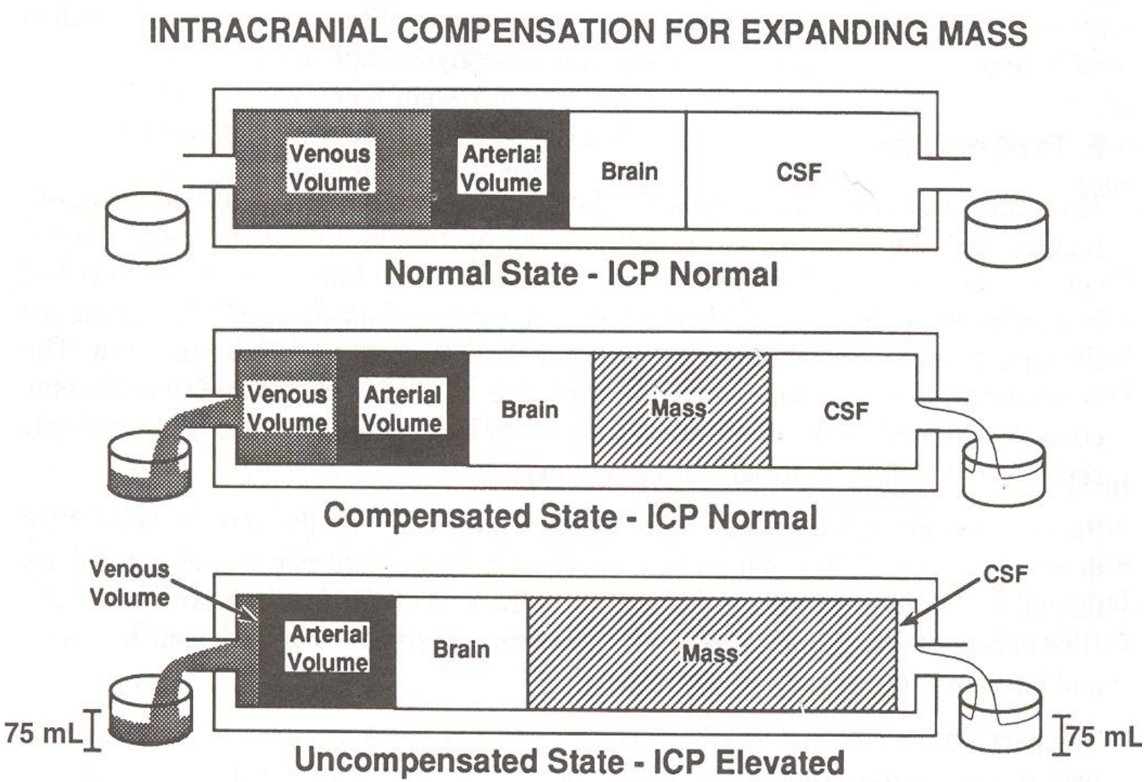
- **Intracranial pressure (ICP)** is the pressure inside the skull – brain tissue and CSF
  - Measured in millimeters of mercury (mmHg)
  - Normally 7-15 mmHg in supine adult
- **Cerebral perfusion pressure (CPP)** is the pressure of the blood flowing in the brain
  - **CPP = MAP – ICP**
- **Intracranial hypertension (IH)**
  - When ICP > 20 mmHg
  - Increased ICP can cause ischemia by decreasing CPP

Normal Values

CPP	70-90 mmHg
MAP	60-150 mmHg
ICP	10 mmHg

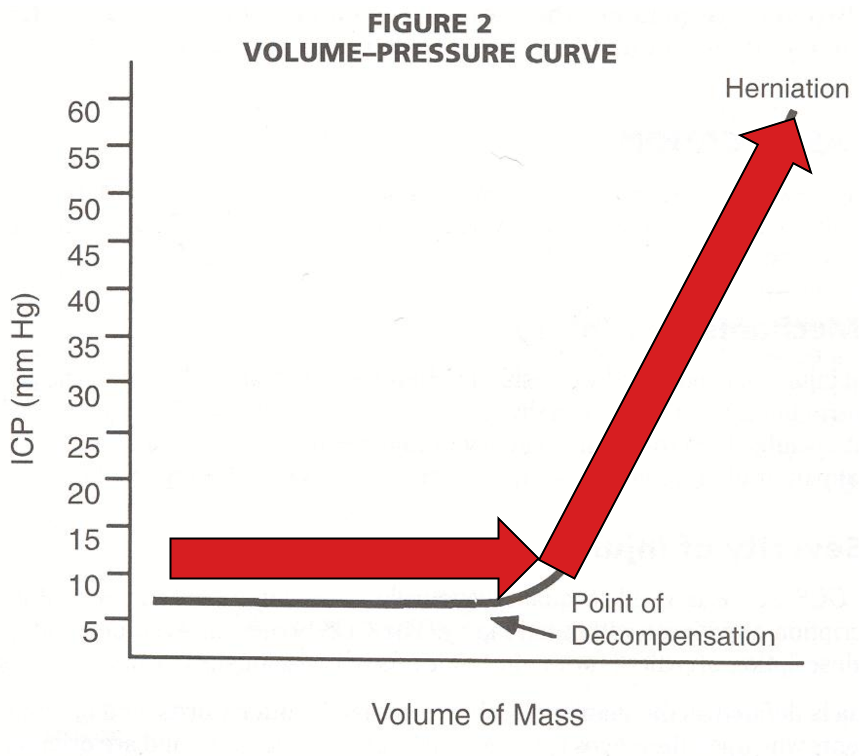
Basic Brain Injury Facts

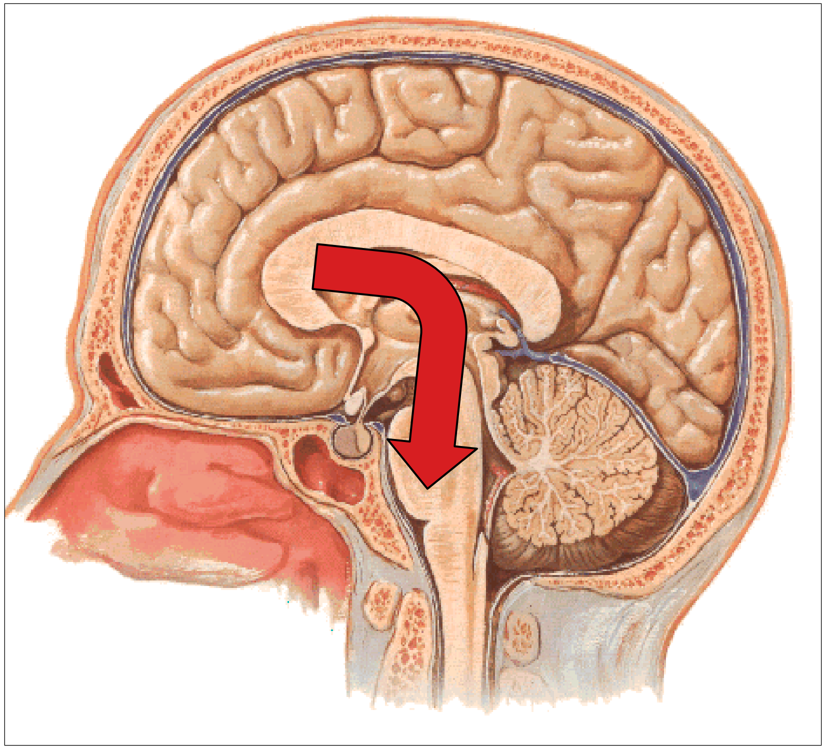
- **Monro-Kellie doctrine**
  - Pressure volume relationship between ICP, volume of CSF, blood, brain tissue and CPP
  - Increase in one must be compensated by decrease of another



Basic Brain Injury Facts

Volume-Pressure Curve





Brain Injury

General Principles

- Stabilize the patient
- Prevent intracranial hypertension
- Maintain stable CPP
  - Raise HOB 30-45° reduces ICP and improves CPP
- Avoid secondary brain insults (SBI)
- Optimize cerebral hemodynamics and O2
- Goal of euvolemia or mild hypervolemia

Brain Injury and Fluid Management

Fluid Principles

- Treat hypotension (e.g. hemorrhage)
  - Isotonic crystalloids (NS) for volume expansion
  - Avoid dextrose and hypotonic solutions
  - Vasopressors if needed
  - Blood products if significant blood loss
  - CVP may be utilized to guide fluid management
    - Goal 8-10 mm Hg

Brain Injury and Fluid Management

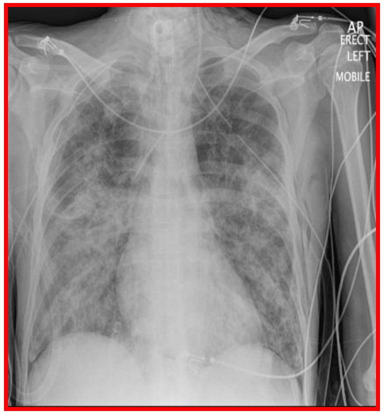
Fluid Principles

- Hypertonic Therapy (decreases ICP)
  - **Mannitol** (0.25-1 g/kg IV over 15-20 min)
    - Reserved only for signs of herniation
    - Several electrolyte complications
  - **Hypertonic Saline** (3%, 5%, 23.4% solution)
    - Benefits: Expands IVF, extracts water from the intracellular space, decreases ICP, increases cardiac contractility
    - Produces osmotic dehydration and viscosity-related cerebral vasoconstriction

What's Happening in THIS Patient?

<u>Pulmonary Insult</u>:

- **Acute Respiratory Distress Syndrome** (ARDS) clinically devastating syndrome affecting patients
- Characterized by <u>inflammation of the lung parenchyma leading to impaired gas exchange resulting in hypoxemia and abnormal lung function</u>
- Great advances in understanding pathogenesis of disease but mortality still high
- Long ICU stays and hospitalization
- Require prolonged rehabilitation
- Acute Lung Injury (ALI) milder form (older term)



Acute Respiratory Distress Syndrome
The Berlin Definition

"An acute, diffuse, inflammatory lung injury that leads to increased pulmonary vascular permeability, increased lung weight, and a loss of aerated tissue"

Table 3. The Berlin Definition of Acute Respiratory Distress Syndrome

	Acute Respiratory Distress Syndrome
Timing	Within 1 week of a known clinical insult or new or worsening respiratory symptoms
Chest imaging[a]	Bilateral opacities—not fully explained by effusions, lobar/lung collapse, or nodules
Origin of edema	Respiratory failure not fully explained by cardiac failure or fluid overload Need objective assessment (eg, echocardiography) to exclude hydrostatic edema if no risk factor present
Oxygenation[b]	
Mild	200 mm Hg < PaO_2/FIO_2 ≤ 300 mm Hg with PEEP or CPAP ≥5 cm H_2O[c]
Moderate	100 mm Hg < PaO_2/FIO_2 ≤ 200 mm Hg with PEEP ≥5 cm H_2O
Severe	PaO_2/FIO_2 ≤ 100 mm Hg with PEEP ≥5 cm H_2O

Abbreviations: CPAP, continuous positive airway pressure; FIO_2, fraction of inspired oxygen; PaO_2, partial pressure of arterial oxygen; PEEP, positive end-expiratory pressure.
[a] Chest radiograph or computed tomography scan.
[b] If altitude is higher than 1000 m, the correction factor should be calculated as follows: [PaO_2/FIO_2 × (barometric pressure/760)].
[c] This may be delivered noninvasively in the mild acute respiratory distress syndrome group.

JAMA. 2012; 307 (23): 2526-2533

Incidence and Outcomes of Acute Lung Injury

Common

Deadly

Expensive

Table 1. Incidence of Acute Lung Injury and ARDS and Mortality from These Conditions.*

Variable	Acute Lung Injury	ARDS
Cases — no.	1,113	828
Crude incidence — no. per 100,000 person-yr	78.9	58.7
Age-adjusted incidence — no. per 100,000 person-yr†	86.2	64.0
Mortality (95% CI) — %	38.5 (34.9–42.2)	41.1 (36.7–45.4)
Estimated annual cases — no.†	190,600	141,500
Estimated annual deaths — no.†	74,500	59,000
Estimated annual hospital days — no.†	3,622,000	2,746,000
Estimated annual days in ICU — no.†	2,154,000	1,642,000

Rubenfeld et al. N Engl J Med 2005; 353: 1685-93.

ARDS/ALI Epidemiology:

CLINICAL DISORDERS ASSOCIATED WITH THE DEVELOPMENT OF ALI/ARDS

Direct Insult

Common

- Aspiration Pneumonia
- Pneumonia

Less Common

- Inhalation Injury
- Pulmonary Contusions
- Fat Emboli
- Near drowning
- Reperfusion Injury

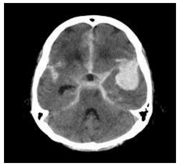
Indirect Insult

Common

- Sepsis
- Severe Trauma
- Shock

Less Common

- Acute Pancreatitis
- Cardiopulmonary bypass
- DIC
- Burns
- **Head Injury**
- Drug overdose
- Transfusion-related TRALI



ARDS/ALI: Epidemiology

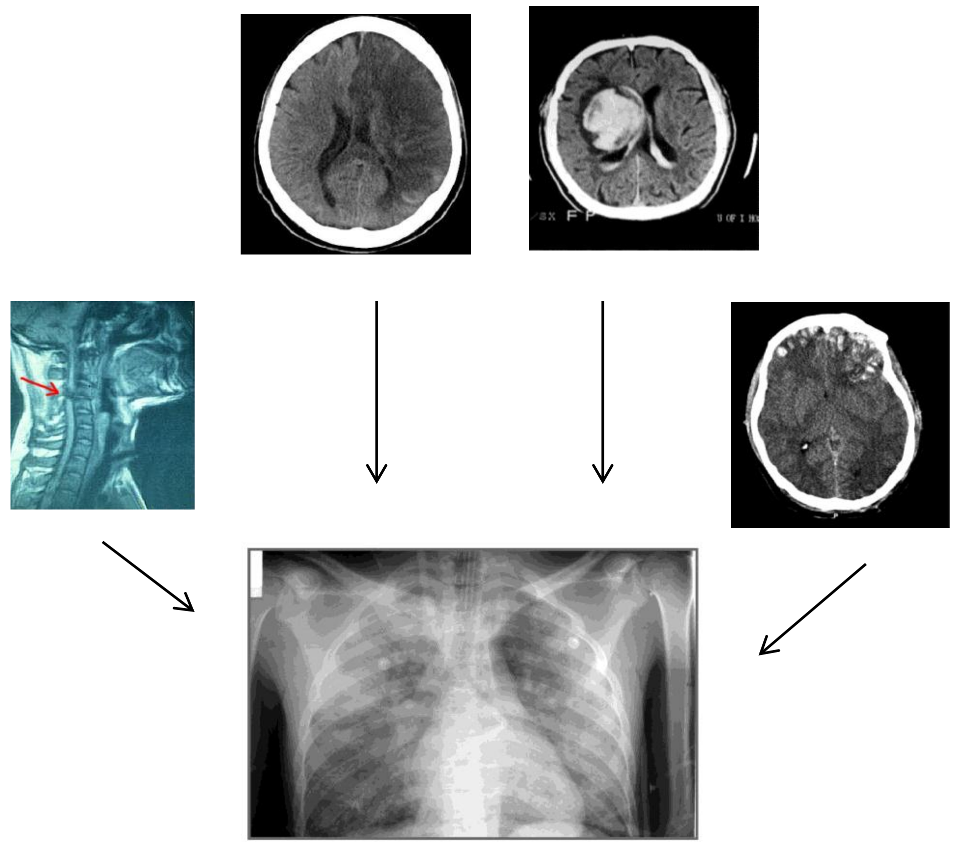
<u>20-25%</u> of all patients with isolated brain injury will develop ARDS/ALI

NPE in Neurological Injury

Neurogenic Pulmonary Edema (NPE): A Hybrid Form of ARDS

Major Causes:

- Cervical Cord Injury
- Acute Stroke
  - – MCA Syndrome
  - – Hemorrhagic
- Epileptic Seizure
- Head Injury



Neurogenic Pulmonary Edema (NPE)

Definition: Clinical syndrome characterized by acute pulmonary edema following a significant central nervous system insult (CNS)

Mechanism: Surge in catecholamines resulting in cardiopulmonary dysfunction

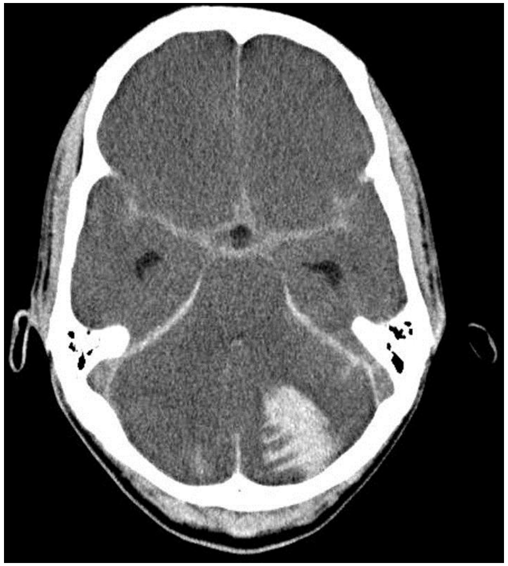
Diagnosis: Very similar to ARDS once all other causes of hypoxemic respiratory failure are eliminated

Types of Acute Brain Injury

- Subarachnoid Hemorrhage (SAH)
- Traumatic Brain Injury (TBI)
- Acute Ischemic Stroke (AIS)
- Intracranial Hemorrhagic (ICH)

Spontaneous Subarachnoid Hemorrhage (SAH)

- Severity associated with outcomes
  - ARDS/NPE in 2-42.9% of all SAH
- Complications (neurological)
  - Cerebral vasospasm
  - Delayed cerebral ischemia
- Treatment "Triple H Therapy"
  - Goal to maintain euvolemia
- Neurogenic Pulmonary edema (NPE)
  - Sudden rise ICP + increased SVR = elevated venous tone/more venous return leading to hydrostatic pressure leaking in pulmonary vasculature



Hunt and Hess scale

Grade	Criteria
0	unruptured aneurysm
1	Asymptomatic, or minimal headache, nuchal rigidity
2	Moderate to severe headache, no neurologic deficit except for cranial nerve palsy
3	Drowsiness, confusion, mild focal deficit
4	stuporous, moderate to severe hemiparesis, early decerebrate
5	Deep coma, decerebrate posturing, moribound

Traumatic Brain Injury (TBI)

- TBI is major cause disability, morbidity and mortality
- ARDS/ALI is common in-hospital after TBI
- 20-year US study of adult admissions with TBI and ARDS/ALI (1988-2008)
- More common in young men, white race, CHF, HTN, COPD CKD, CLF, sepsis
- Prevalence increased from 2% to 22%

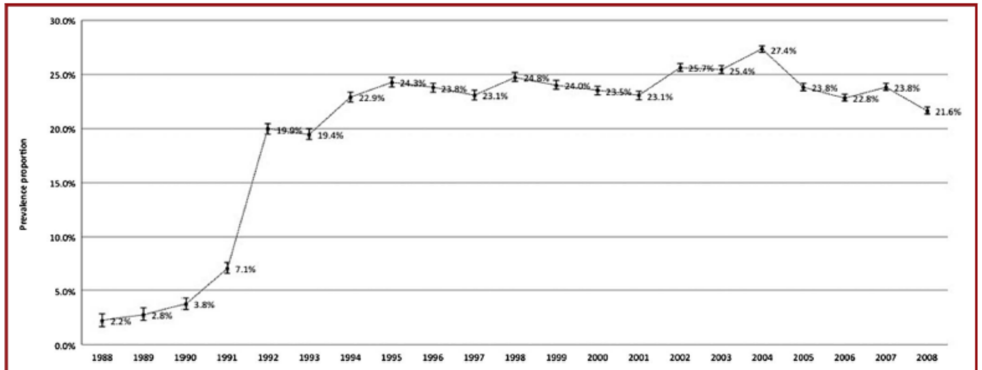


FIGURE 1. *Prevalence of ARDS/ALI in admissions of patients with TBI from 1988 through 2008 in the United States (Squares represent the point prevalence of ARDS/ALI after TBI. The I bars represent standard error). ARDS, acute respiratory distress syndrome; ALI, acute lung injury; TBI, traumatic brain injury.*

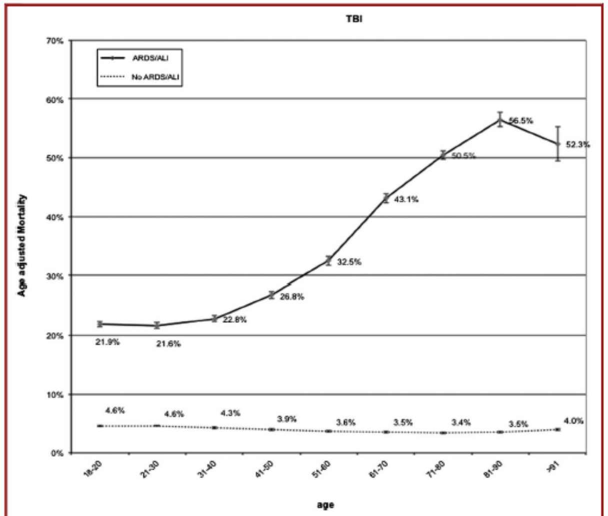


FIGURE 2. *Age-adjusted in-hospital mortality proportion after TBI in the United States (The I bars represent standard error, age per deciles). ARDS, acute respiratory distress syndrome; ALI, acute lung injury; TBI, traumatic brain injury.*

Rincon F. Neurosurgery 2012;71:792-802

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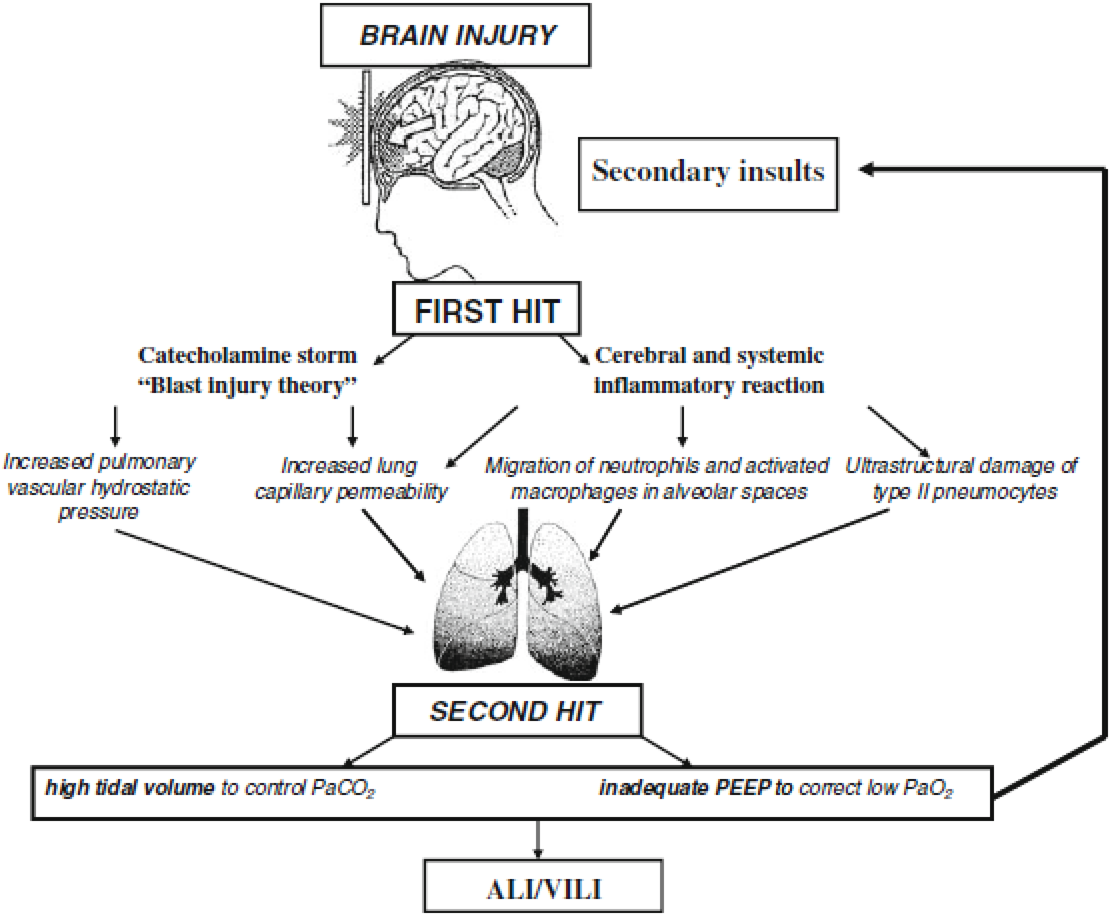
Proposed Mechanism: "Double Hit Model"



<u>Pathophsyiology</u>

- Injury creates inflammatory reaction
- Catecholamine storm
- Leading to pulmonary changes
- Increased ventilation requirements
  - High VT to control PaCO2
  - ↑ PEEP needed to correct PaO2
  - Leads to ARDS
- Secondary brain insults

Mascia L. Neurocrit Care. 2009

Acute Ischemic Stroke (AIS) and ICH

- Lower mortality than hemorrhage (5%)
- Death mostly related to medical complications
  - DVT, PE, UTI, Pneumonia
- Respiratory failure in AIS
  - Severity related, e.g. Malignant MCA Infarcts
  - Risk factors: Infarct size, age > 60, GCS < 10
  - Mortality as high as 50% in AIS on ventilator

Mayer SA. Stroke. 200;31:2346-2353.

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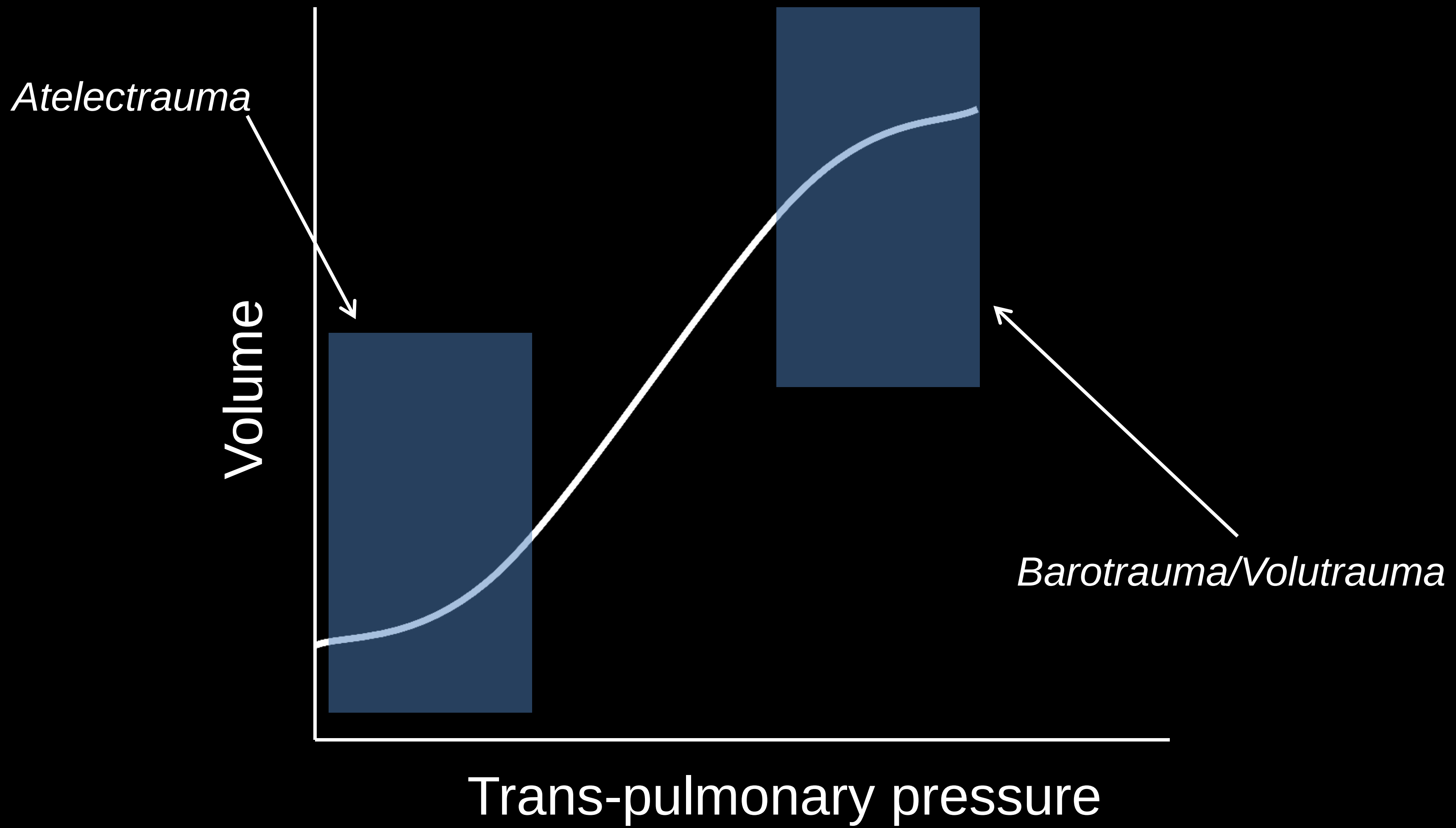
Treatment Options: Ventilation Challenges

- Mechanical ventilation is supportive therapy
  - Basics:
    - Supply O2 and remove CO2
    - **Head of Bed Elevated 30-45 degrees**
    - Diagnosis and treat precipitating cause
    - Prevent ventilator-induced lung injury (VILI/VALI)***
- PaCO2 Control
  - Hyperventilation treats intracranial hypertension lowering ICP by reducing cerebral blood volume
  - Brain Trauma Foundation Recommendations
    - Avoid prophylactic hyperventilation ($PaCO_2 \leq 25$ mmHg)
    - Hyperventilate only temporary to reduce ICP
    - Avoid 1st 24 hours when CBF is compromised
    - If needed, invasively monitor jugular oxygen saturation

VALI occurs at high and low lung volumes



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Treatment Options: Ventilation Challenges

- <u>Oxygenation and Positive End-Expiratory Pressure (PEEP)</u>
  - Hypoxemia associated with ↑morbidity and mortality
  - BTF guidelines:
    - Avoid PaO_2 < 60 mmHg or O_2 saturation < 90%
  - Use adequate FiO2 and PEEP
  - PEEP recruits collapsed alveoli = improved O_2
  - Tradeoff:
    - PEEP decreases arterial pressure and cerebral blood flow in autoregulation impaired patients
    - PEEP impairs local venous return and increases right atrial pressure (RAP)

Treatment Options: Alternative Techniques

- **Lung protective strategy**
- Permissive hypercapnia
- Prone Positioning
- ECMO
- Inhaled nitric oxide (NO)
- HFOV
- Corticosteroids

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Treatment Options: Ventilation Challenges

- **Lung-Protective Mechanical Ventilation**
  - Mechanical ventilation using limited tidal volumes
  - Goals: Avoid injury to overexpansion of alveoli during inspiration and injury due to repetitive opening an closing of alveoli during inspiration and expiration

Tiny is Now the New Big-When it Comes to Ventilation Goals

Low Tidal Volume Ventilation (LTVV)

Initial Settings

- Calculate Ideal Body Weight (IBW) in KG
  - Males: IBW = 50 kg + 2.3 kg for each inch over 5 feet.
  - Females: IBW = 45.5 kg + 2.3 kg for each inch over 5 feet.

1 foot = 30.48 cm

1 inch = 2.54 cm

- Set initial tidal volume to 8 ml/kg IBW
- Reduce TV to 7 ml/kg IBW then 6 ml/kg over next 1-3 hrs
- Set RR to ≤ 35 bpm to match baseline minute ventilation

Low Tidal Volume Ventilation (LTVV)

<u>Adjusting Settings</u>

- Adjustments to tidal volume are based on Plateau pressures
- Goal: Plateau pressure ≤ 30 cm $H_2$0
- IF Plateau pressures > 30 cm $H_2$0, the TV setting $\downarrow$ by 1 ml/kg IBW increments to minimum of 4 ml/kg IBW
- <u>Note</u>: Using LTVV when Plateau pressure are normal also shown to benefit

Treatment Options: Ventilation Challenges

- Permissive Hypercapnia
  - Defined as clinician-allowed hypercapnia during assisted ventilation, despite ability to achieve a level of minute ventilation sufficient to maintain a normal pCO_2
  - Though in acute brain injury with ARDS, protective ventilation with prevention of hypercapnia is required to avoid acute elevations in ICP

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Treatment Options: Ventilation Challenges

- Prone Positioning
  - 2/3 ARDS patients exhibit improved oxygenation with prone positioning
  - Mechanism: Redistribution of lung perfusion and a change in regional diaphragm motion
  - Mixed clinical data
  - Reserved for severest ARDS patients
    - 14-16 hours prone then supine

Abroug F. Intensive Care Med. 2008;34:1002-1011.
Guerin C. N Engl J Med 2013;368:2159-2168

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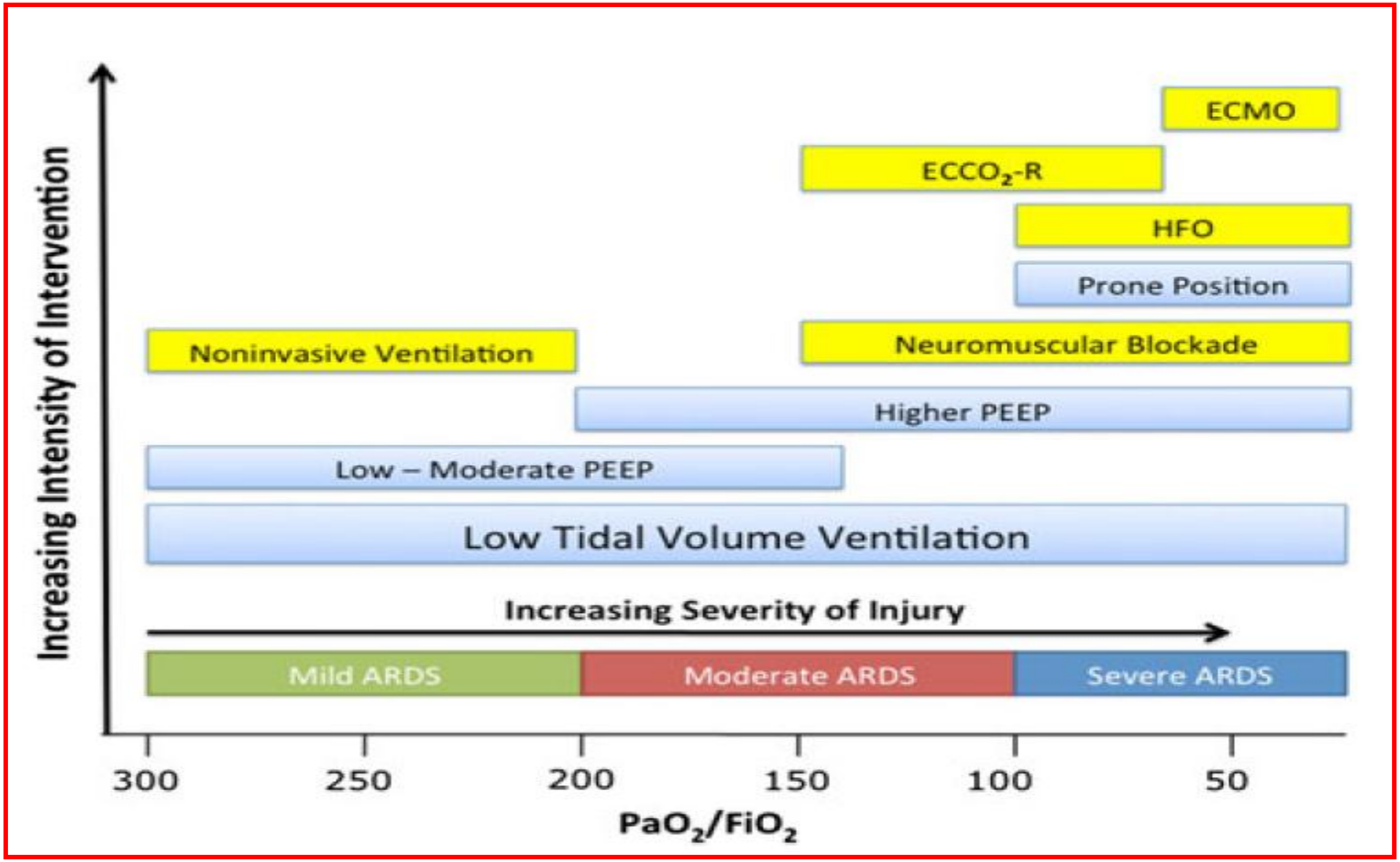
Treatment Options: Ventilation Challenges

- Adjuncts to Lung Protective MV
  - Inhaled selective pulmonary vasodilator
    - Nitric Oxide
    - Prostacyclin
  - High-Frequency Ventilation
  - Extracorporeal Gas Exchange (ECMO)
  - Steroids
  - Neuromuscular Blocking Agents

Summary Mechanical Ventilation Options



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Case Conclusion

- Difficulty in oxygenation (80% on FiO2, RR 40)
- Adjusted TV 650 ml (9.2 ml/kg) to 560 ml (8 ml/kg) then 490 ml (7 ml/kg)
- Increased PEEP to 12 cm H20 (from 5 cm H20)
- 1.5 L Fluid bolus (NS) to maintain MAP 90 mm Hg
- Paralyzed patient
- Allow RR to remain 35-40 bpm
- Improve O2 saturation to 94% on FiO2 .5
- HTS 23.4% through central line for worsening ICP
- Transferred to Trauma ICU

Conclusion

- Fluid management goals: Euvolemia
- Selective use of hypertonic solutions
- ARDS is multisystem syndrome
- Brain injury can trigger NPE/ARDS
- Treatment is supportive BUT should consider effects of MV on brain physiology
- Lung-protective strategy most encouraging to avoid VILI
- Many alternative therapies for extreme cases

Thank You! Teşekkür Ederim!

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