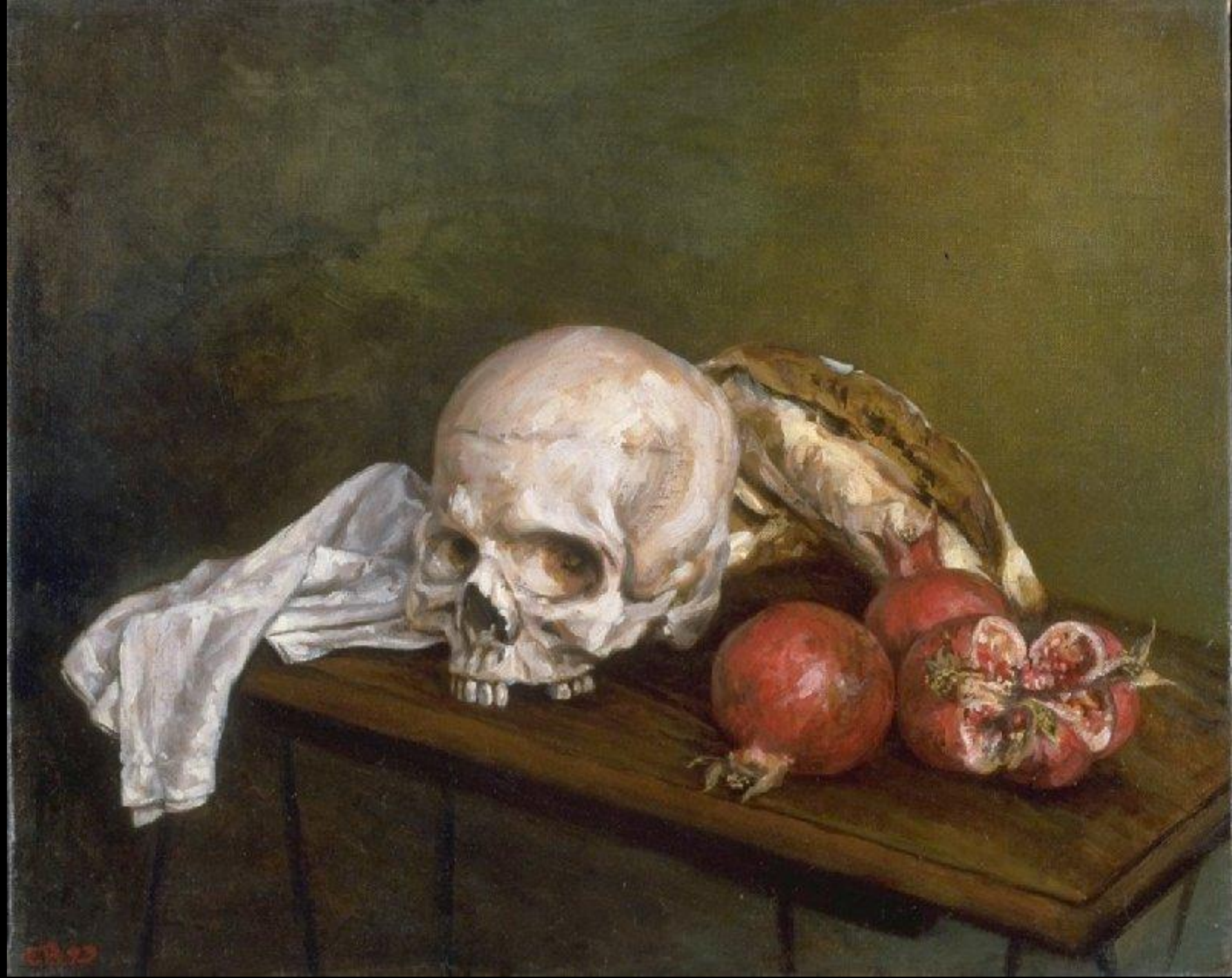


ICP

A Stepwise Protocol

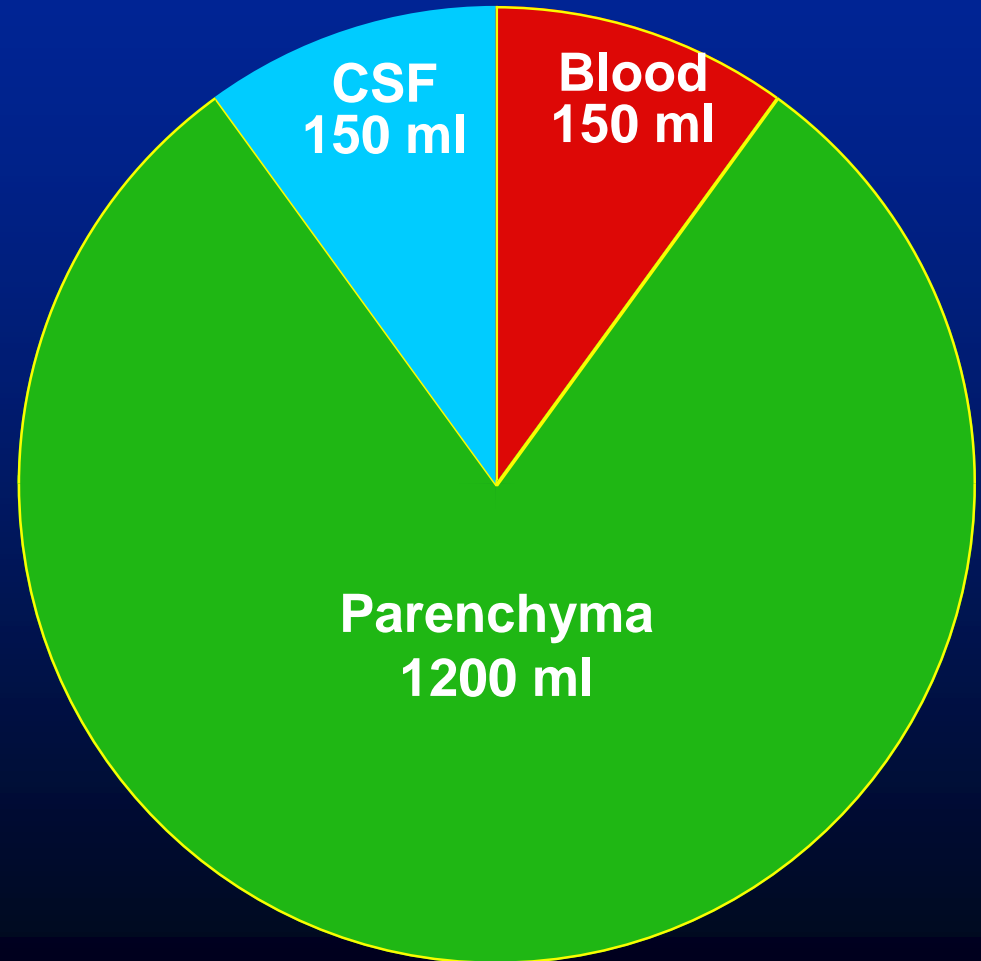
Stephan A. Mayer, MD

Neurological Intensive Care Unit
Neurological Institute of New York
Columbia-Presbyterian Medical Center
New York, NY



ICP: Basic Concepts

- **Monroe-Kellie doctrine:**
skull = fixed volume
- **3 components of intracranial volume**
- **Normal ICP**
 - ≤ 20 cm H₂O
 - ≤ 15 mm Hg



Causes of Increased ICP:

Space Occupying Lesion

Hematoma, Tumor, Abscess

Increased CSF

Hydrocephalus

Inc. Blood Volume (Vasogenic edema)

Trauma, Tumor, Abscess, Hypertensive encephalopathy

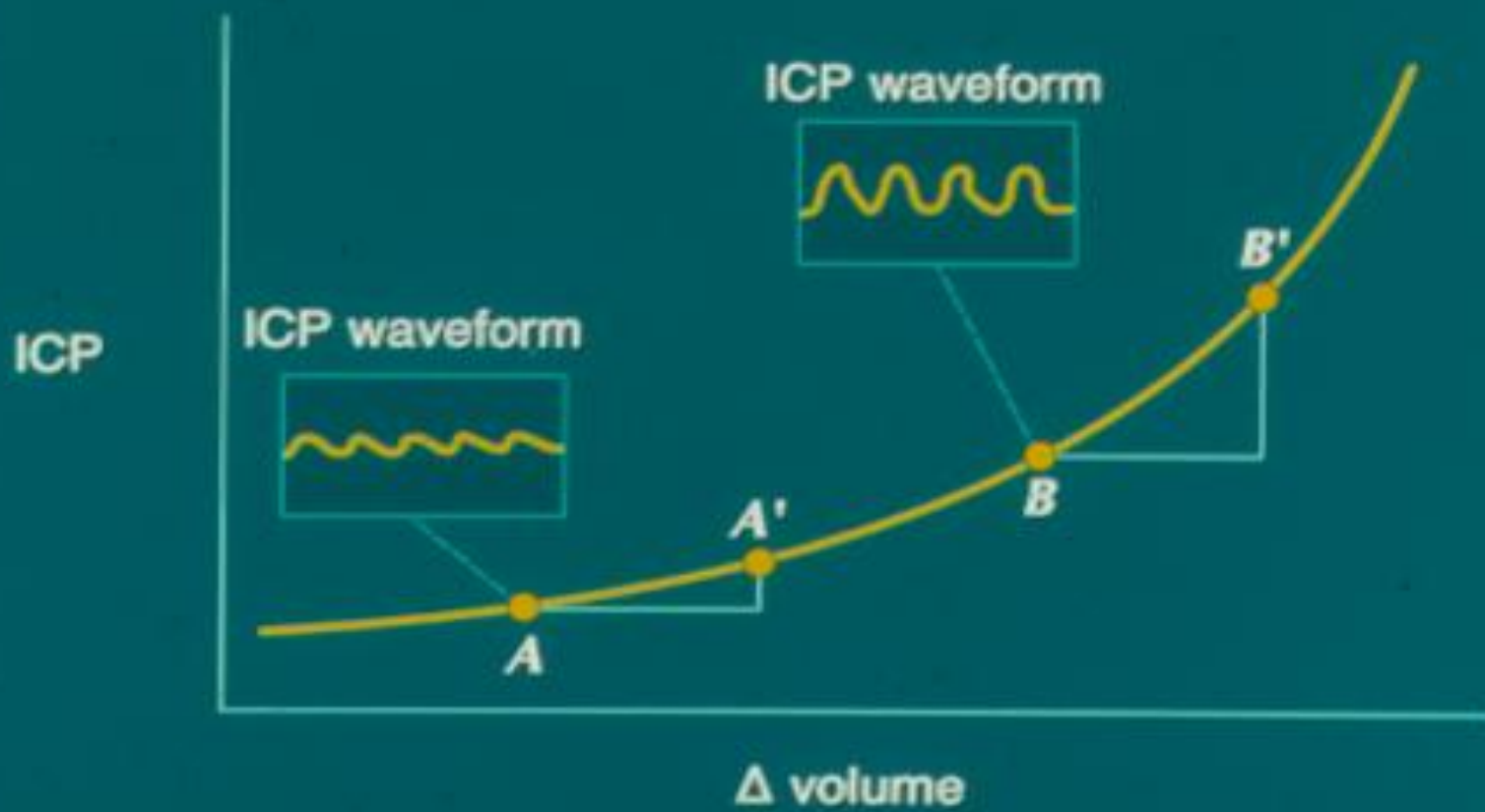
Inc. Brain Volume (Cytotoxic edema)

Infarction, Ischemia

Methods to Reduce Elevated ICP

- **Remove Mass Lesion** **Surgical Evacuation**
- **Reduce CSF Volume** **Ventricular Drainage**
- **Reduce Cerebral Blood Volume** **Hyperventilation, Barbiturates, Hypothermia**
- **Reduce Parenchymal Volume** **Osmotic Diuretics (Mannitol, Hypertonic Saline)**

ICP





Normal ICP; normal compliance

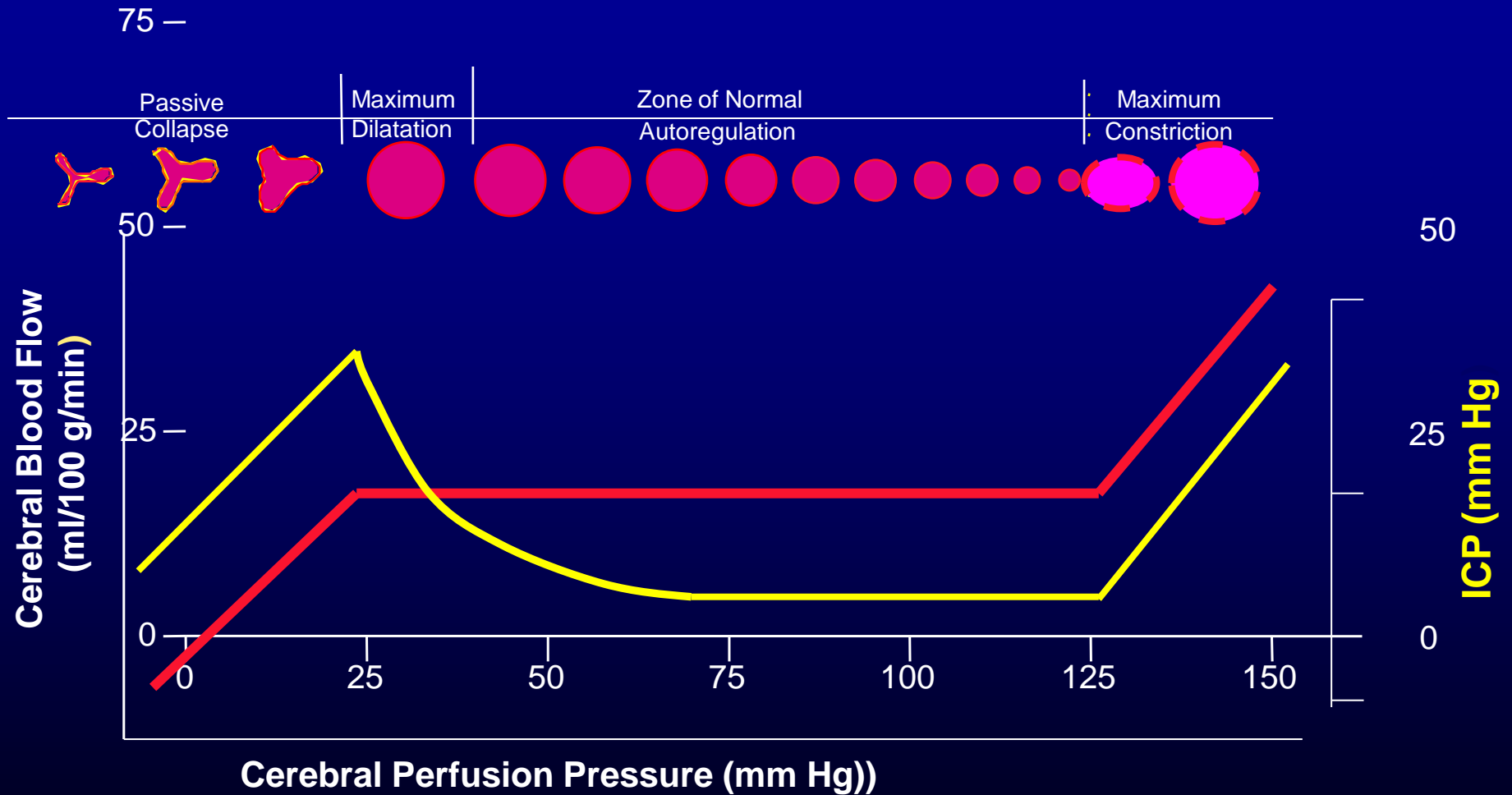


Elevated ICP; reduced compliance

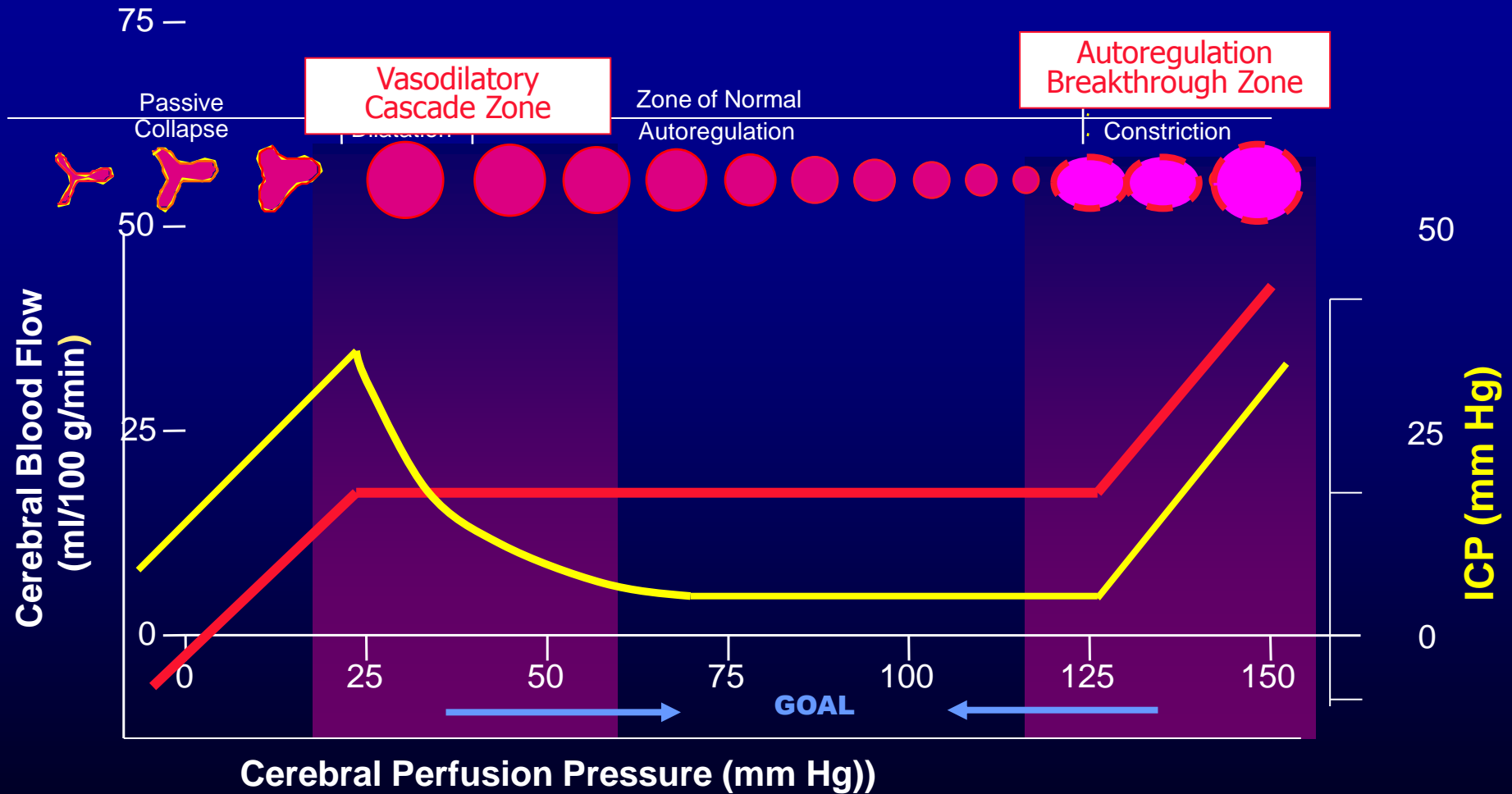
20 mm Hg

1 sec

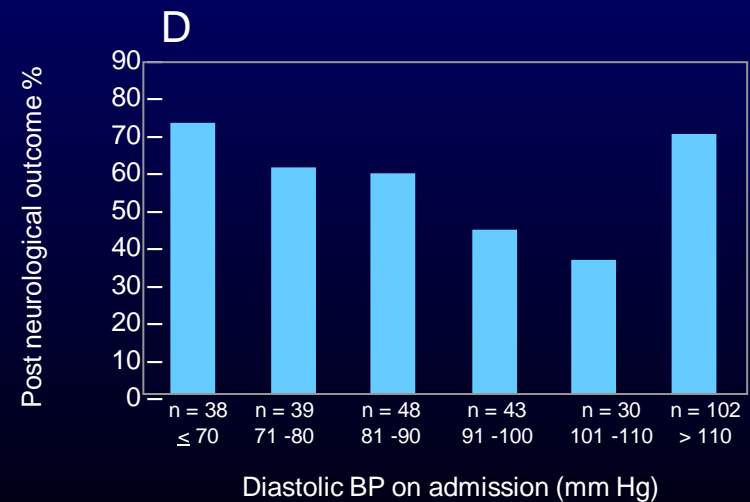
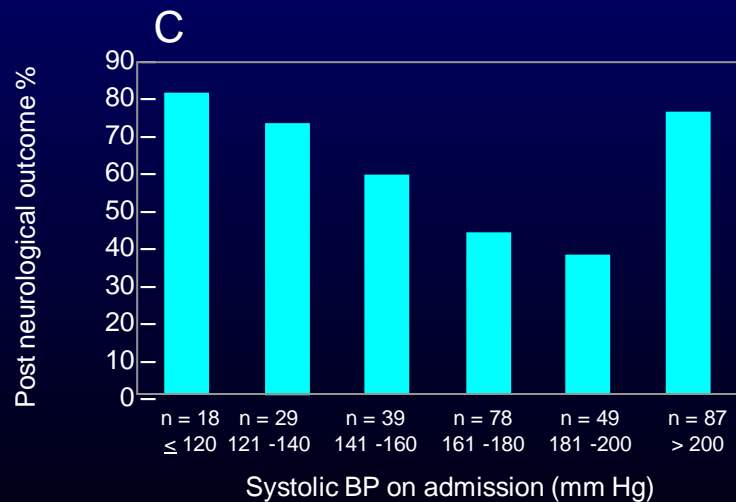
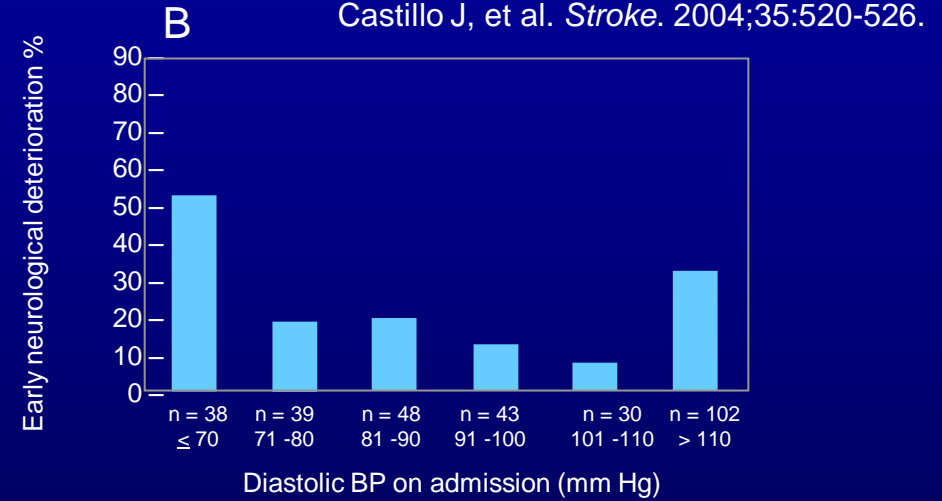
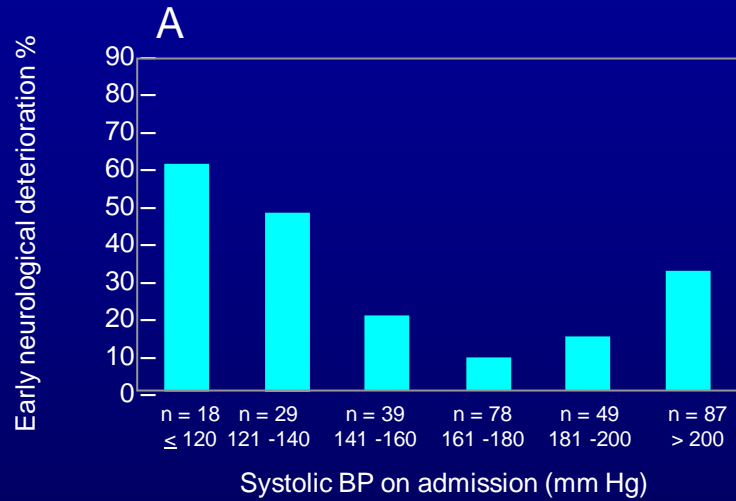
ICP/CPP Management



CPP can influence ICP when you run out of room



Outcome after Acute Ischemic Stroke by Admission Blood Pressure



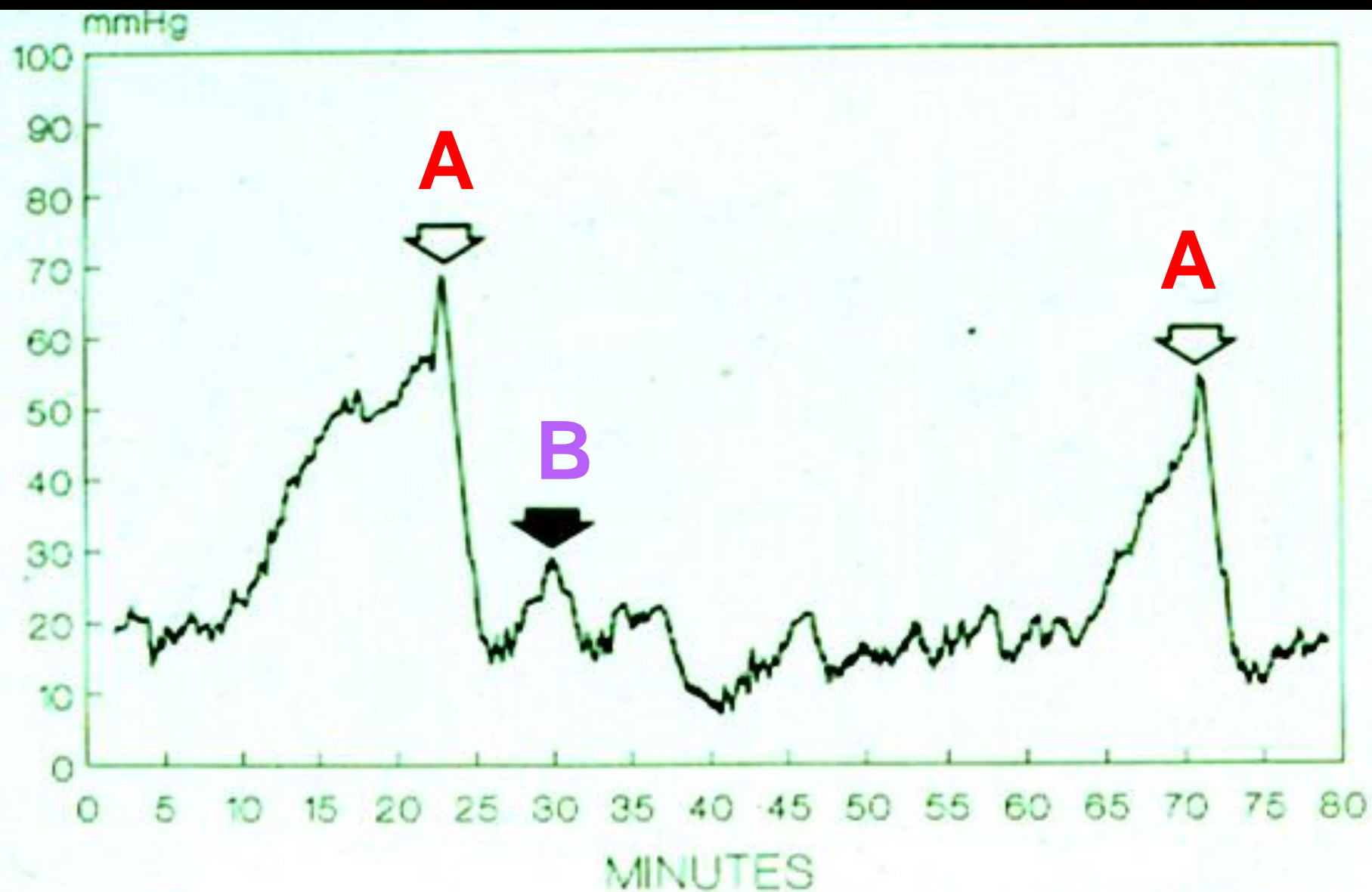
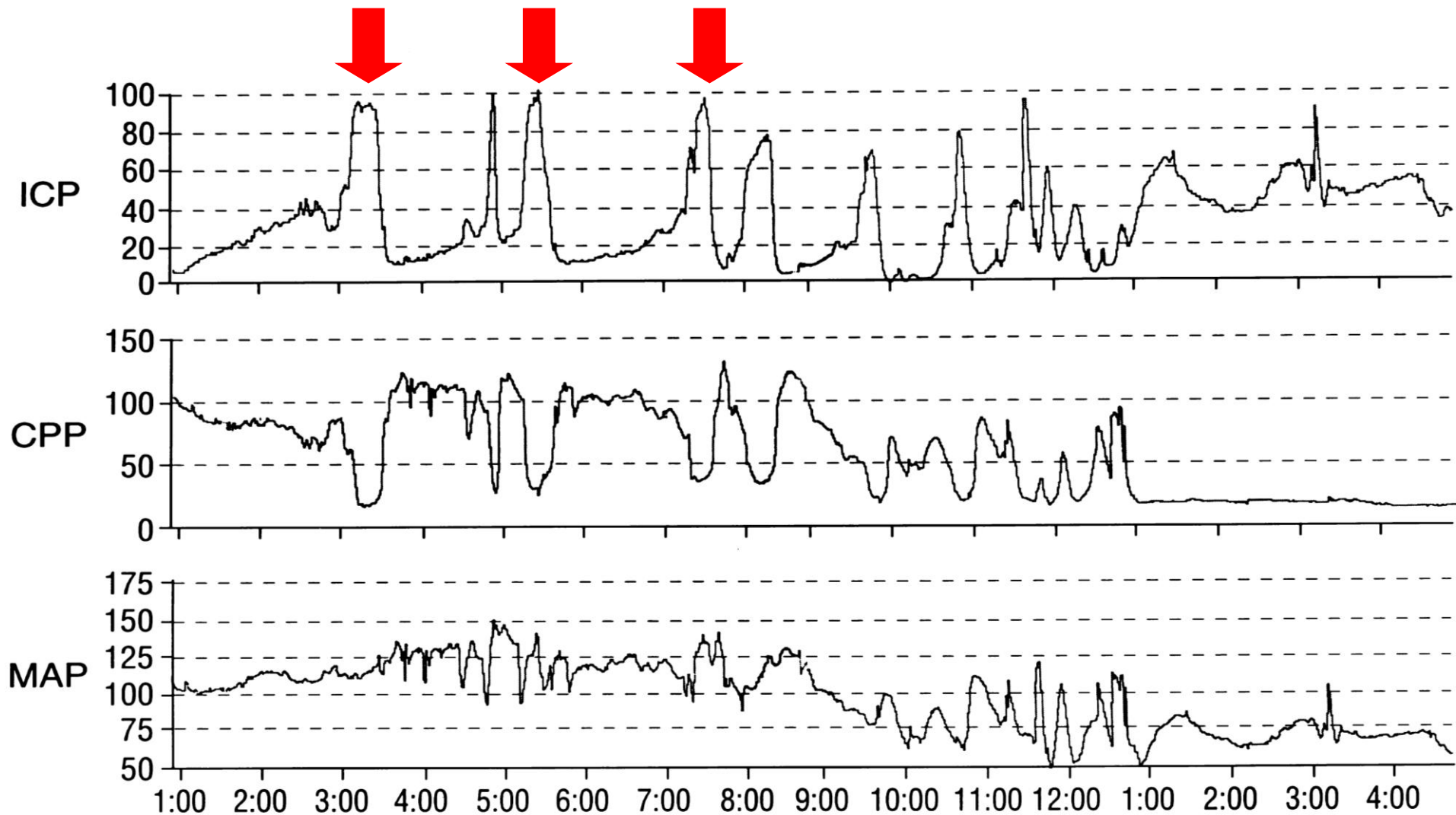


Figure 5-5. Two classic A waves are shown (open arrows). Note that when the ICP falls after the A wave (closed arrow), it does not return to the baseline preceding the first wave.



PLATEAU (LUNDBERG A) WAVES

Indications for ICP Monitoring

- Coma (Glasgow Coma Scale score ≤ 8)
- CT evidence of intracranial mass effect
 - Extra-axial mass lesion
 - Midline shift
 - Effacement of basal cisterns
 - Exception: severe TBI with motor posturing
- Prognosis is such that aggressive ICU care is warranted

Clinical Signs

- **Increased ICP**
 - Depressed level of consciousness
 - Pressor response
 - Projectile vomiting
 - CN 6 palsies
- **Brainstem herniation**
 - CN 3 palsy
 - Motor posturing
 - Lower extremity rigidity
 - Loss of lateral EOMs
 - Hyperventilation

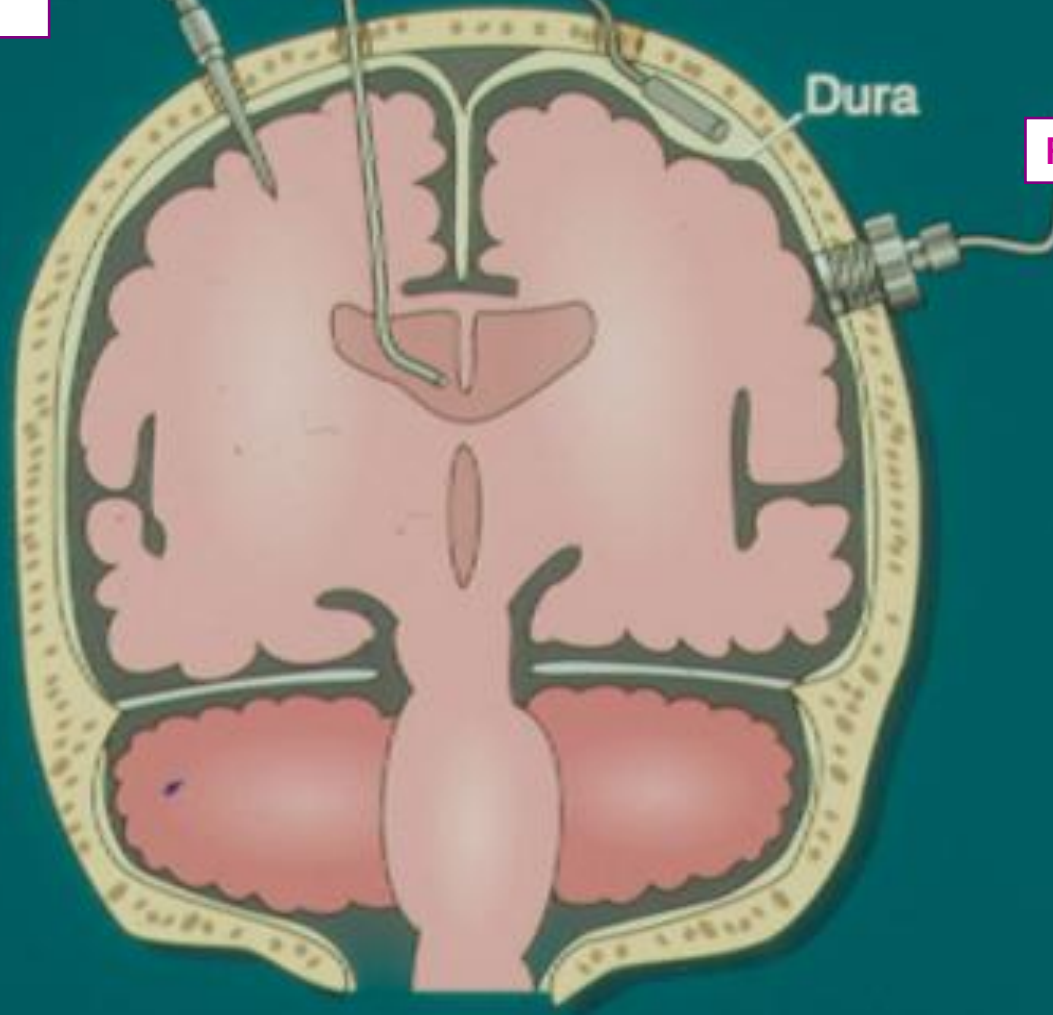
Parenchymal
Micosensor

Ventricular catheter

Epidural Monitor

Dura

Richmond Bolt



ICP/CPP Treatment Thresholds

Guideline

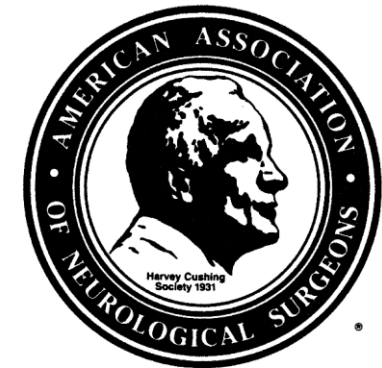
- **ICP treatment should be initiated at an upper threshold of 20 mm Hg.**

Option

- **Cerebral Perfusion Pressure should be maintained at a minimum of 60 mm Hg.**

GUIDELINES for the Management of

SEVERE HEAD INJURY

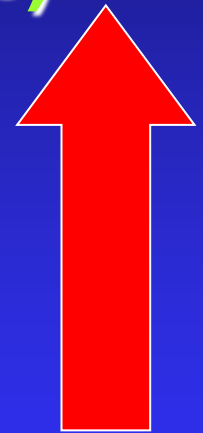


A Joint Initiative of:

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

ICP: General Care Issues

- Elevate head of bed 30°
- **Use only isotonic fluids (0.9% saline)**
- Control fevers aggressively
- Seizure prophylaxis
- No routine steroids use

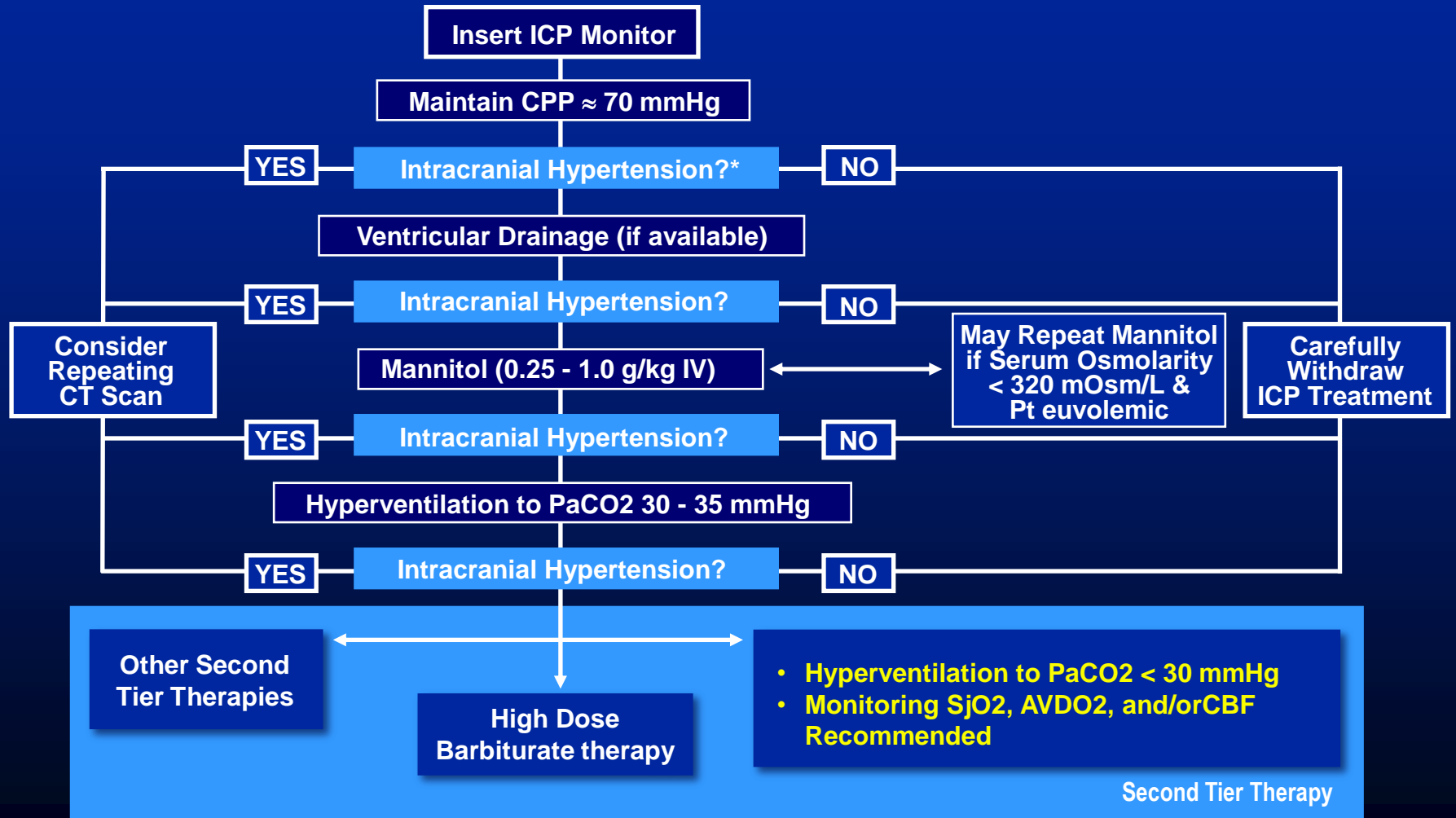


Option: 3% saline or mannitol for target osmolality of 300-320 mOsm/L

Emergency Treatment of Increased ICP

- **Un-monitored patient with clinical signs of herniation**
 - Elevate head of bed 30°
 - Normal saline 100 ml/hr
 - Intubate and hyperventilate (pCO₂ 30 mm Hg)
 - Mannitol 20% 1.0 to 1.5 g/kg rapid IV infusion
 - Foley catheter
 - CT scan and neurosurgical evaluation

Critical Pathway for Treatment of Intracranial Hypertension in the Severe Head Injury Patient (Treatment Option)



**ICP
PROTOCOL**



Columbia Stepwise ICP Protocol

7 HYPOTHERMIA

6 PENTOBARBITAL

5 HYPERVENTILATION

4 OSMOTHERAPY

3 CPP OPTIMIZATION

2 SEDATION

1 SURGICAL DECOMPRESSION

Revised
Columbia
Stepwise ICP
Protocol

7 PENTOBARBITAL

6 HYPOTHERMIA

5 HYPERVENTILATION

4 OSMOTHERAPY

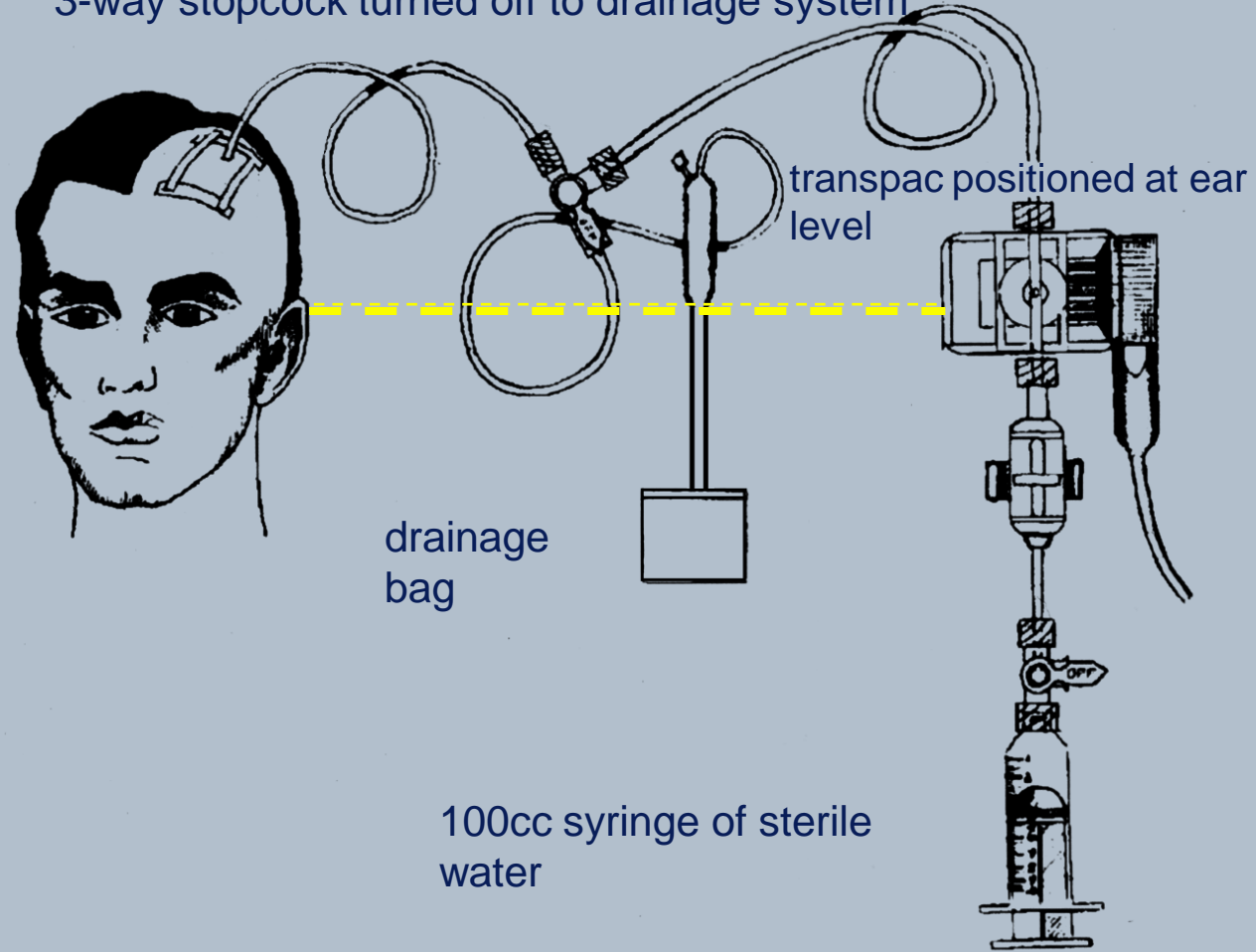
3 CPP OPTIMIZATION

2 SEDATION

1 SURGICAL DECOMPRESSION

1

3-way stopcock turned off to drainage system



LEVEL

Ventricular Drainage



Aneurysmal SAH with associated subdural hematoma and bilateral motor posturing

2

ICU Intravenous sedation

- **Goal is reversibility to allow repeated neurologic assessment**
- **Alternatives (in intubated pts):**
 - *Fentanyl or Remifentanyl*
 - *Midazolam*
 - *Propofol*
 - » **Ultrashort acting**
 - » **Allows “wake-up” in 5-15 mins**
 - » **Reduces ICP, CMRO₂**
 - » **Drawbacks: hypotension, infection**

Columbia
Stepwise ICP
Protocol

⑦ *HYPOTHERMIA*

⑥ *PENTOBARBITAL*

⑤ *HYPERVENTILATION*

④ *OSMOTHERAPY*

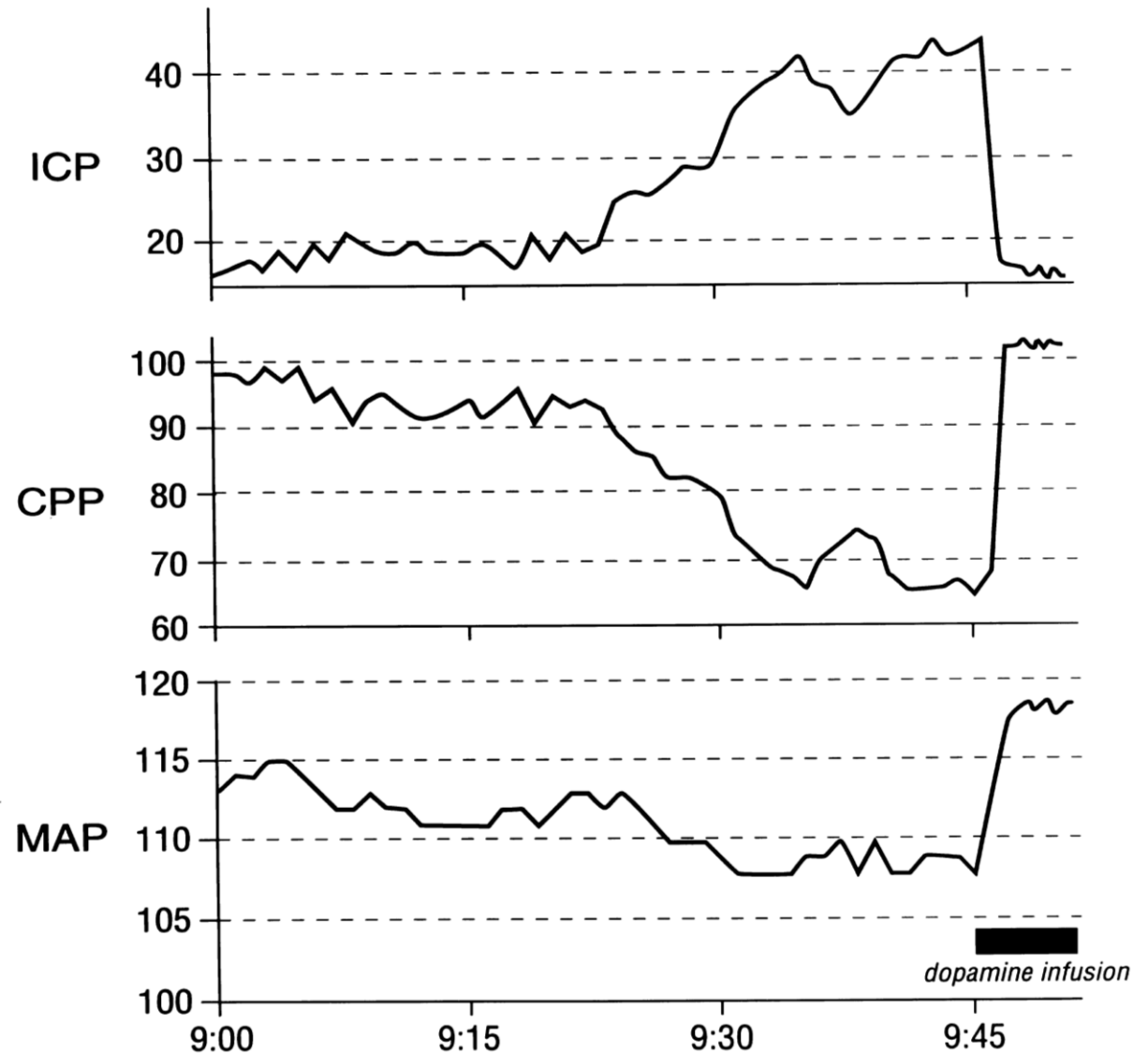
③ ***CPP OPTIMIZATION***

② *SEDATION*

① *SURGICAL DECOMPRESSION*

3

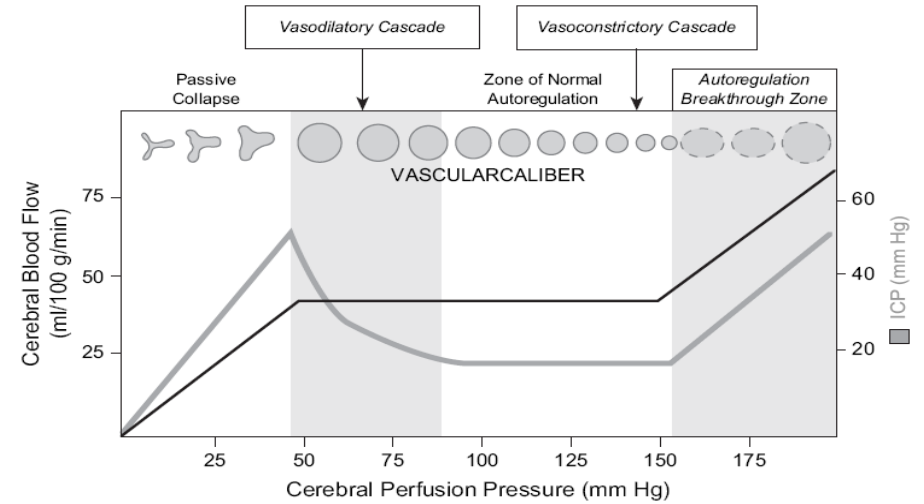
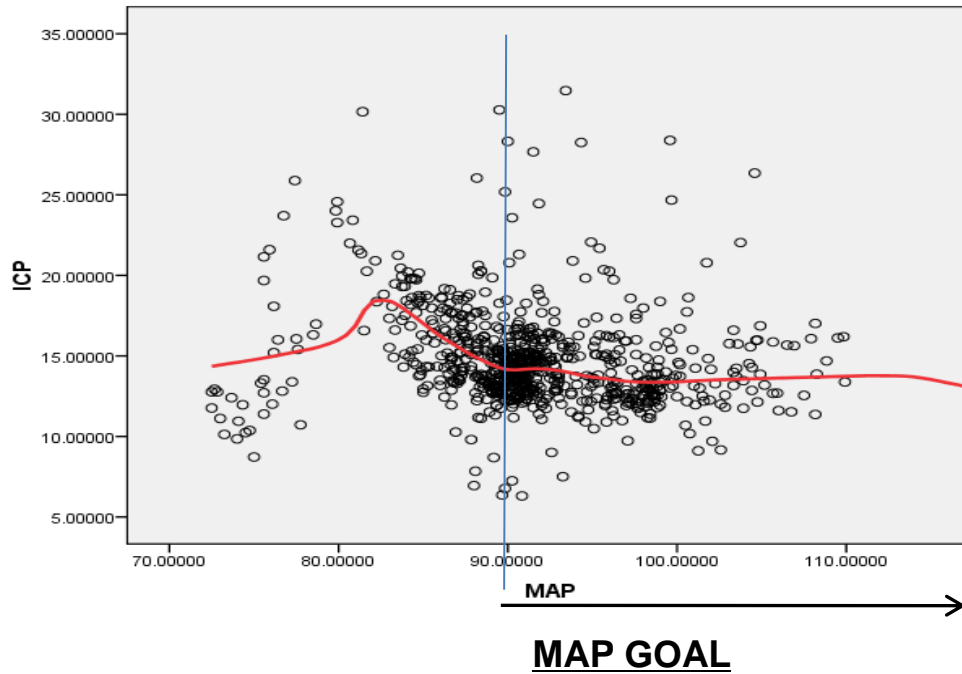
CPP
OPTIMIZATION:
Dopamine
infusion
resulting in
increased MAP
and CPP, and
decreased ICP



Lund protocol

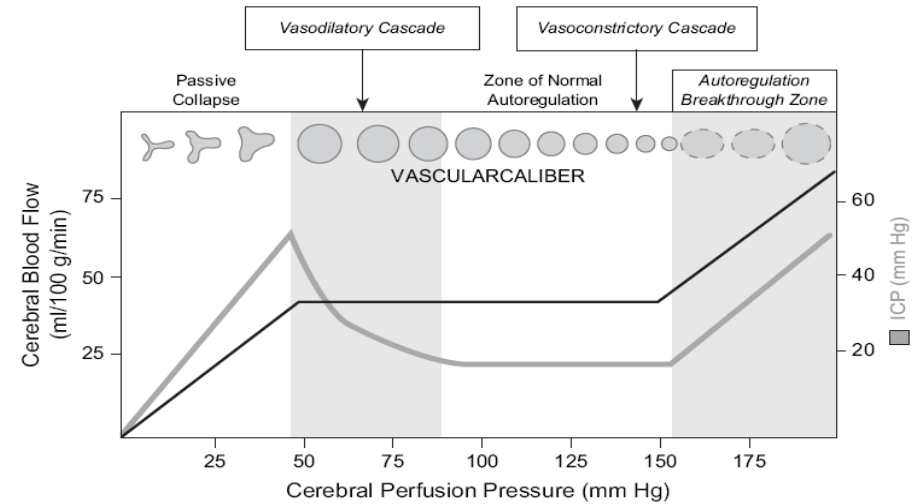
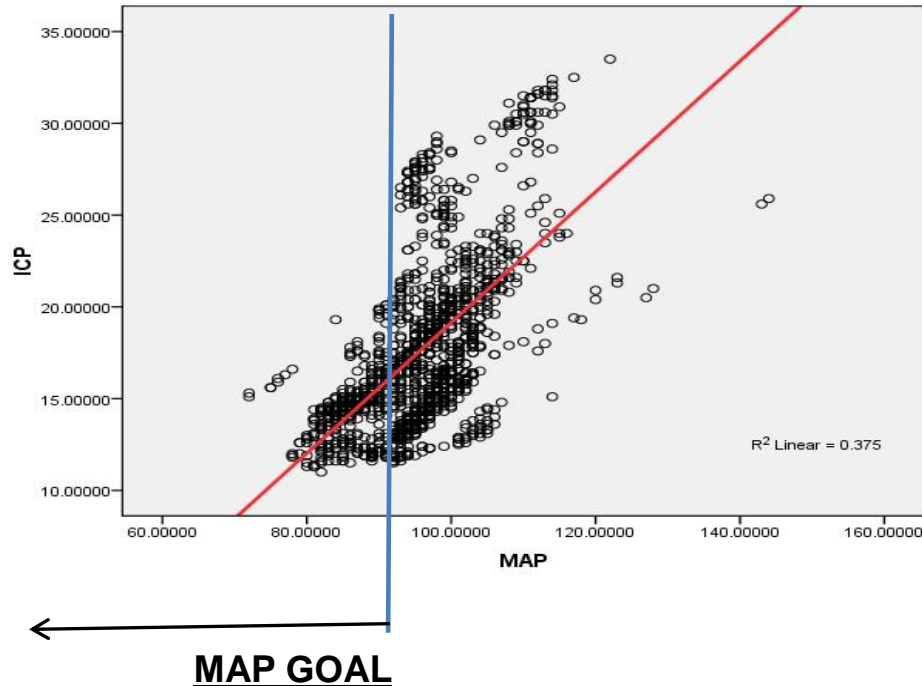
- Reduce $CMRO_2$ with sedation
 - Miazolam, thiopental, fentanyl
- Reduce BP and capillary hydrostatic pressure (CPP 50-70 mm Hg)
 - IV Metoprolol (.2 mg/kg/hr) and clonidine (.5 mcg/kg prn)
- Dihydroergotamine .1-.8 mcg/kg/hr
- Maintain normal hematocrit, CVP, and albumin levels

MMM: “Imaging” Vasodilatory Cascade Physiology



Crit Care Clin 23 (2007) 507–538

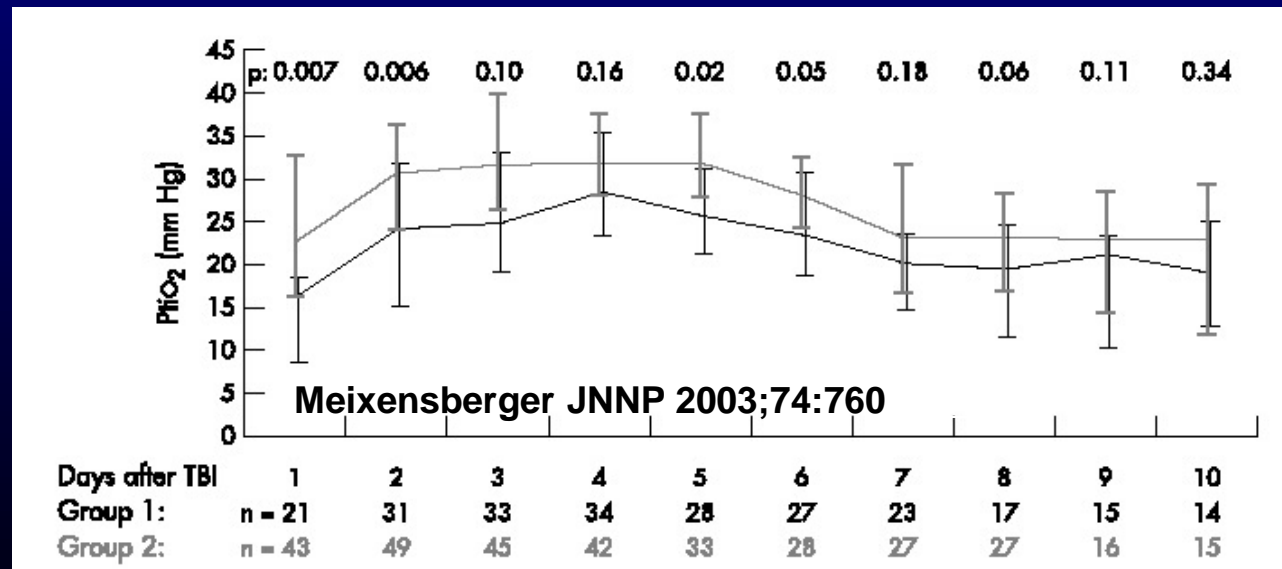
MMM: “Imaging” Perfusion Pressure Breakthrough Physiology



Crit Care Clin 23 (2007) 507–538

Brain tissue oxygen guided treatment supplementing ICP/CPP therapy after traumatic brain injury

- 93 severe TBI patients
 - 40 treated for CPP >70 alone (1993-96)
 - 53 treated for CPP >70 + PbrO₂ >10 mm Hg as needed (1997-00)
- Boosting CPP lead to less cerebral hypoxia
- Larger studies needed to see if this improve outcomes



4

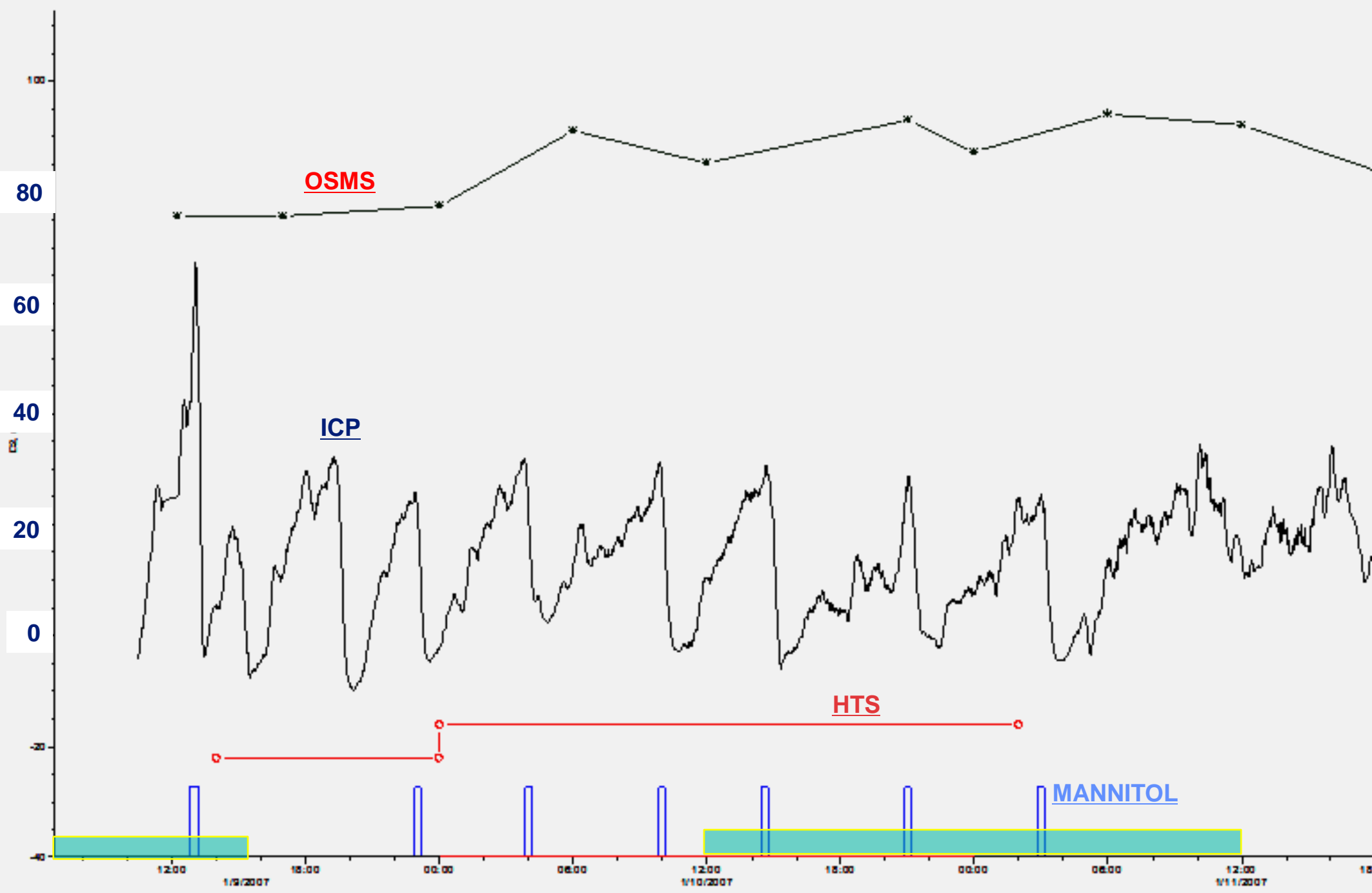
Osmotherapy

- **Mannitol**

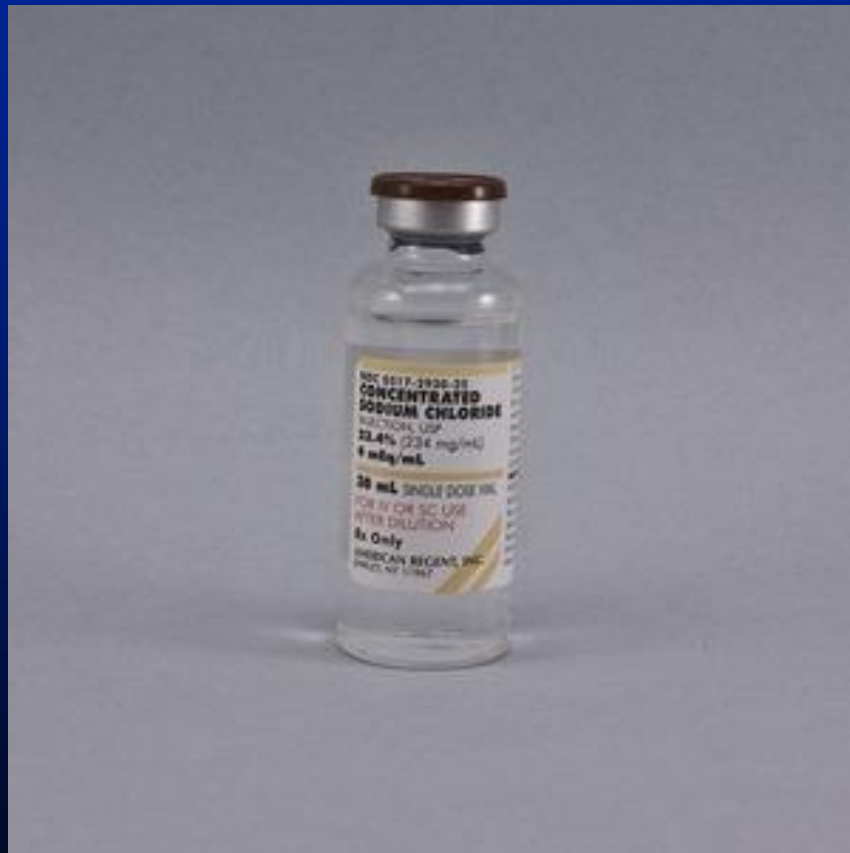
- 0.25 to 1.5 g/kg IV wide open
- Dose up to Q1H on an as-needed basis
- Mechanisms:
 - » Acute dehydrating effect (osmotic gradient across BBB)
 - » Secondary hyperosmolality (diuretic effect)
 - » Reflex vasoconstriction (viscosity effect)

- **Hypertonic Saline**

- Varying concentrations: 3%, 7.5%, 10%, and 23.4%
- Optimal dosing not known



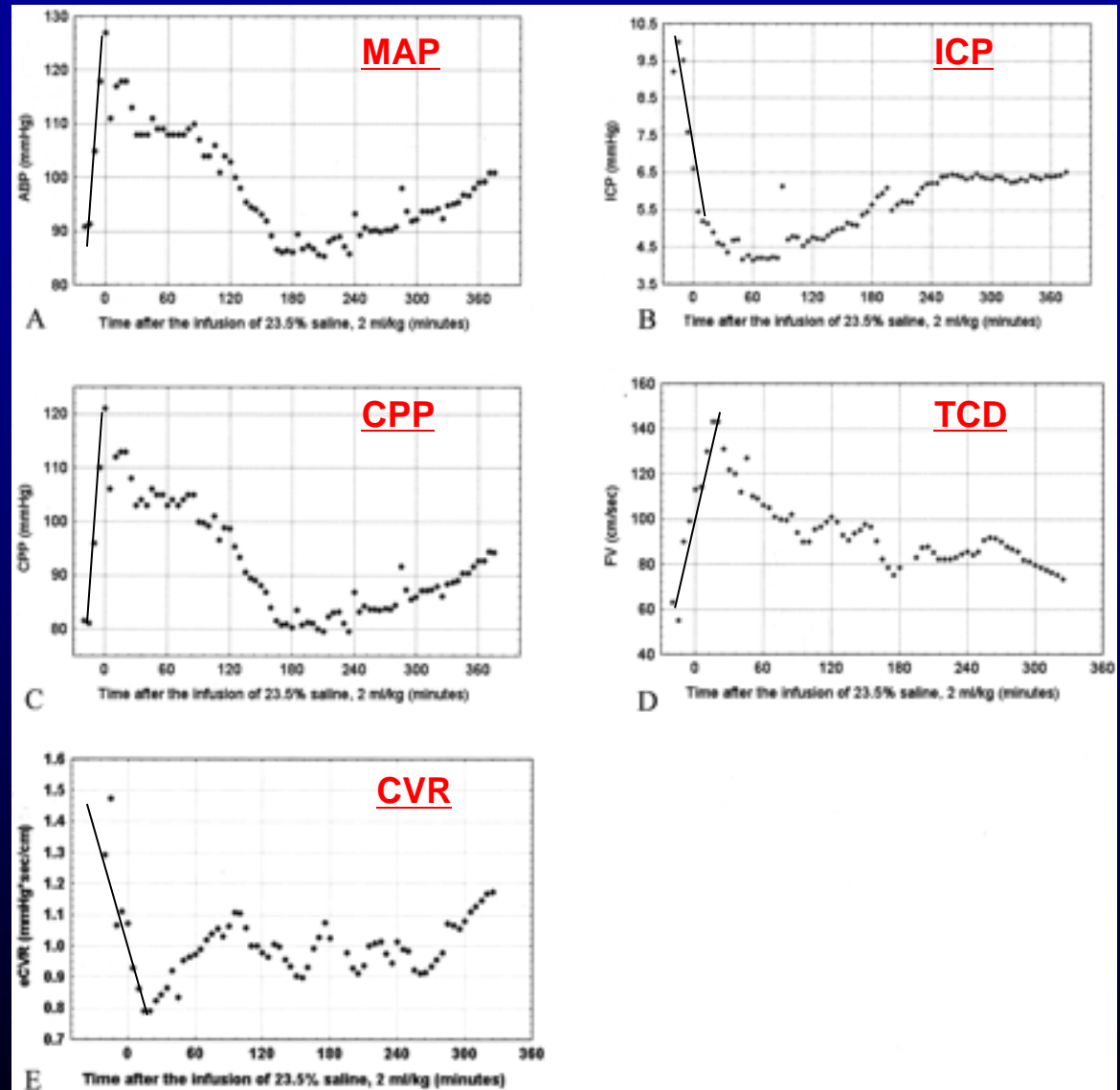
30 ml 23.4% Bullets



Effect of Hypertonic Saline in CBF in SAH patients

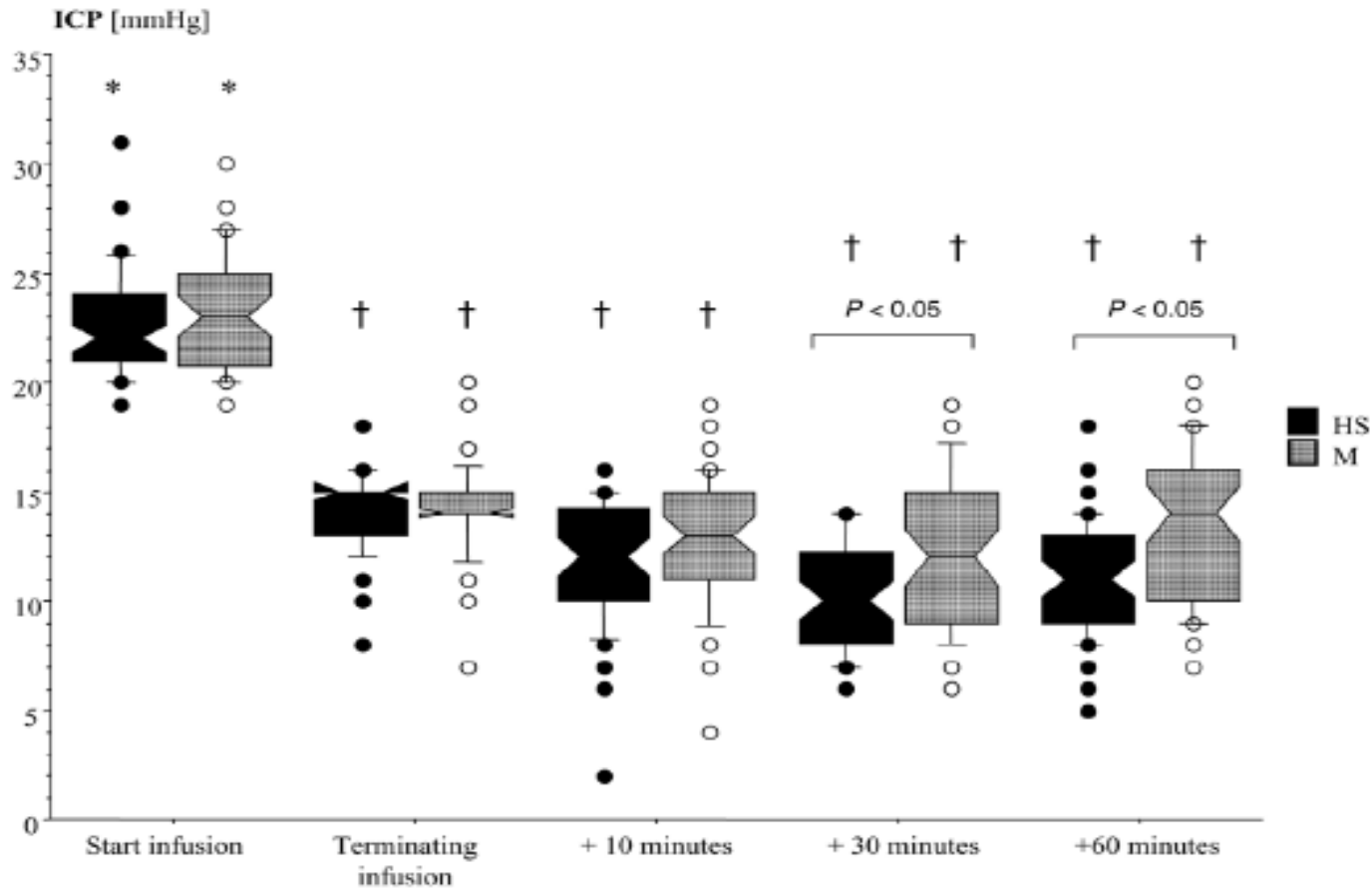
TSENG M-Y, Stroke 2003;34:1389.)

- 10 poor grade SAH patients
- 2 mL/kg of 23.5% saline
- ICP fell 74%
- CPP rose 27%
- CBF rose 23%
- Peak effect @ 20-60 minutes



7.2% Hypertonic Saline vs 15% Mannitol Solution for Treating ICP in Neurosurgical Patients

Critical Care 2005;9:R530



MYTHBUSTERS



Pascal = N

Crack
MOMENT OF IMPACT

NOT TO SCALE

LAWYER FRAME FOR CORRECT MASS DIST.

56" SHOULDER

SAND BAG ON EVERY THING

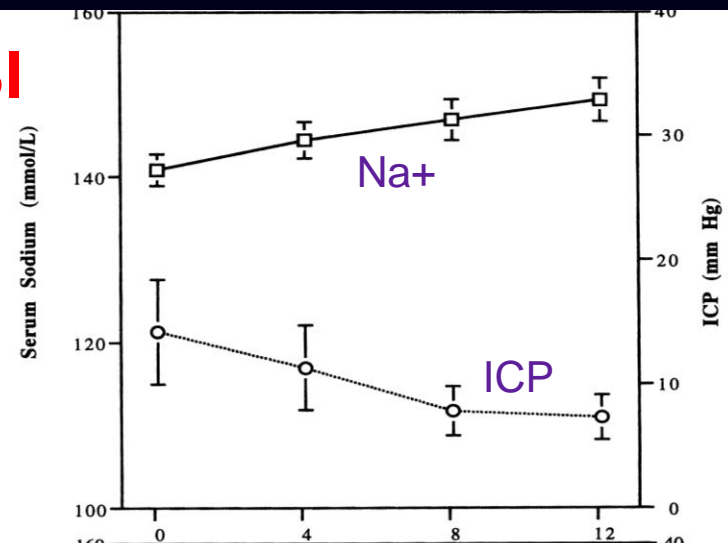


**Slowly raising a sodium level over
days with continuous infusion
hypertonic saline effectively
reduces brain edema**

MYTH

Changes in Sodium concentration & ICP in NICU patients treated with 3% saline solution

TBI



Post-op

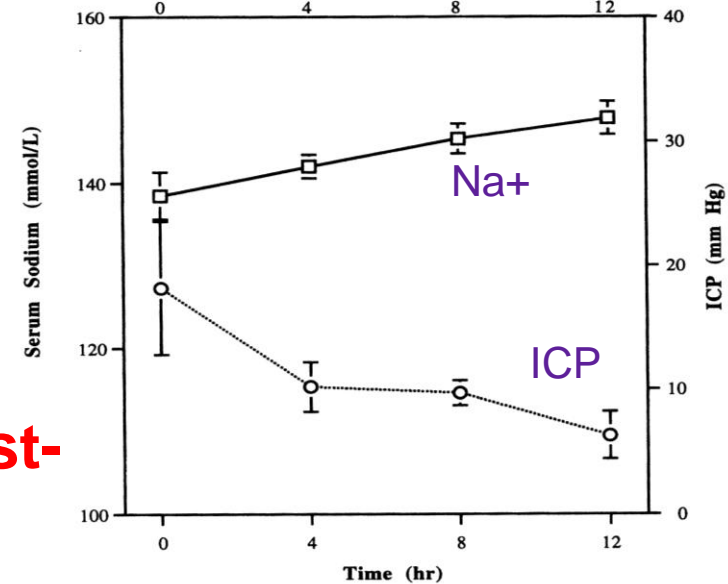
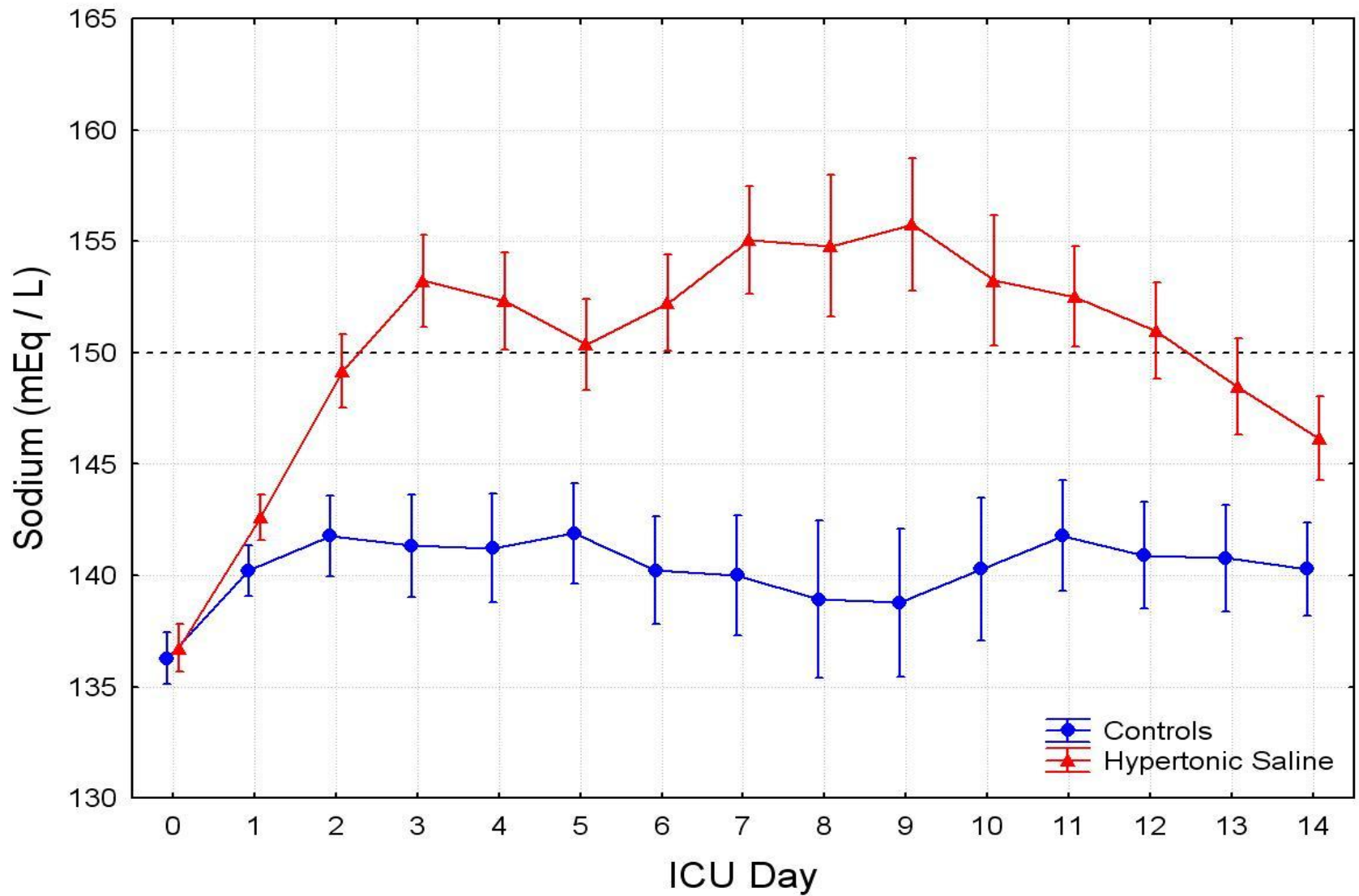


Figure 1. Acute trend in mean intracranial pressure (ICP) and serum sodium concentrations in patients with head injury (*top*), and patients with postoperative cerebral edema (*bottom*). Note the reduction in intracranial pressure associated with increasing serum sodium concentration. Values are expressed as mean \pm SEM. *Open squares* represent serum sodium; *open circles* represent ICP.

Evidence: 3% Saline Infusion

- **Qureshi Al, et al, Crit Care Med 2000;28:3001**
 - ◆ 3% saline reduced ICP and CT midline shift in patients with TBI or neoplasm
 - ◆ Effect not seen with ICH or infarction
- **Fisher, et al, J Neurosurg Anesth 1992;4:4-10**
 - ◆ 3% saline infusion reduced ICP in pediatric TBI patients
 - ◆ Effect modest : 4 mm Hg over 2 hours

3% HTS infusion has a modest and inconsistent effect on ICP



Median (IQR) days to HTS start: 1.0 (0 – 3) days

Median (IQR) duration of HTS use: 10.5 (6 – 14) days

Poor Grade SAH: In-Hospital Complications

	HTS Cases (N= 36)	Controls (N= 57)	<i>P</i>
Neurological			
Herniation	8 (23%)	10 (18%)	0.56
Infarction secondary to vasospasm	6 (17%)	13 (23%)	0.45
Seizures	10 (28%)	8 (14%)	0.11
Medical			
Pneumonia	21 (58%)	14 (25%)	0.001
Arrhythmia	10 (28%)	3 (5%)	0.002
Pulmonary Edema	17 (47%)	10 (18%)	0.003
Congestive Heart Failure	7 (19%)	4 (7%)	0.07
Fever (Temp. >101.5 F)	31 (86%)	47 (83%)	0.64

Hypertonic Saline: Complications

- **Congestive heart failure**
- **Rebound brain edema**
- **Hypokalemia**
- **Hyperchloremic metabolic acidosis**
 - ◆ Use 50/50 chloride/acetate solution
- **Coagulopathy**
- **Central pontine myelinolysis**

5

Hyperventilation

**HYPERVENTILATION IS THE MOST RAPID WAY TO REDUCE ICP
EXCESSIVE HYPERVENTILATION CAN WORSEN CEREBRAL ISCHEMIA**

MECHANISM OF ACTION:

HYPOCARBIA INDUCES SERUM AND CSF ALKALOSIS

ALKALOSIS INDUCES CEREBRAL VASOCONSTRICTION

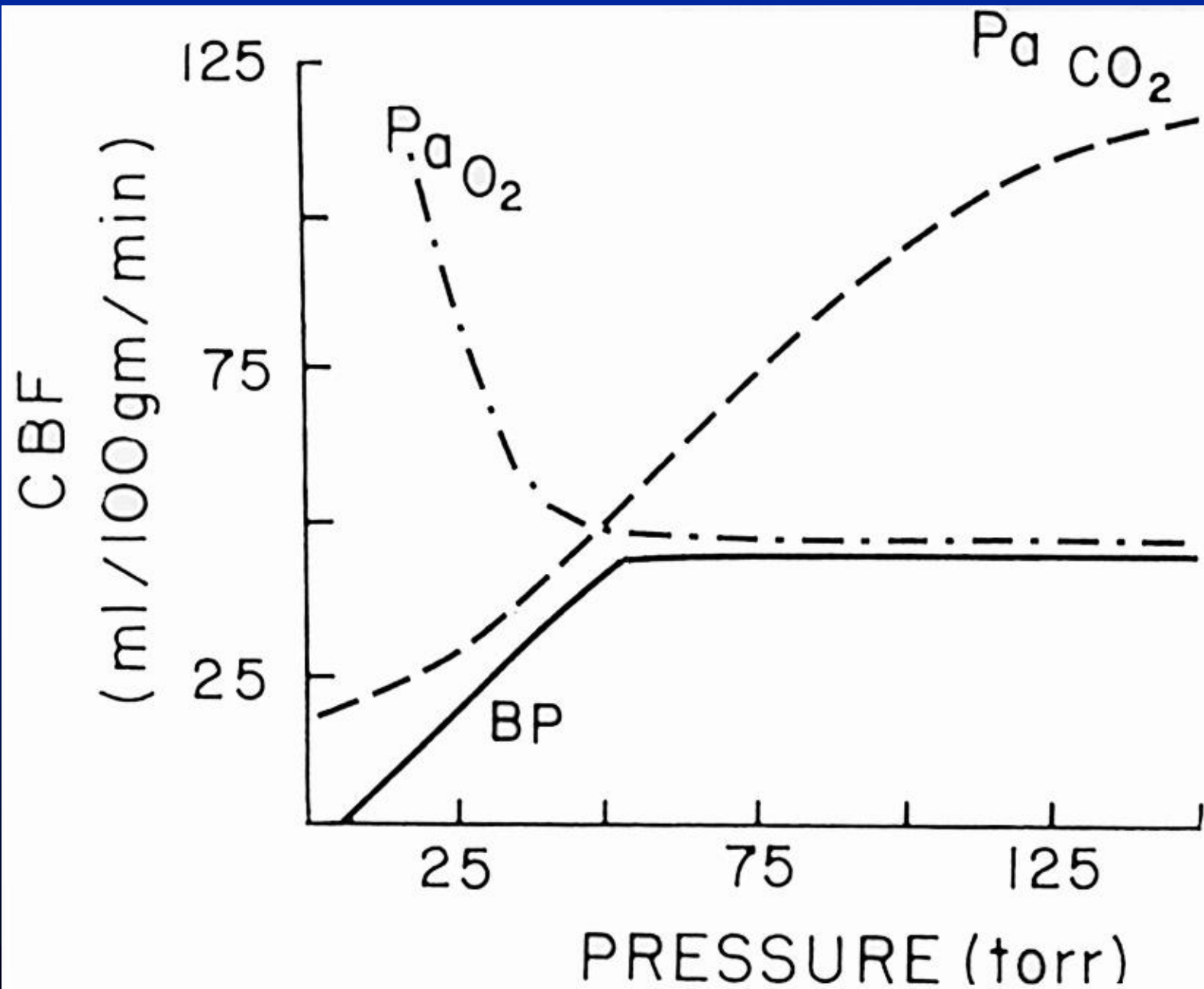
VASOCONSTRICTION REDUCES CEREBRAL BLOOD VOLUME

TIME COURSE:

ICP IS REDUCED ALMOST IMMEDIATELY

PEAK REDUCTION IN 5-10 MINUTES

**Hyperventilation Can Have Sustained Effects In Patients With
Vasodilatory Vasogenic Cerebral Edema**



6

Hypothermia

MECHANISM OF ACTION:

**PROFOUNDLY REDUCES REDUCES CEREBRAL METABOLISM, AND
HENCE CEREBRAL BLOOD VOLUME**

TARGET

32-33° C

INDICATION

PENTOBARBITAL-REFRACTORY ICP

COMPLICATIONS:

ARRYTHMIA AND CARDIOVASCULAR DEPRESSION

IMMUNOSUPPRESSION

COAGULOPATHY

METABOLIC: SHIVERING AND REWARMING







MEDIVANCE ARCTIC SUN

NEVER SLEEP DOWNHILL
NE JAMAIS DORMIR EN
NE JAMAIS APPLIQUER CE COUSSIN SUR
NE JAMAIS APPLIQUER CE COUSSIN SUR
NE JAMAIS APPLIQUER CE COUSSIN SUR

Hypothermia and ICP in TBI

<i>Author</i>	<i>N</i>	<i>Year</i>	<i>Effect on ICP</i>
Clifton	42	1993 JNT	No difference compared to controls
Marion	43	1993 JNS	Reduced from baseline by 40%
Metz	10*	1997 JNS	Reduced from baseline
Marion	82	1997 NEJM	ICP lower (15 vs 20 mm Hg) and CPP higher; but reversed with rewarming
Jiang	87	2000 JNS	Reduced from baseline (29 vs 19 mm Hg) at day 7
Shiozaki	91	2001 JNS	No effect on concurrent therapeutic intensity for ICP management
Clifton	392	2001 NEJM	Reduced proportion with ICP >30 mm Hg on day 2 (14% vs. 28%)

Contemporary Management of Traumatic Intracranial Hypertension: Is There a Role for Therapeutic Hypothermia?

Matthew Schreckinger · Donald W. Marion

Table 3 Comparison of contemporary therapies for intracranial hypertension

Therapy	Total number of patients	Average decrease in ICP	Standard deviation
Hyperventilation	126	6.08	4.22
Mannitol	140	7.93	5.34
Barbiturates	167	8.47	6.71
Hypothermia	367	9.97	6.66
Hypertonic saline	133	15.06	7.34
CSF drainage	72	15.45	4.67
Decompressive craniectomy	192	19.15	7.70

Hypothermia for Barbiturate Refractory ICP in TBI: *TITRATION of Cooling*

- **Tateshi (Neurosurgery 1998)**
 - Titrated the depth of cooling between 33-36 ° C to keep ICP <20 mm Hg in 9 patients refractory to thiopental.
 - All patients responded:
 - Mean brain reduction of temperature of 2° C
 - Mean reduction of ICP from 24 to 15 mm Hg.
 - The frequency of infectious and hematologic complications in this relatively small series was still high.

7

High-Dose Barbituates

MECHANISM OF ACTION:

PROFOUNDLY REDUCES REDUCES CEREBRAL METABOLISM, AND HENCE CEREBRAL BLOOD VOLUME

PENTOBARBITAL:

LOADING DOSE = 5-20 mg/kg IV OVER 1 mg/kg/min

MAINTAINANCE DRIP = 1-4 mg/kg/hr

GOAL: NORMAL ICP, BURST SUPPRESSION ON EEG

COMPLICATIONS:

MYOCARDIAL DEPRESSION AND HYPOTENSION

NEUROLOGICAL EXAM IS OBSCURED

ILEUS

BLOOD STREAM INFECTION

ICP CONTROL: TRAUMATIC BRAIN INJURY

- *Shiozaki (J Neurosurg 1993)*
 - Randomized controlled study of hypothermia (34° C) for ICP refractory to pentobarbital (N=33).
 - Hypothermia resulted in:
 - » Lower ICP (36.9 to 26.5 mm Hg)
 - » Increased CPP
 - » Reduced CBF and CMRO₂
 - » Reduced arteriojugular venous oxygen differences
 - Survival was 50% in hypothermia patients compared to 18% in the control group (P<.05).

HYPOTHERMIA IS

WIRED

PENTOBARBITAL IS



Complications of pentobarbital

- Hypotension
- Immunosuppression
- **An extra 4 weeks in coma**
- **ICU neuromyopathy**

Re-Revised
Columbia
Stepwise ICP
Protocol

7 PENTOBARBITAL

6 HYPOTHERMIA

5 HYPERVENTILATION

4 OSMOTHERAPY

3 CPP OPTIMIZATION

2 SEDATION

1 SURGICAL DECOMPRESSION

Re-Revised
Columbia
Stepwise ICP
Protocol

⑥ HYPOTHERMIA

⑤ HYPERVENTILATION

④ OSMOTHERAPY

③ CPP OPTIMIZATION

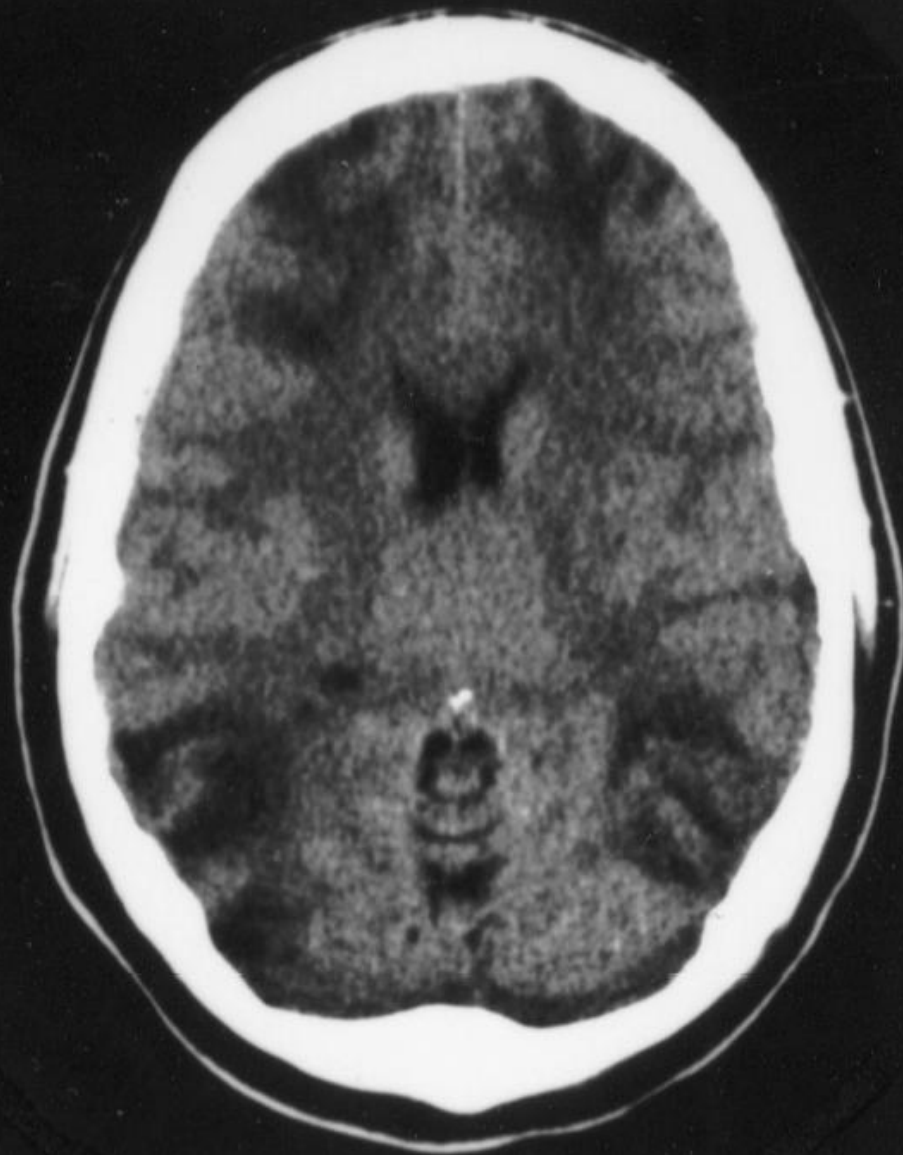
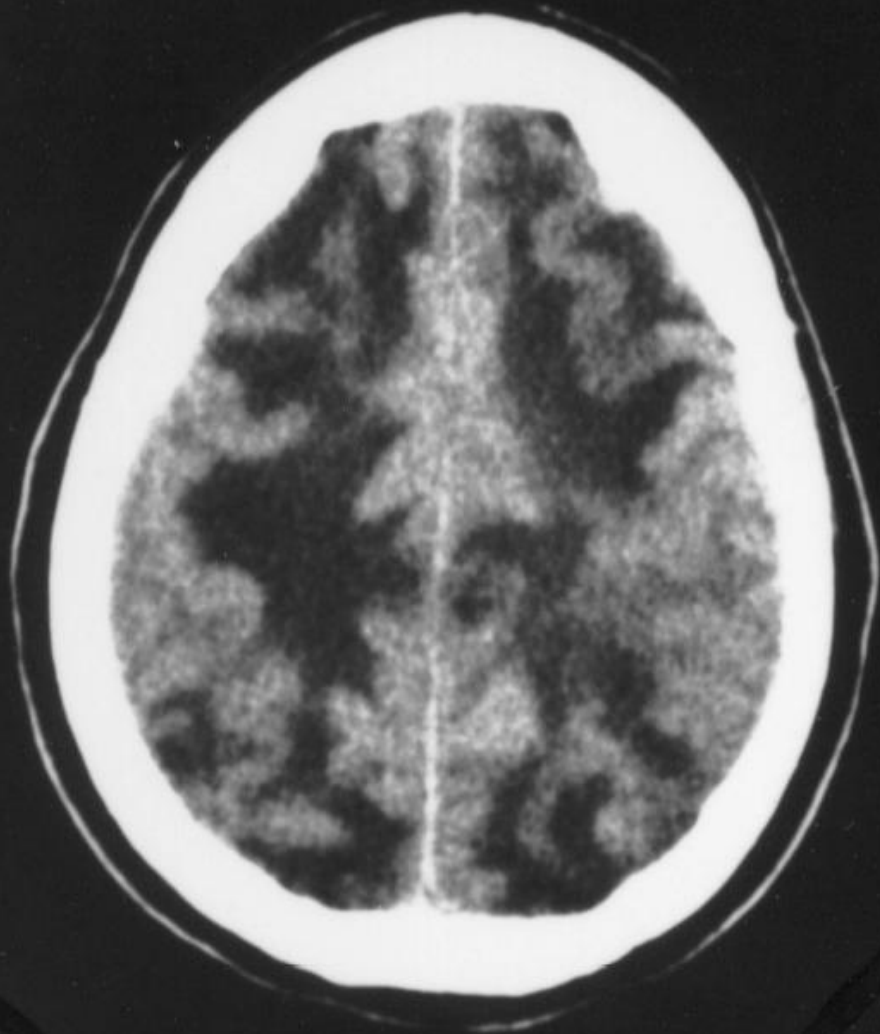
② SEDATION

① SURGICAL DECOMPRESSION

ICP Control in Action

Neuro-ICU: July 4, 1995

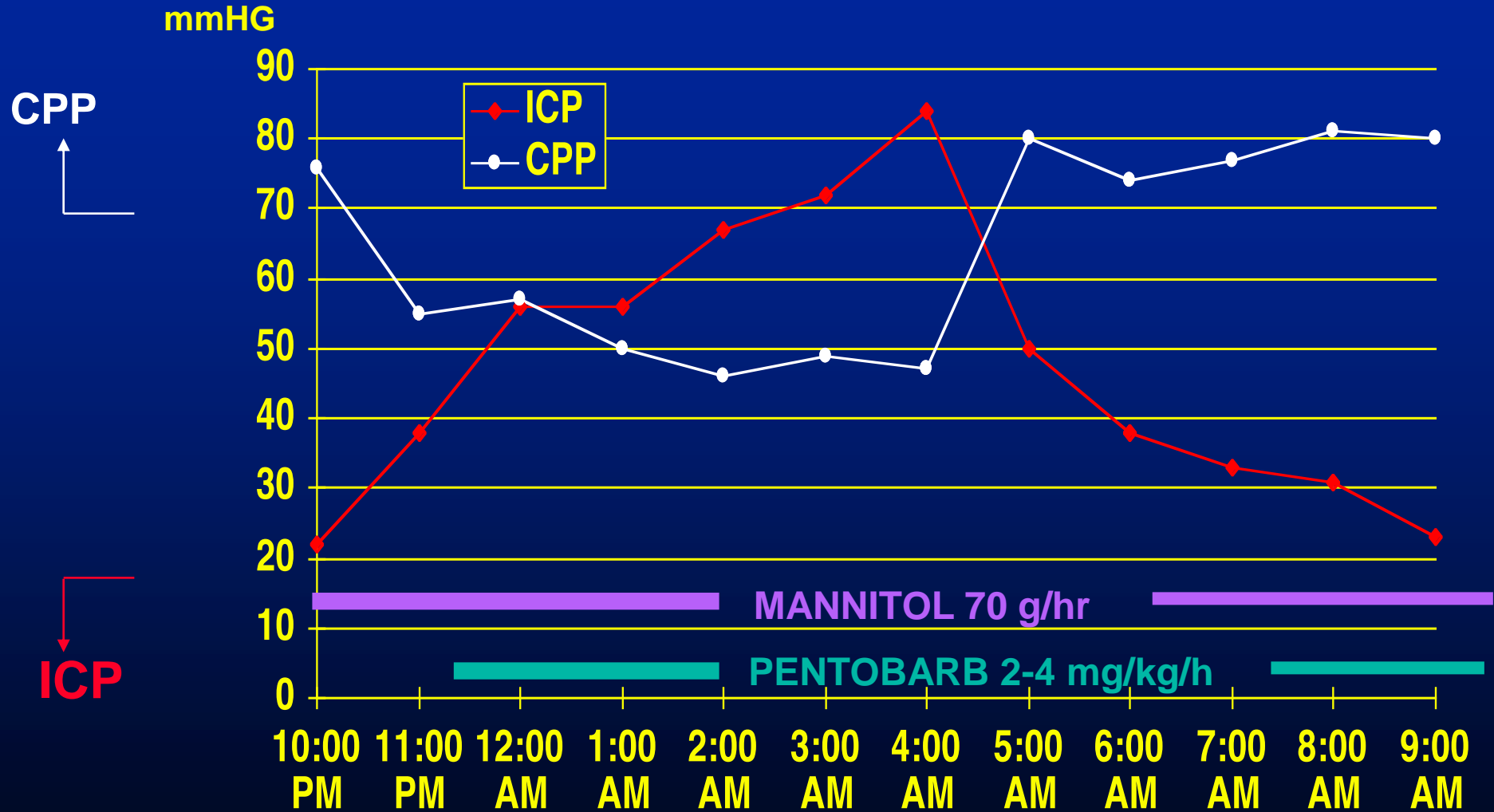
- **Healthy 35 yr old woman @ 34 wks gestation with pre-eclampsia and HELLP syndrome**
- **July 31: Rx Mg sulfate and emergent delivery**
- **Aug 1: At 8:00 AM, sudden HA and lethargy, progressing rapidly to deep coma**
- **CT SCAN: diffuse cerebral edema**



Neuro-ICU: On Arrival

- 2:00 PM: Arrival in deep coma with intact brainstem reflexes
- 4:00 PM: BP 100/65, Rx dopamine
- 7:00 PM: ICP monitor inserted, ICP 22 and CPP 76 mmHg
- 11:00 PM: ICP 38, CPP 55 mmHg, mannitol started...

Early Day 2: Massive Elevation of ICP



Neuro-ICU: Day 2

- EXAM: Sluggish pupils and corneals, no EOMs to ice water, no withdrawal to deep pain
- EEG: Flatline
- TCD: Hi velocity (90-100 cm/s), low PI
- LABS: Osms 401, Hct 17%, Na⁺ 121

Neuro-ICU: Day 3

- Oliguric renal failure 2° mannitol
- Hemodialysis x 1 = good response

Subsequent Course

- Day 4: GI bleed (Tx 5 units), LLL pneumonia with large Aa gradient
- Day 5: Post ATN diuretic phase, increased Na⁺ 152
- Day 6: ICP = 6 mmHg, light coma
- Day 7: ICP monitor d/c' ed
- Day 8: HR 145, new inverted T waves
- Day 12: LUE DVT 2° central line (positive lupus A/C noted)

Later Hospital Course

- **Day 14**: Looks at a voice, grimaces and frowns, bibrachial paresis, central hyperventilation
- **Day 21**: Rarely obeys commands
- **Day 27**: EF 30% on echo
- **Day 30**: Consistently obeys commands, anarthric with pseudobulbar affect
- **Day 50**: Talking, laughing, writing messages, walking with assistance

0 22 40

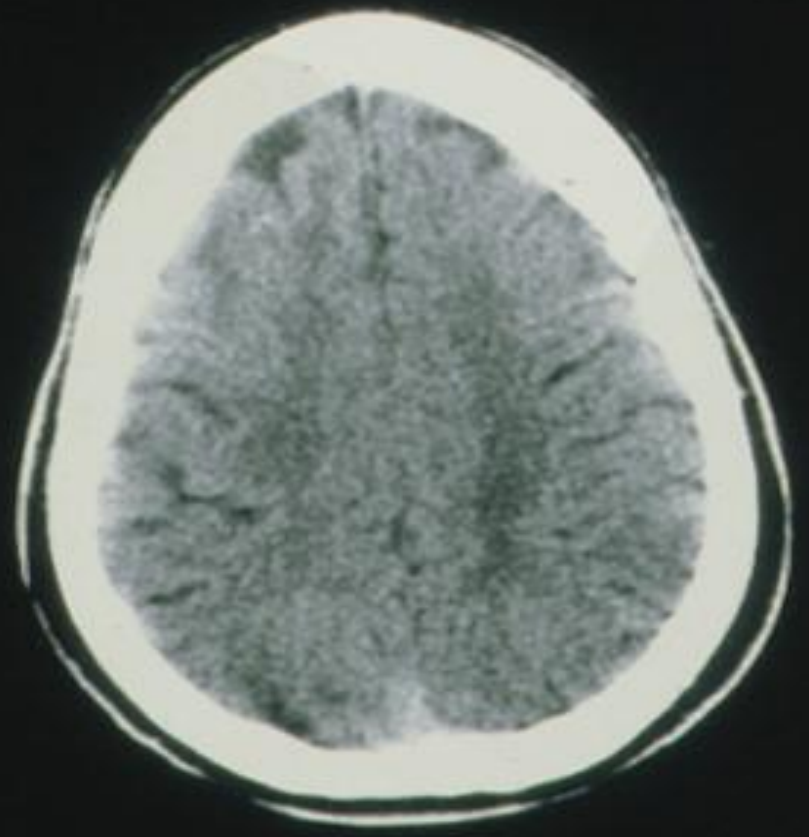
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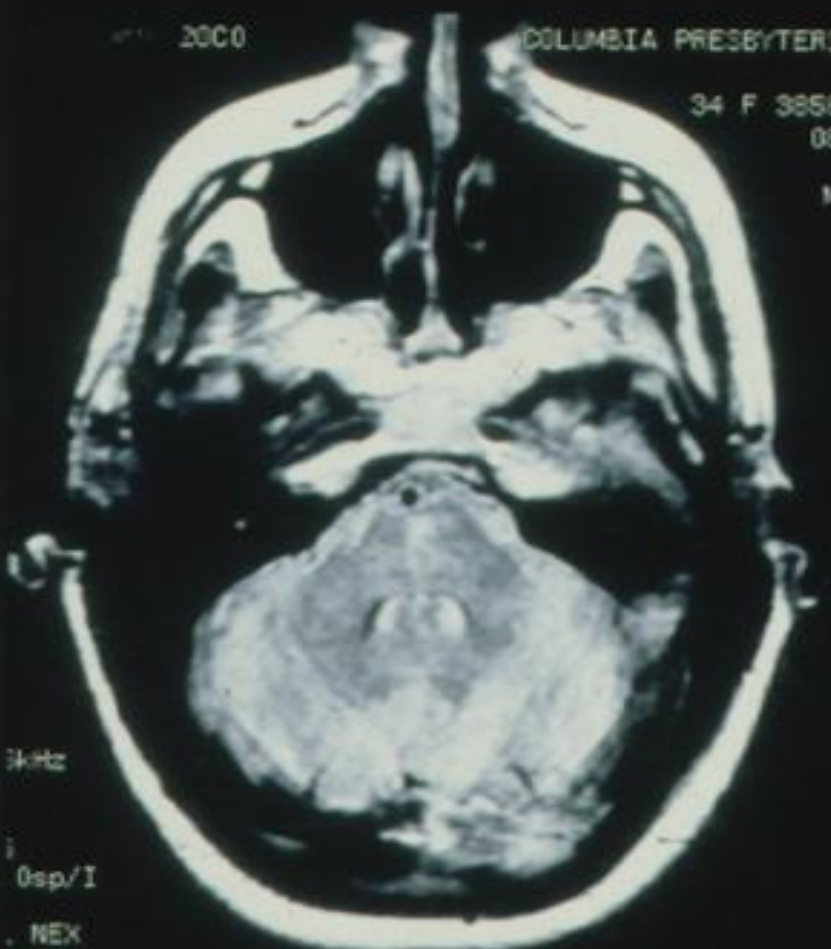
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COLUMBIA PRESBYTERIAN MR2

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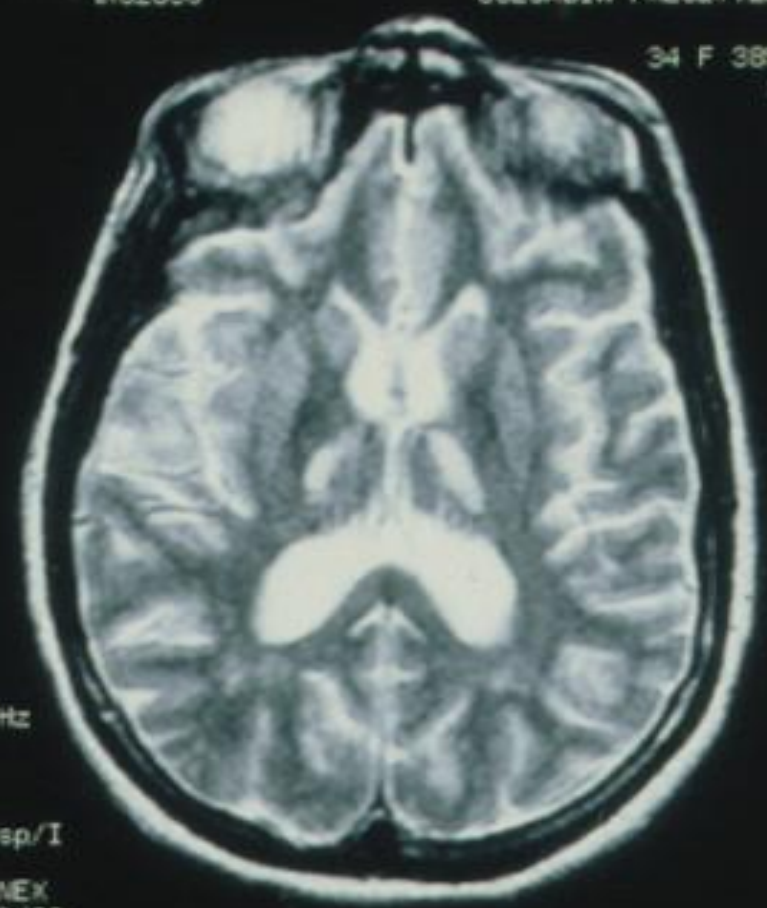
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COLUMBIA PRESBYTERIAN M

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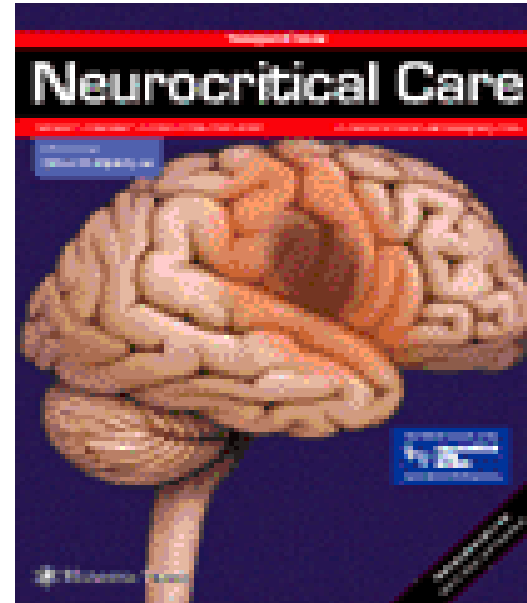
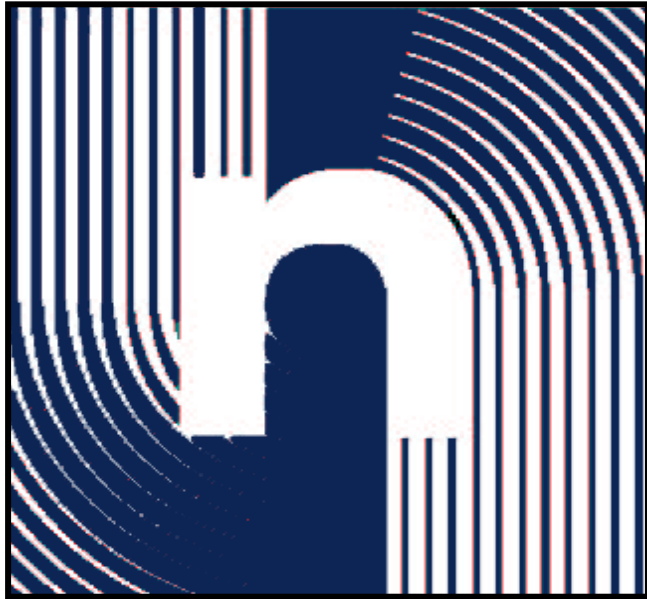
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:I/ED/30
L = 215

V

3 Month Outcome

- **Home with family and children**
- **Functionally independent, doing light housework, in outpatient rehab**
- **Mild deficits in memory, concentration, and visual-spatial function**
- **Mild spastic left hemiparesis**
- **Cerebellar dysarthria**
- **Mild dysmetria**





Neurocritical Care Society
www.neurocriticalcare.org