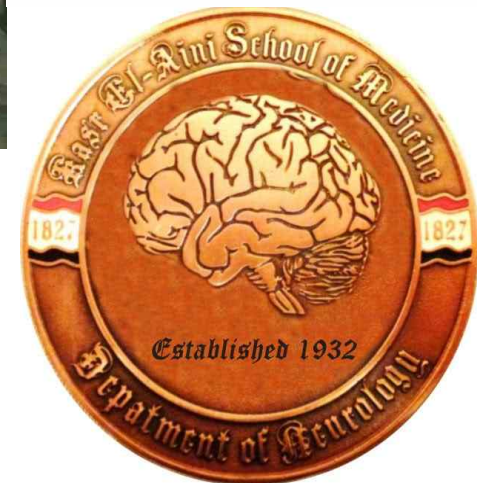




INCREASED ICP

Amr Hassan, M.D.
Associate Professor of Neurology
Cairo University



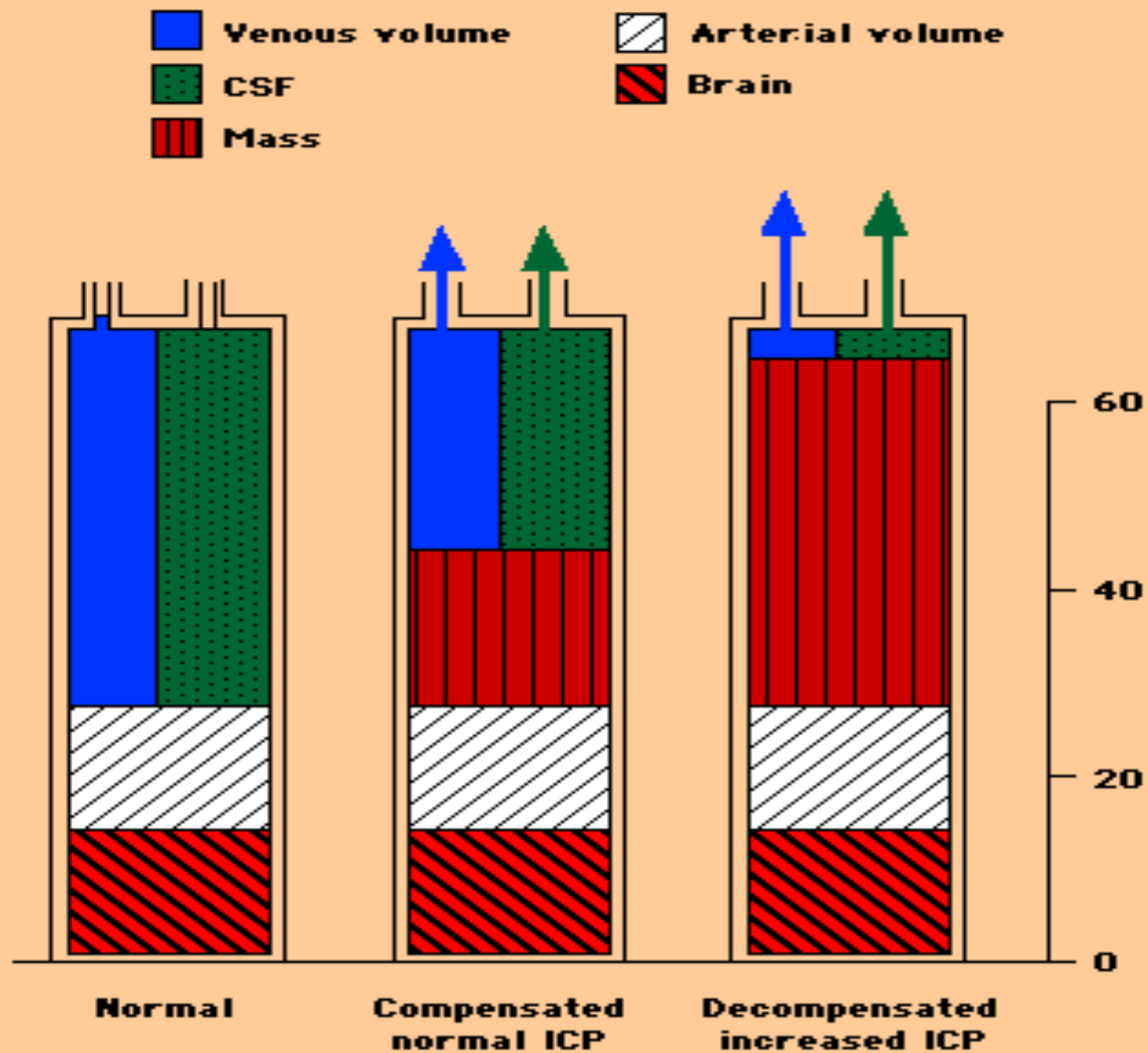
INCREASED ICP

Outline

- **Anatomy of the intracranial vault**
- **Physiology: CBF and CPP**
- **Pathophysiology**
- **Monitoring**
- **Treatment**

Intracranial Vault

- **Bony structure**
- **Brain & interstitial fluid** 80%
- **Blood (CBV)** 10%
- **CSF** 10%



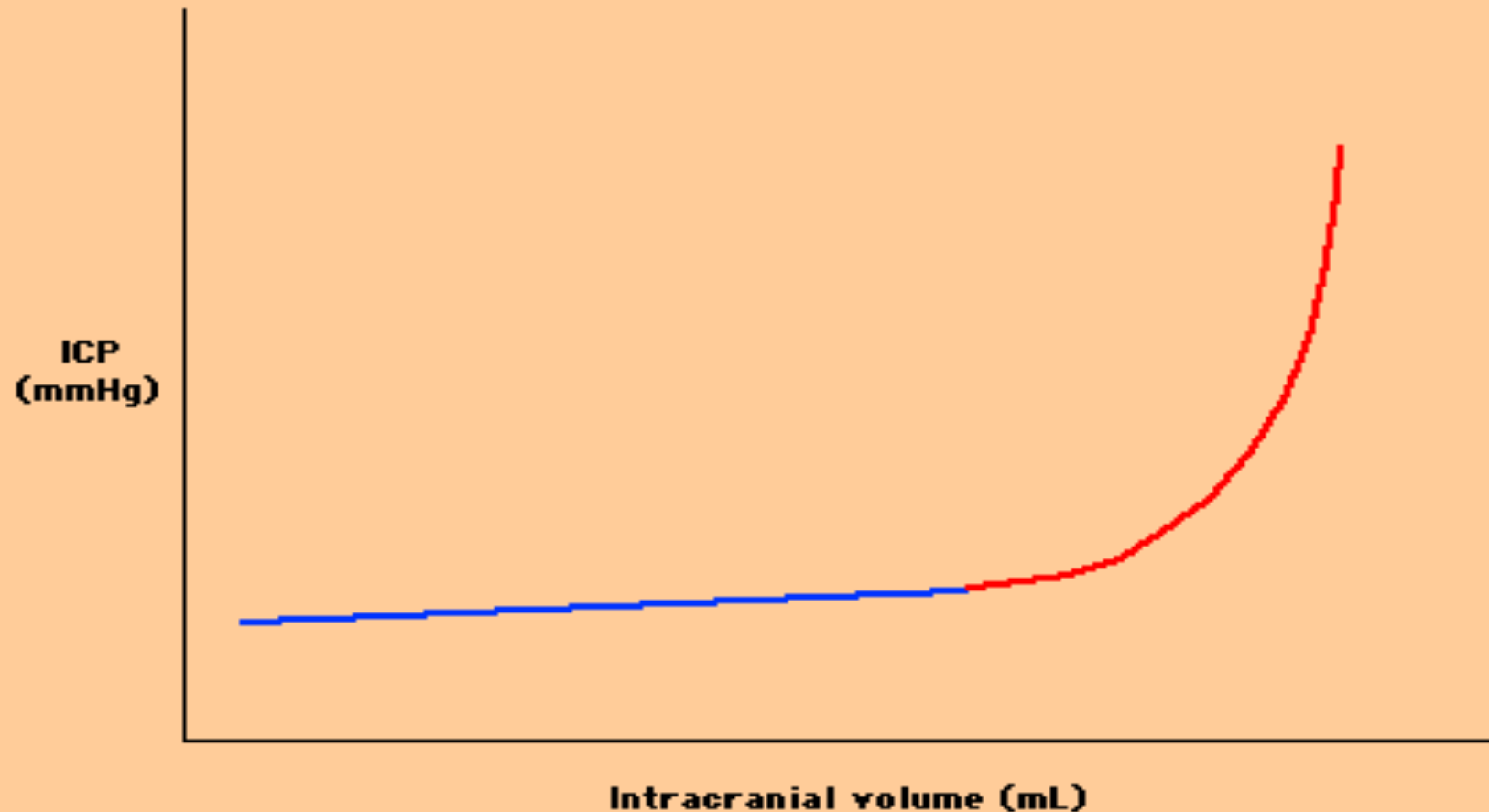
Intracranial compensation for an expanding mass lesion

Data from Pathophysiology and management of the intracranial vault. In: Textbook of Pediatric Intensive Care, 3rd ed, Rogers, MC (Ed), Williams and Wilkins 1996. p. 646; figure 18.1.

Monroe- Kellie Doctrine

Because of a rigid skull, the intracranial contents cannot expand significantly.....

Intracranial Pressure-Volume Relationship



The relationship between intracranial volume and pressure is nonlinear An initial increase in volume results in a small increase in pressure because of intracranial compensation (blue line). Once intracranial compensation is exhausted, additional increases in intracranial volume result in a dramatic rise in intracranial pressure (red line).

Intracranial vault

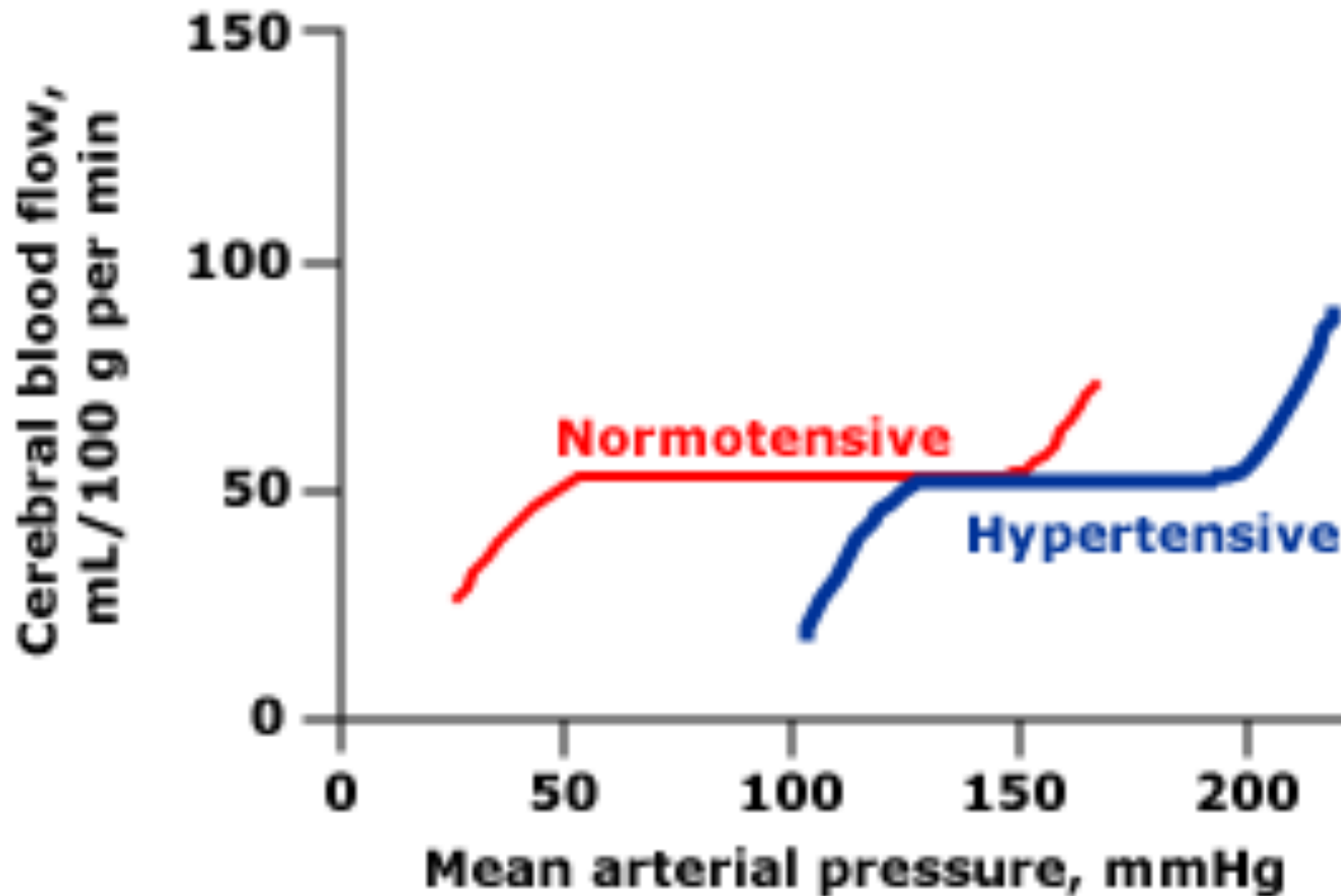
- Brain
- Blood
- CSF

Cerebral Blood Flow

- Amount of blood in transit through brain
- Certain CBF for given pressure gradients and given metabolic state
- CBF is not altered by compartment size
- Since vasculature resides in rigid skull, it is possible to increase CBV and ICP and have decreased CBF

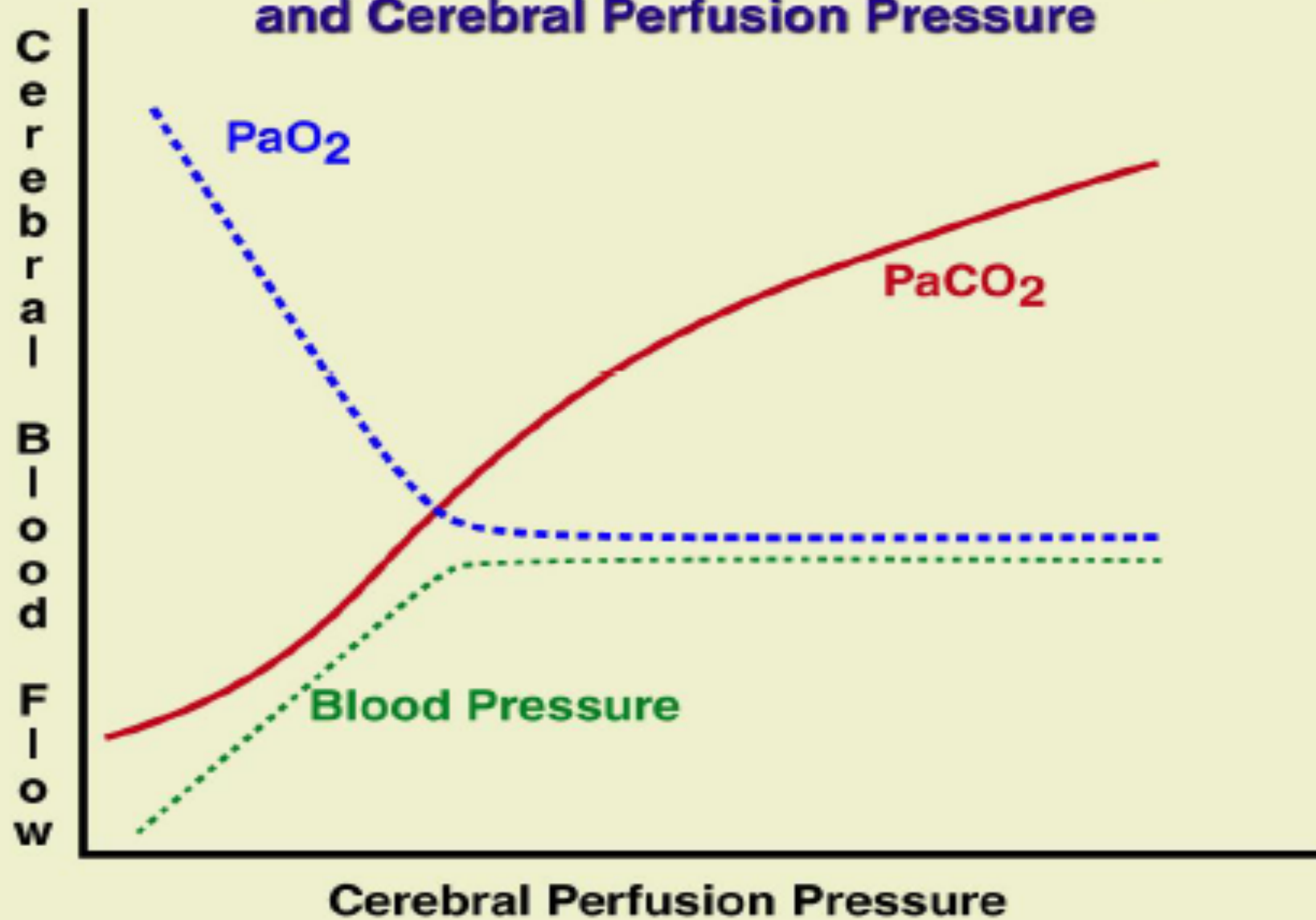
Cerebral Blood Flow

- **Blood supply matches metabolic needs**
- **Regulated:**
 - **Mechanically – metabolic by-products which alter blood vessel caliber**
 - **By sensitivity to CO₂ and O₂**
 - **By adenosine and oxygenases**
 - **Perfusion pressure**



Kaplan, NM, Lancet 1994; 344:1335.

Changes in CBF due to PaCO₂, PaO₂, and Cerebral Perfusion Pressure



Cerebral Perfusion Pressure

- **CBF = inflow - outflow**
- **CPP more sophisticated measure due to the inclusion of a third pressure → CSF pressure**

Cerebral Perfusion Pressure

- $CPP = \text{inflow} - \text{outflow}$
- $CPP = P \text{ carotid} - P \text{ intracranial}$
or
 $P \text{ carotid} - P \text{ jugular}$
- $CPP = MAP - ICP$

Autoregulation

- CBF is regulated over a wide range of MAP
- Range of 60-150 mmHg
- Regulated by the tone of small arteries and arterioles and by Blood Brain Barrier (BBB)

Causes of Intracranial Hypertension

Intracranial hemorrhage

 Traumatic brain injury

 Ruptured aneurysm

 Arteriovenous malformation

 Other vascular anomalies

Central nervous system infections

Neoplasm

Vasculitis

Ischemic infarcts

Hydrocephalus

Pseudotumor cerebri

Idiopathic

PATHOPHYSIOLOGY

- **Primary injury**
 - parenchymal injury
- **Secondary injury**
 - reaction of neural tissue to primary injury
 - ◆ edema
 - ◆ cell death

Pathophysiology

■ Cerebral Edema

- ◆ increase in brain volume
- ◆ increase in Na⁺ and H₂O

Classification of Cerebral Edema

- **Interstitial**
- **Vasogenic**
- **Cytotoxic**

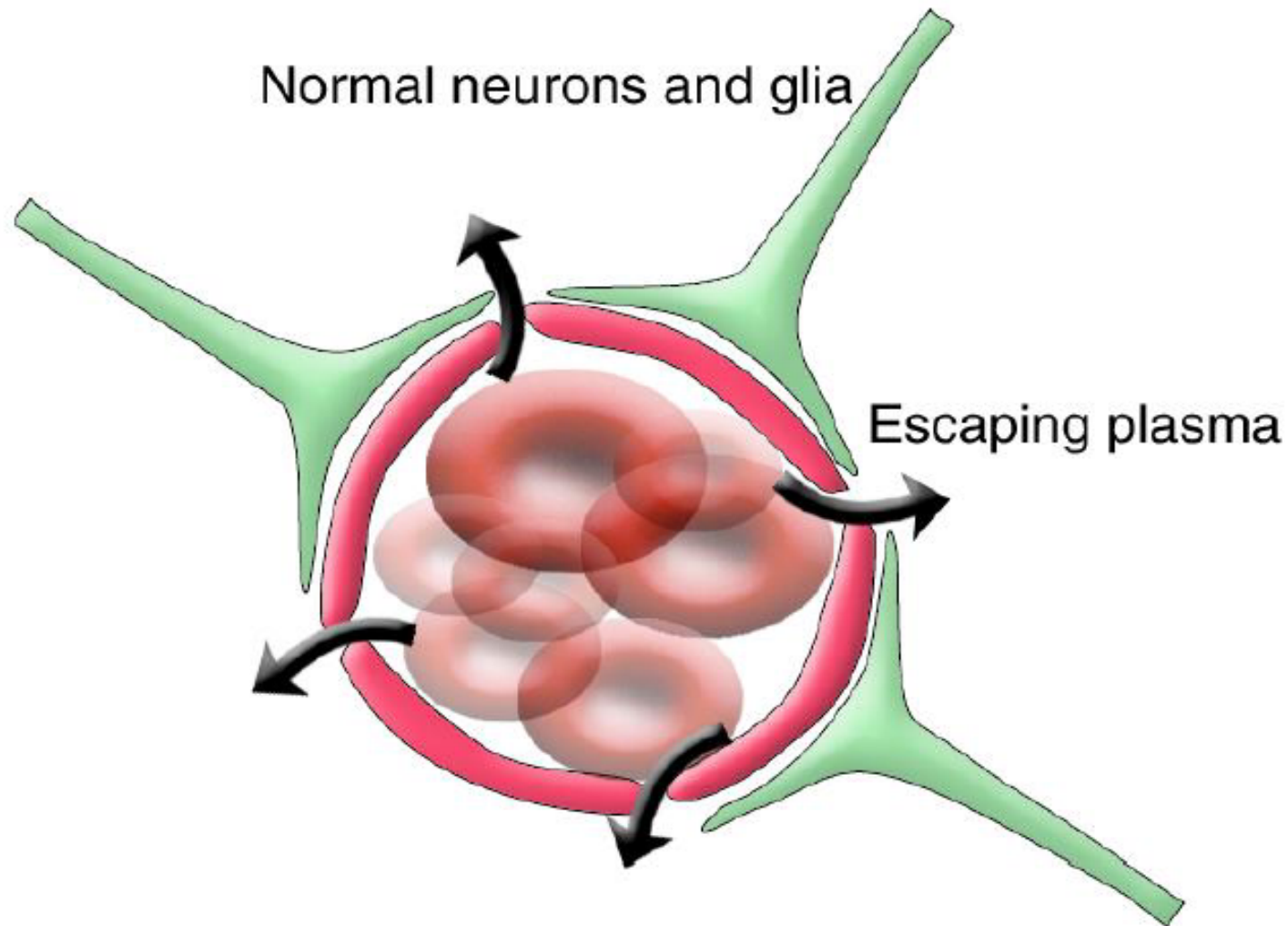
Interstitial Edema

- Increased CSF hydrostatic pressures
- Altered absorption of CSF
- Increased edema of periventricular white matter due to CSF movement across ventricles.
- Prototype
 - obstructive hydrocephalus

Vasogenic Edema

- Increased permeability of brain capillary endothelial cells to macromolecules.
- Neurons are not primarily injured

Vasogenic Edema



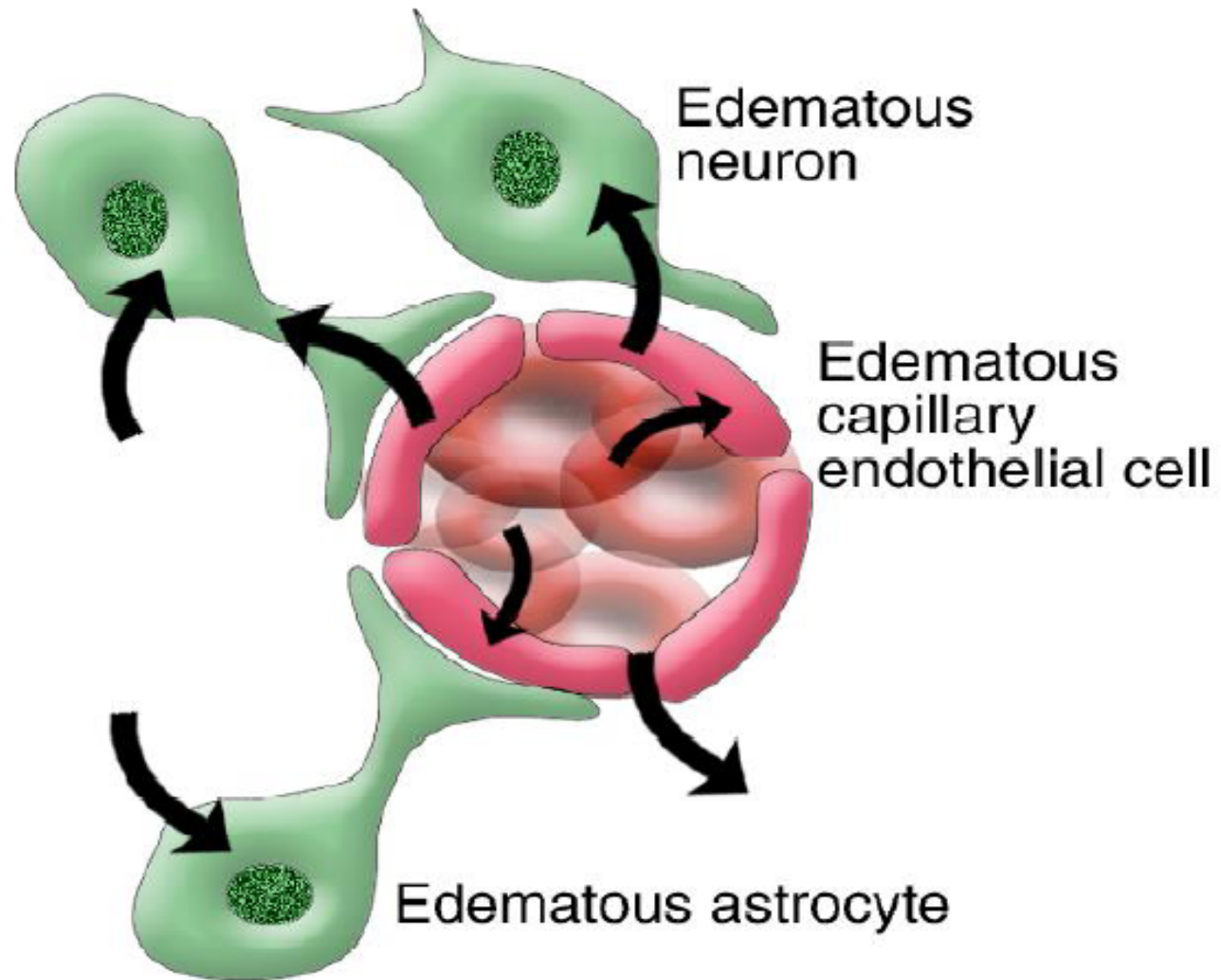
Vasogenic Edema

- Tumor
- Abscess
- Hemorrhage
- Contusion
- Infarction
- Meningitis
- Lead encephalopathy

Cytotoxic Edema

- Cellular swelling due to cell injury
 - neuronal, glial, and endothelial
- Failure of ATPase dependant Na exchange
- Edema is a reflection of cell death rather than a contributing factor

Cytotoxic Edema



Cytotoxic Edema

- HIE
- Re-perfusion injury
- Osmotic disequilibrium

Symptoms of Increased ICP

- Headache
- Bulging fontanel
- Papilledema
- Altered mental status
- Neurological deficit
 - common is 3rd nerve palsy
 - dilated pupil(s)



Papilledema Papilledema, characterized by blurring of the optic disc margins, loss of physiologic cupping, hyperemia, and fullness of the veins, in a five year old girl with intracranial hypertension due to vitamin A intoxication. Courtesy of Gerald Striph, MD.

Glasgow Coma Scale

Eye opening

Spontaneous	4
Response to speech	3
Response to pain	2
None	1

Best verbal response

Oriented	5
Confused	4
Inappropriate	3
Incomprehensible	2
None	1

Best motor response

Obeying	6
Localizing	5
Withdrawing	4
Flexing	3
Extending	2
None	1

Children's Coma Scale for Ages ≤3 years (Modified Glasgow Coma Scale)[†]

Eye Opening

Spontaneous	4
Response to verbal command	3
Response to pain	2
None	1

Best Motor Response

Spontaneous (obeys verbal command)	6
Localizes pain	5
Withdraws in response to pain	4
Abnormal flexion in response to pain (Decorticate posture)	3
Abnormal extension in response to pain (Decerebrate posture)	2
None	1

Best Verbal Response

Smiles, oriented to sound, follows objects, interacts	5
---	---

Crying

Consolable	
Inconsistently consolable	
Inconsolable	
None	

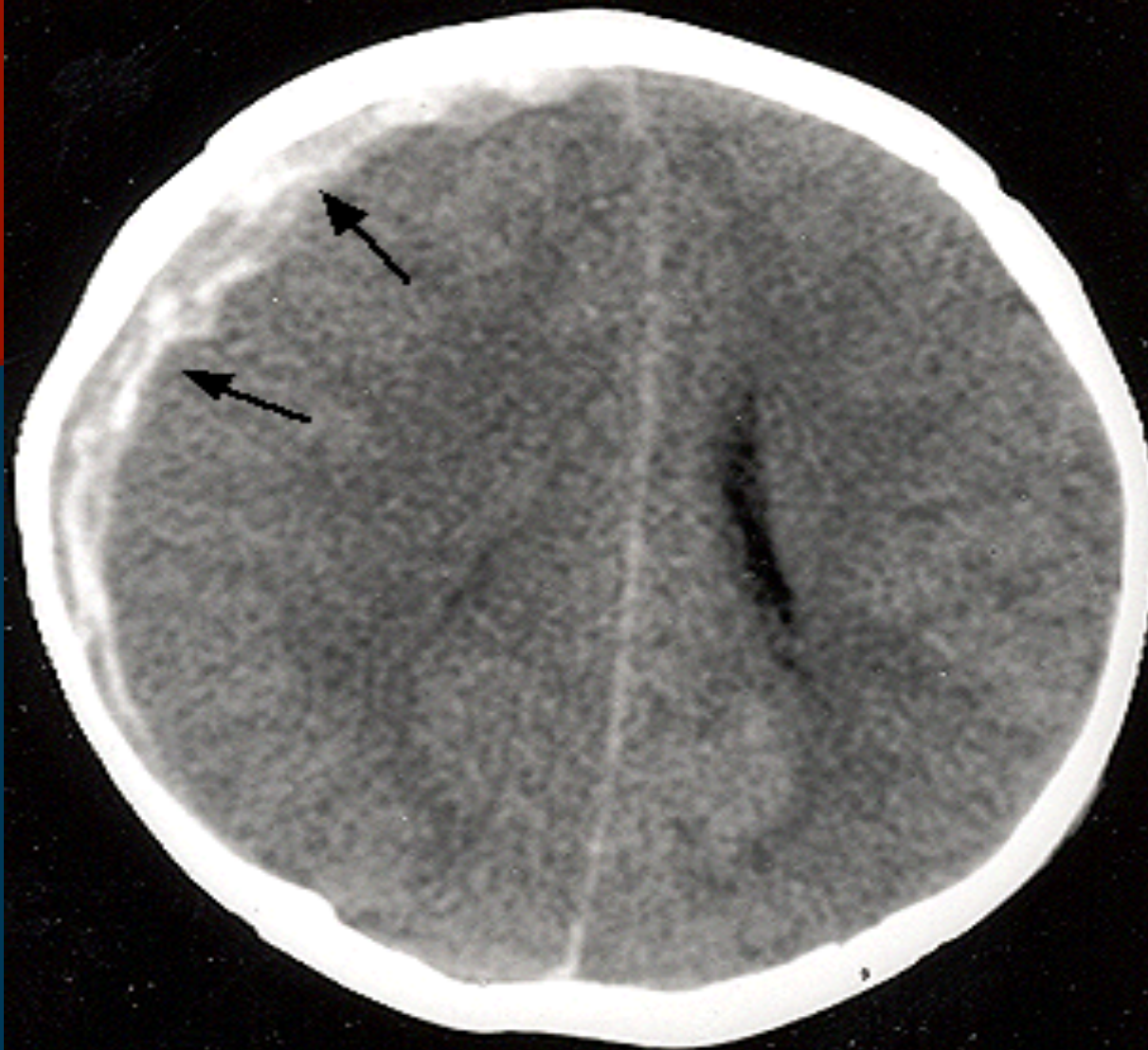
Interacts

Inappropriate	4
Moaning	3
Irritable, restless	2
None	1

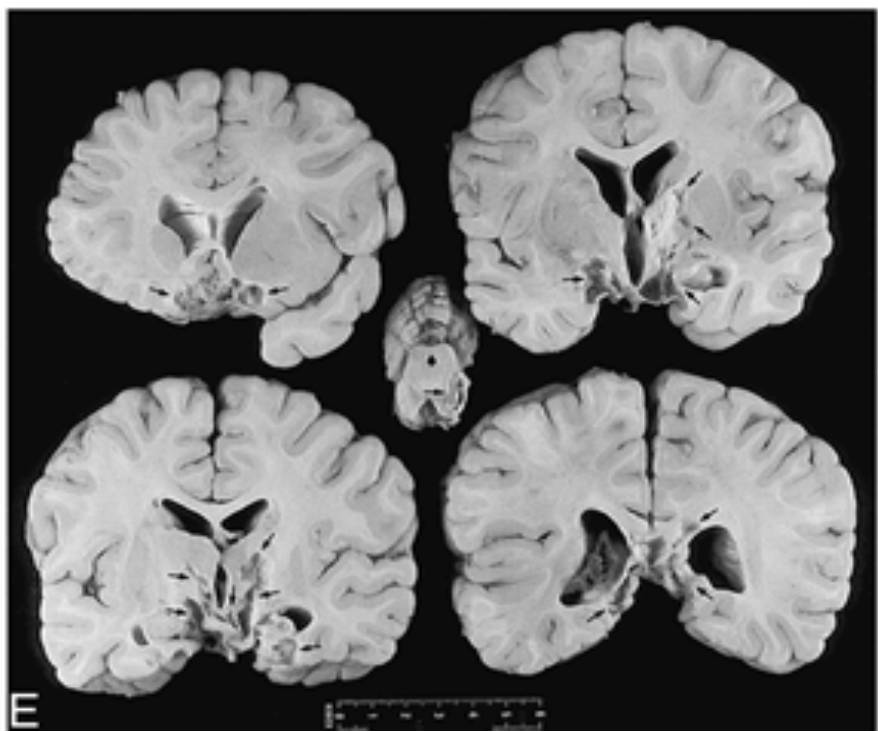
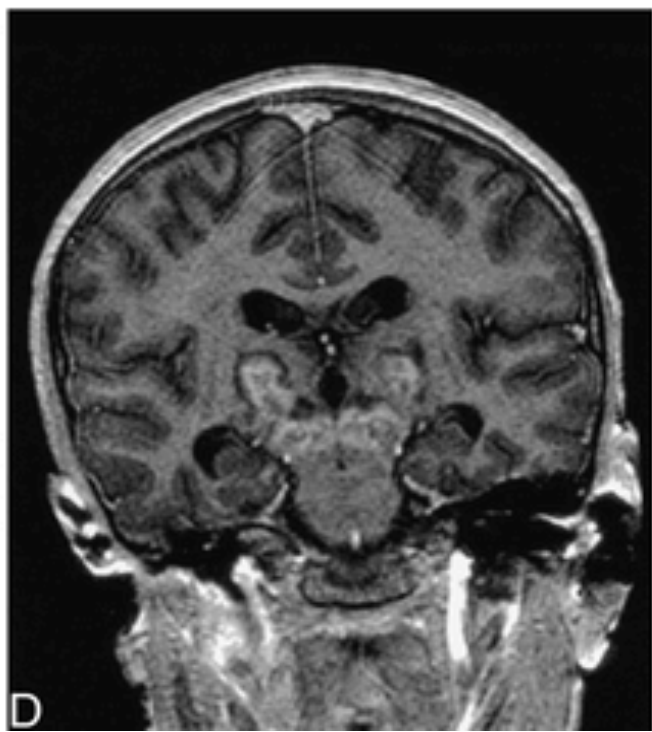
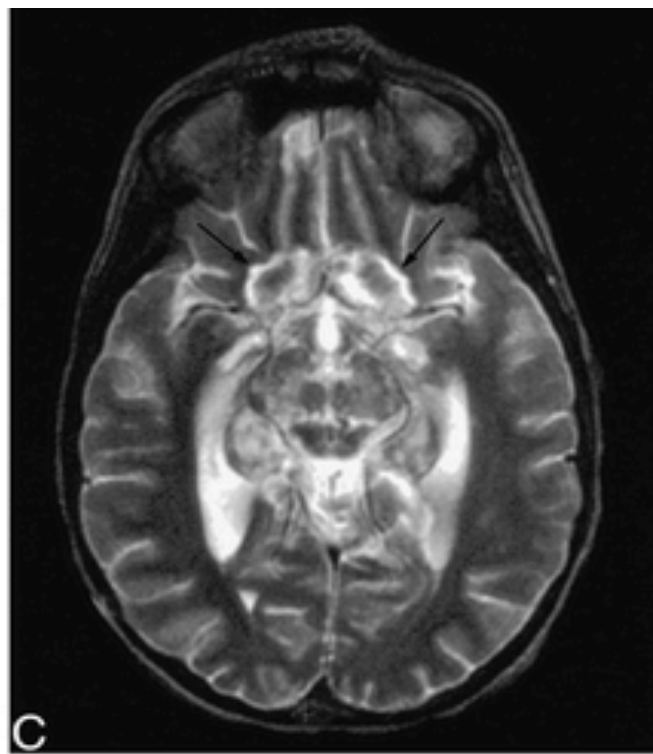
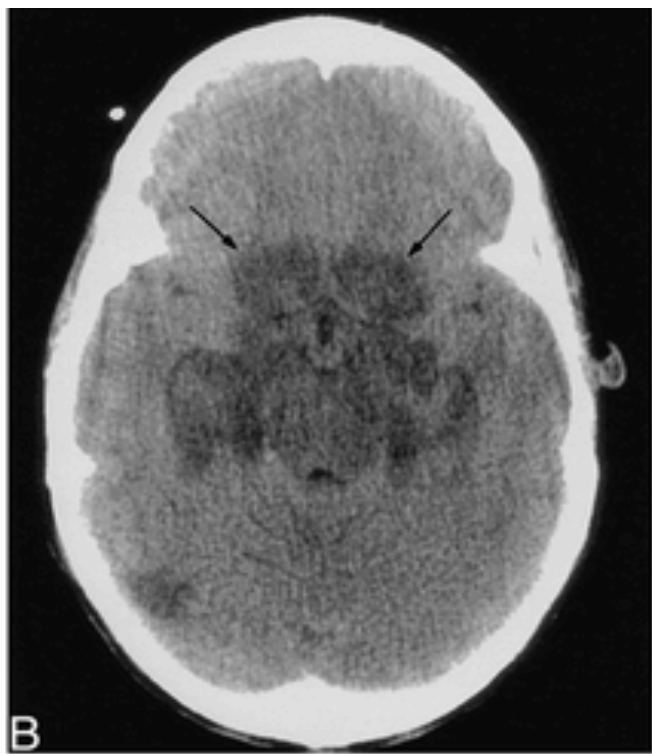
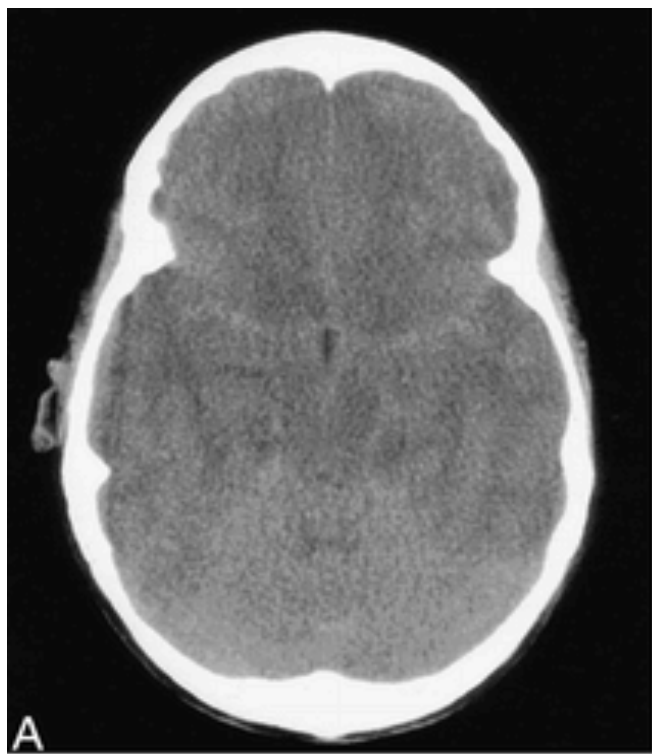
CGS Total

3 to 15

[†] Courtesy of John P Laurent, MD.



Subdural hemorrhage and cerebral edema
CT image (axial view) of acute-hyperacute right subdural hemorrhage (arrows) and asymmetric cerebral edema in an eight-month-old male infant who sustained nonaccidental injury (child abuse).



Increased ICP

Try to prevent

- **Primary injury**
 - parenchymal damage
- **Secondary injury**
 - reaction of neural tissue to injury
 - ◆ edema
 - ◆ cell death

Factors That Worsen Secondary Injury

- ↓ BP
- ↓ PaO₂
- ↑ PaCO₂

INCREASED ICP Monitoring

Non-Invasive

- Assess perfusion
- BP measurement
- Pulse Oximetry

Invasive

- Intubation
- A-line
- CVP
- Jugular venous bulb
- ABG'S, LYTES, OSM
- ICP device

INCREASED ICP

General Care

- HOB elevated 30° → ↑ venous drainage
- Head midline → ↑ venous drainage
- No jugular catheters → prevent venous obstruction
- Normothermia → avoid ↑ metabolism
- ↓ Pleural pressures (zero peep) → ↑ venous drainage

INCREASED ICP

Sedation

- Prevents \uparrow BP \rightarrow \uparrow ICP
- Maintenance of artificial airway
- Prevents agitation

INCREASED ICP

Glucocorticoids

Useful in peritumoral and intratumoral edema

- Improves tumor glucose utilization, decreasing necrosis and edema formation.
- Phospholipase A2 activity is blocked , less arachidonic acid is formed and PG, TXns, and LTs thus less endothelial permeability.
- Inhibits inflammatory cell lysozyme thus decreasing inflammatory cells.

Indications for ICP monitoring

- **GCS \leq 8**
- **Cisterns compressed or absent**
- **Midline shift $>$ 5mm**
- **Post surgical removal of intracranial hematoma**
- **Less severe brain injury in a setting that requires deep sedation or anesthesia**

ICP Monitoring Devices

Location

- Ventricular
- Parenchymal
- Subarachnoid
- Subdural
- Epidural

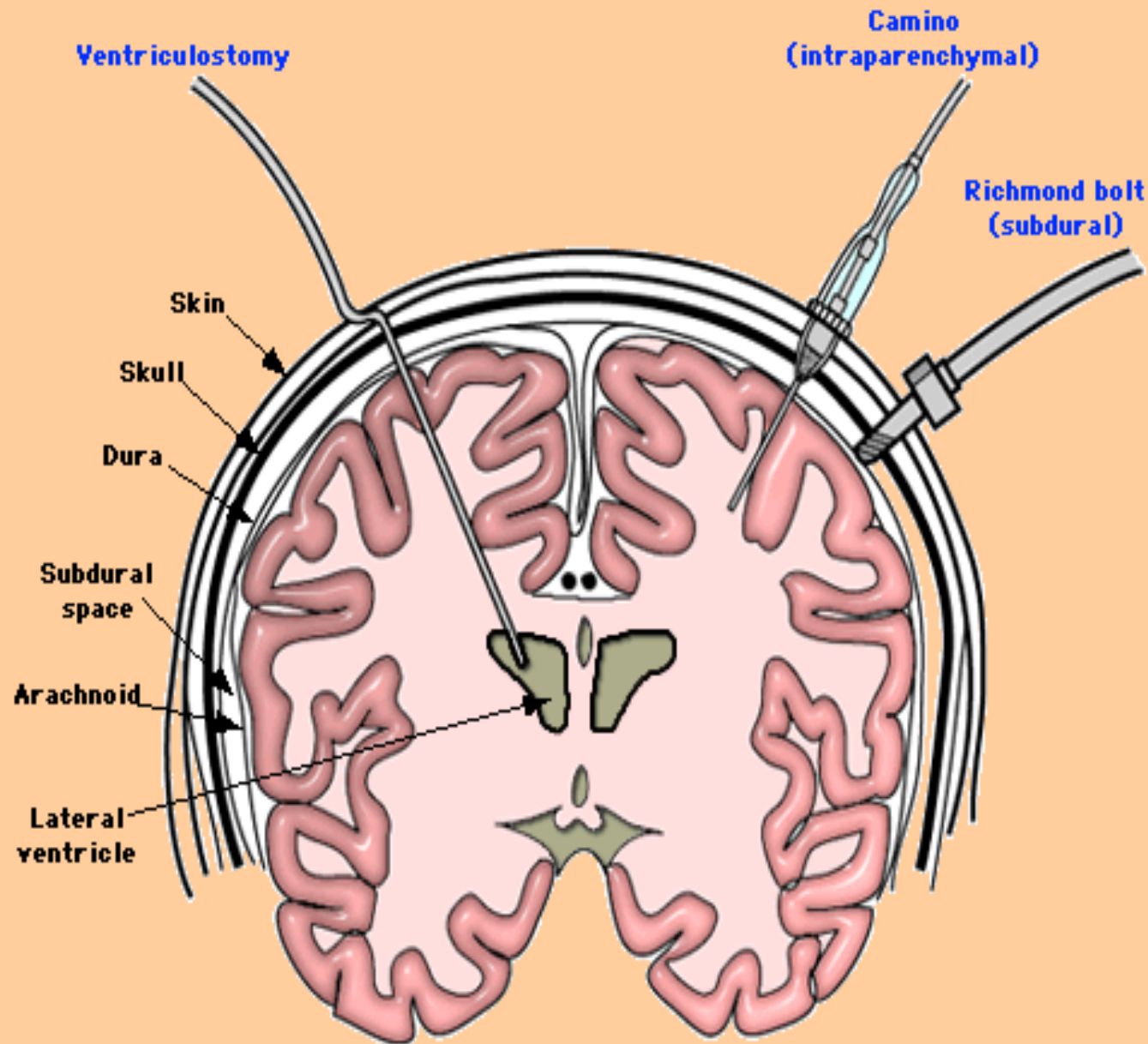
Product

catheter with drainage

Codman, Camino

Bolt system

Codman, Camino



Intracranial pressure monitors Ventriculostomy allows both ICP monitoring and therapeutic drainage of cerebrospinal fluid (CSF). Subdural and intraparenchymal monitors cannot be used to drain CSF.

Purpose of ICP Monitoring

Prevention of Herniation...

What to do with the information?

- **Goal: Adequate oxygen delivery to maintain the metabolic needs of the brain.**
- **Intracranial pressure < 20.**
- **Cerebral perfusion pressure 50-70.**

Manipulation of ICP

Brain

- **Mannitol**
 - dehydrates the brain, not the patient
 - monitor osmolality
- **Hypertonic saline**

Manipulation of ICP Blood

- **Decrease cerebral metabolic demands**
 - sedation, analgesia, barbiturates
 - avoid hyperthermia
 - avoid seizures
- **Hyperventilation**
 - decreases blood flow to the brain
 - only acutely for impending Herniation.
- **Mannitol**

Manipulation of ICP

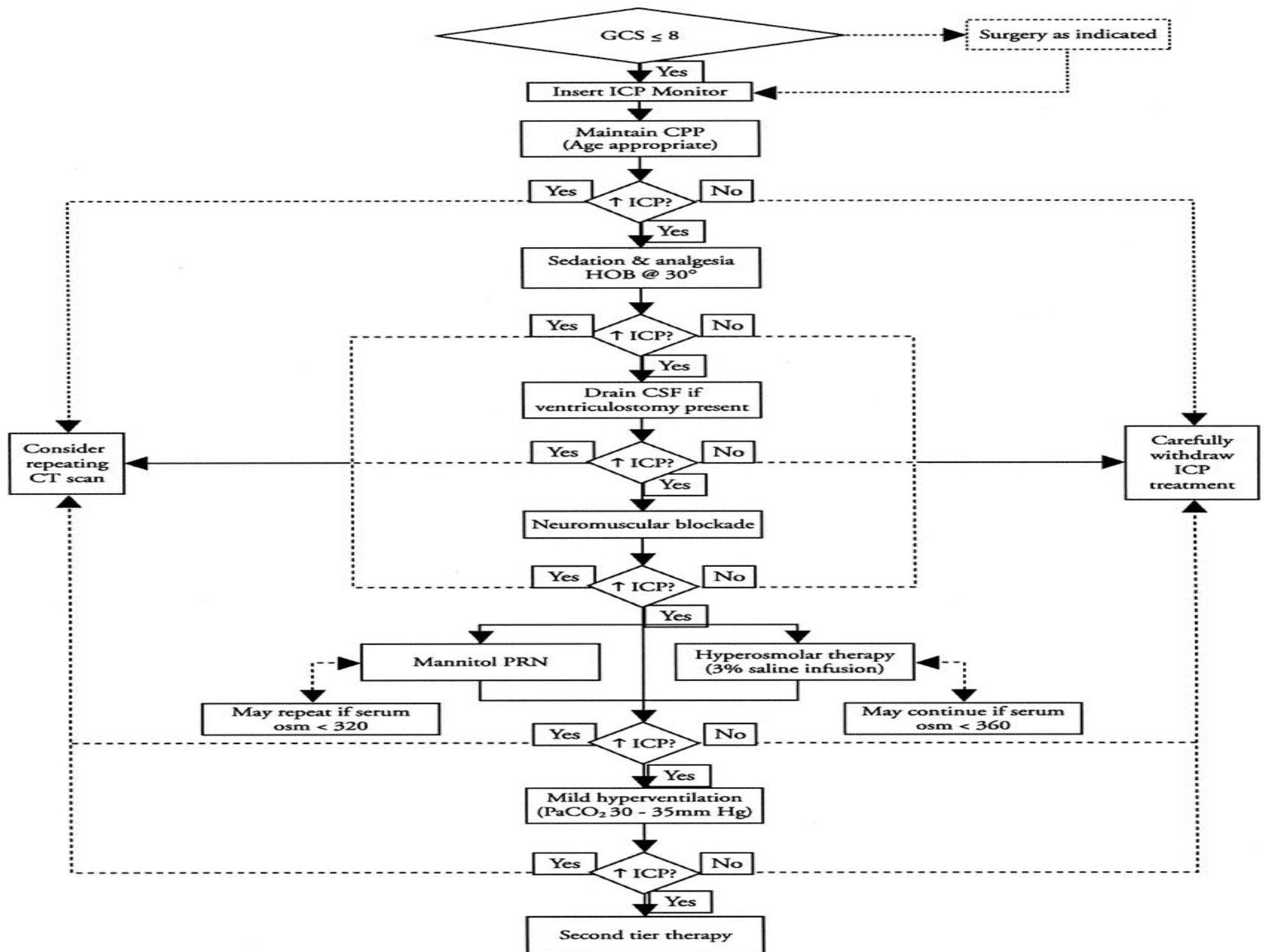
CSF

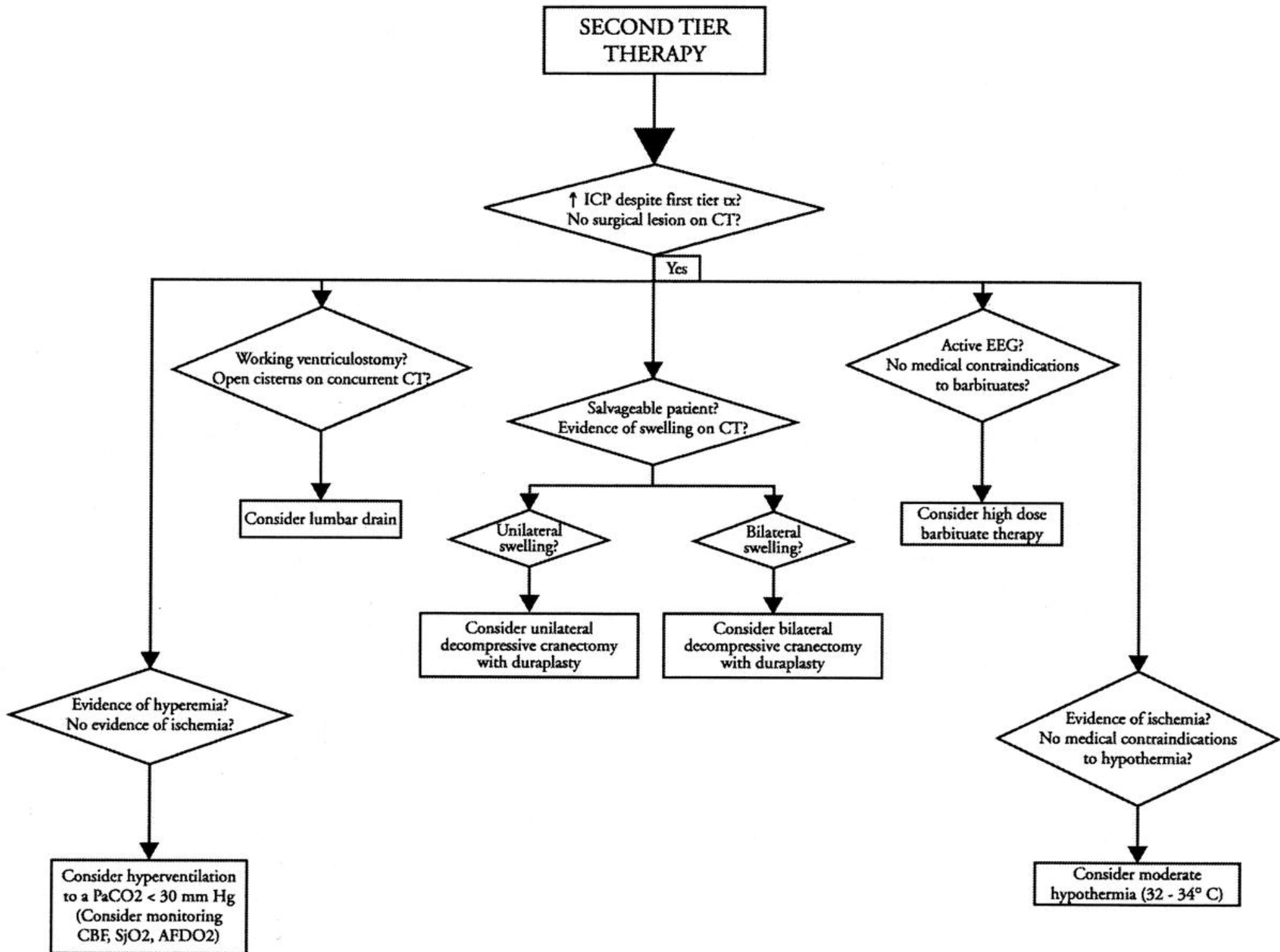
- **External drainage**
 - therapeutic as well as diagnostic
 - technical issues
 - infectious issues

Manipulation of CPP

$CPP = MAP - ICP$

- **Maintain adequate intravascular volume**
 - CVP
 - replace losses – urine, CSF, Blood
- **Increase MAP**





SECOND TIER THERAPY

↑ ICP despite first tier tx?
No surgical lesion on CT?

Yes

Working ventriculostomy?
Open cisterns on concurrent CT?

Consider lumbar drain

Salvageable patient?
Evidence of swelling on CT?

Unilateral swelling?

Consider unilateral
decompressive craniectomy
with duraplasty

Bilateral swelling?

Consider bilateral
decompressive craniectomy
with duraplasty

Active EEG?
No medical contraindications
to barbituates?

Consider high dose
barbituate therapy

Evidence of hyperemia?
No evidence of ischemia?

Consider hyperventilation
to a PaCO₂ < 30 mm Hg
(Consider monitoring
CBE, SjO₂, AFDO₂)

Evidence of ischemia?
No medical contraindications
to hypothermia?

Consider moderate
hypothermia (32 - 34° C)

Herniation Syndromes

Critically important herniation syndromes:

■ Uncal Herniation:

- occurs when a lateral expanding mass lesions pushes the uncus and hippocampal gyrus over the lateral edge of the tentorium

- Unilateral dilated pupil → progresses to brain stem dysfunction

Herniation Syndromes

■ Central Herniation

- downward displacement of the hemispheres and basal nuclei → inferior displacement of midbrain & surroundings thru tentorial notch

- initially affects diencephalon then progresses into midbrain and pons.

Clinical Progression of Transtentorial Herniation

Headache

Altered level of consciousness

Dilation of ipsilateral pupil

Cranial nerve III palsy

 Ptosis

 Loss of medial gaze

Decerebrate posturing

Hemiparesis

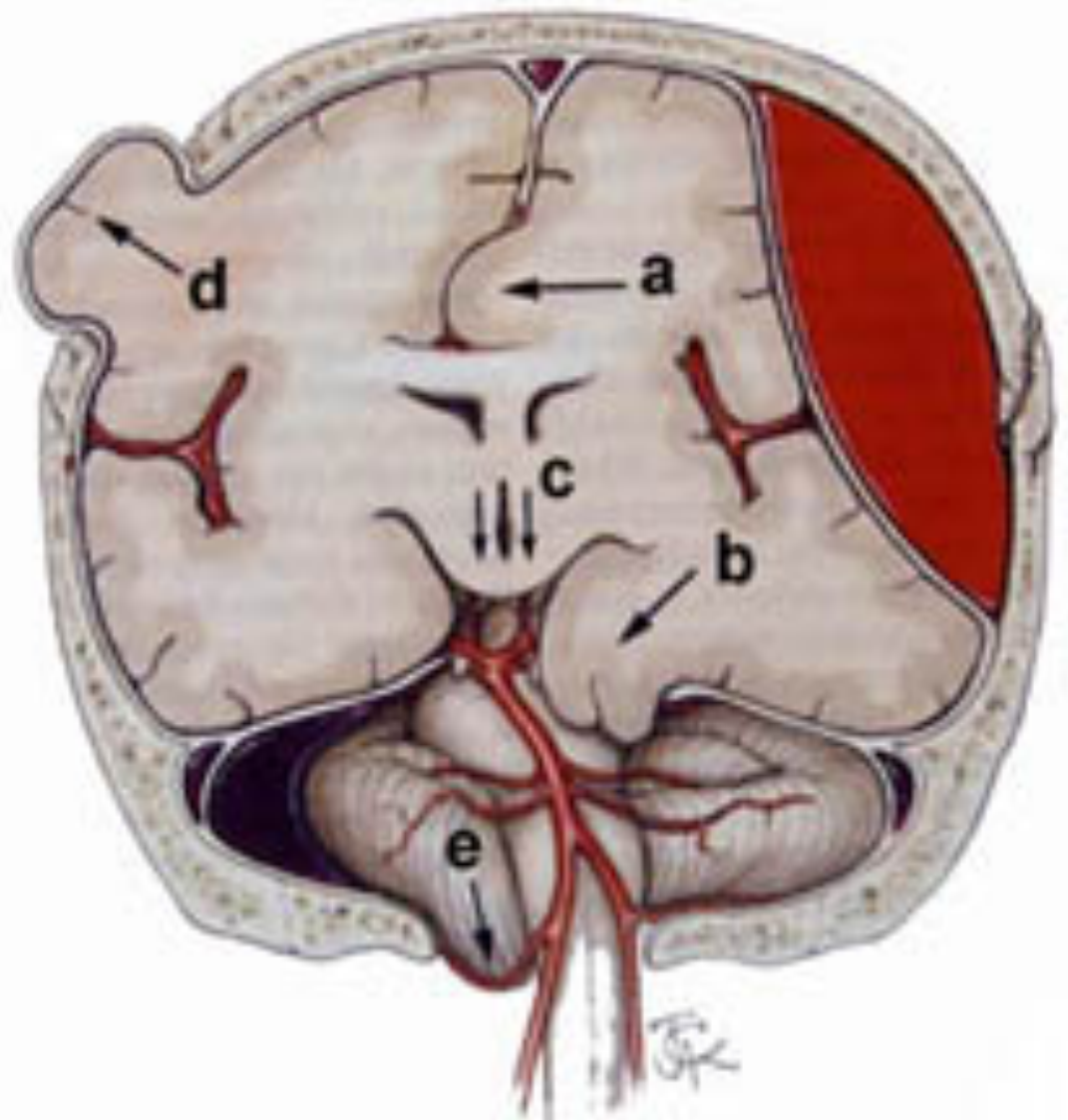
Dilation of opposite pupil

Alteration of respiration

Bradycardia

Hypertension

Respiratory arrest



**a) Subfalcial (cingulate) herniation ;
b) uncal herniation ; c) downward
(central, transtentorial) herniation ;
d) external herniation ; e) tonsillar
herniation.**

**Types a, b, & e are usually caused
by focal, ipsilateral space
occupying lesions, ie., tumor or
axial or extra-axial hemorrhage.**

Cushing Reflex

- Bradycardia
- Hypertension
- Altered respiratory status

OFTEN A VERY LATE CLINICAL FINDING!

Intrinsic causes

- Idiopathic degeneration (aging)
- Infarction* or ischemia
- Infiltrative diseases
 - Sarcoidosis
 - Amyloidosis
 - Hemochromatosis
- Collagen vascular diseases
 - Systemic lupus erythematosus
 - Rheumatoid arthritis
 - Scleroderma
- Myotonic muscular dystrophy
- Surgical trauma
 - Valve replacement
 - Correction of congenital heart disease
 - Heart transplantation
- Familial diseases
- Infectious diseases*
 - Chagas' disease
 - Endocarditis

*This condition causes atrioventricular conduction disturbances only.

Extrinsic causes

- Autonomically mediated syndromes
 - Neurocardiac syncope
 - Carotid-sinus hypersensitivity
- Situational disturbances
 - Coughing
 - Micturition
 - Defecation
 - Vomiting
- Drugs
 - Beta adrenergic blockers
 - Calcium-channel blockers
 - Clonidine
 - Digoxin
 - Antiarrhythmic agents
- Hypothyroidism
- Hypothermia
- Neurologic disorders
- Electrolyte imbalances
 - Hypokalemia
 - Hyperkalemia

†With permission from Mangrum, JM, DiMarco, JP. N Engl J Med 2000; 342:703.

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THANK YOU