

Evaluation & Management of Elevated Intracranial Pressure in Adults

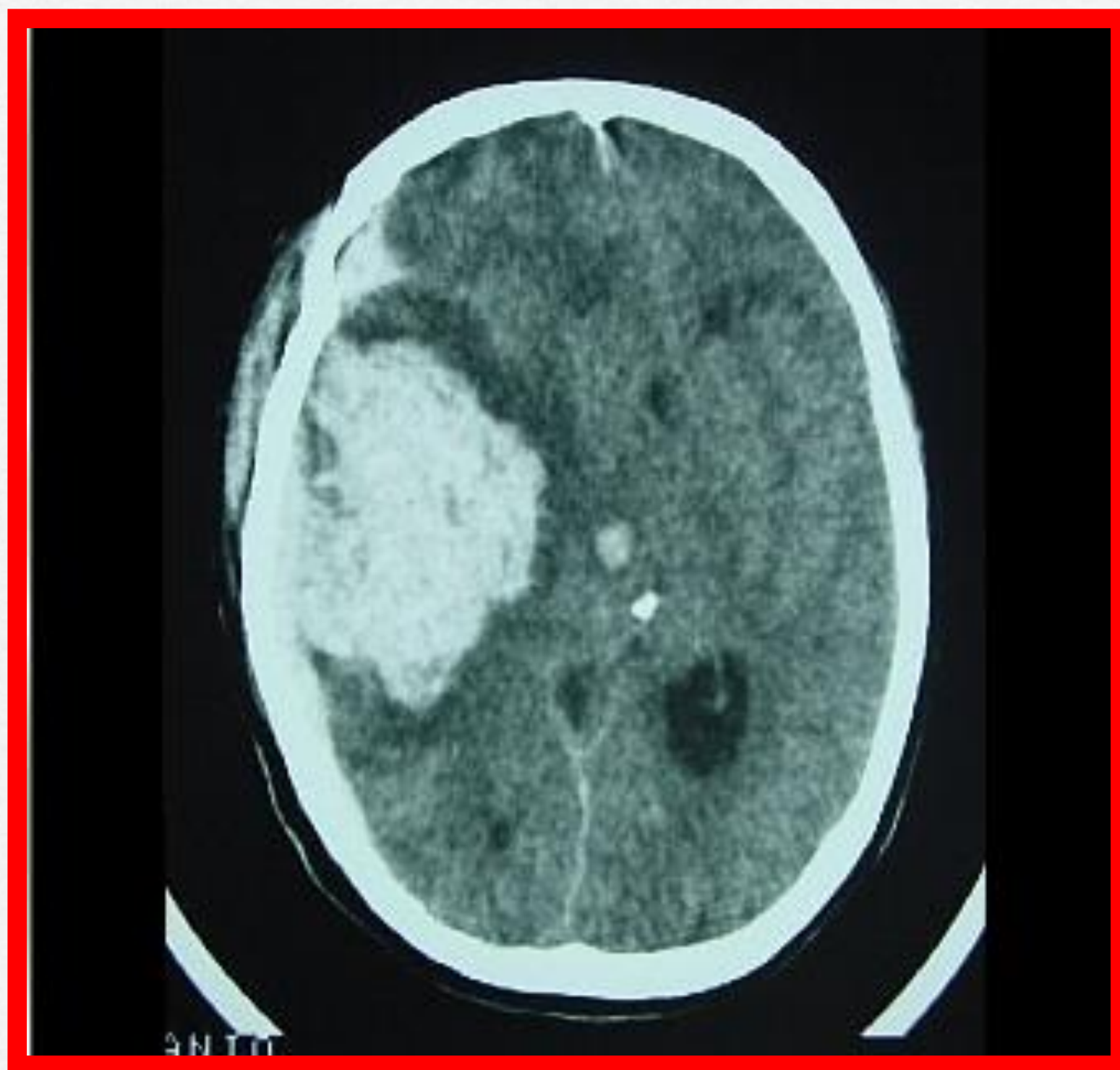
Dr. Tawfiq Almezeiny

Objectives

- ❑ **Pathophysiology of elevated intracranial pressure.**
- ❑ **Clinical features and sequences.**
- ❑ **Management : Investigations and Therapeutics.**

Case !

- ❑ **60 years old male**
- ❑ **CAD , HTN,COPD .**
- ❑ **Sudden LOC at home.**
- ❑ **M1V1E1 associated with new-onset seizure in ED.**
- ❑ **230/130 , 140 , 34 , 91%-LMA**



Introduction

- ❑ **Elevated intracranial pressure is a potentially devastating complication of neurologic injury.**
- ❑ **Prompt recognition, invasive monitoring, and therapy directed at both reducing ICP and reversing its underlying cause.**

Pathophysiology

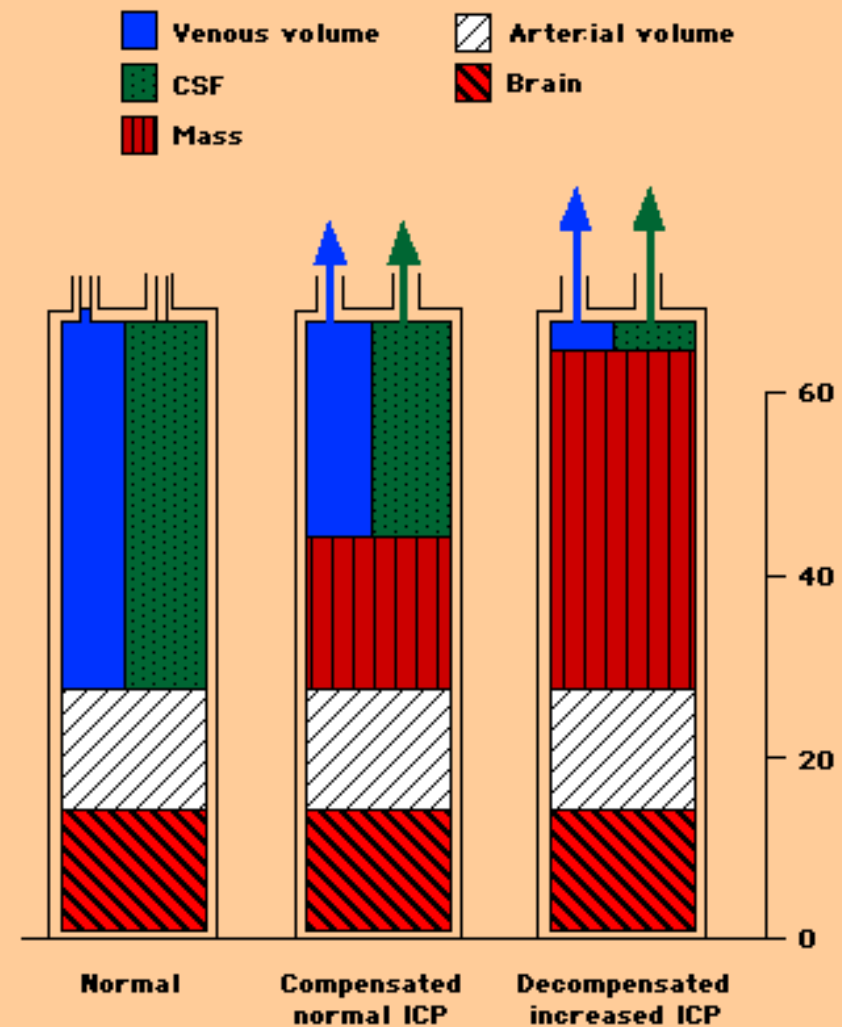
- ❑ **In adults, the intracranial compartment is protected by the skull, a rigid structure with a fixed internal volume of 1400 to 1700 mL**
- ❑ **Under physiologic conditions, the intracranial contents include (by volume) :**
 - Brain parenchyma — 80 %**
 - Cerebrospinal fluid — 10 %**
 - Blood — 10 %**

Pathophysiology

- **pathologic structures, including mass lesions, abscesses, and hematomas also may be present within the intracranial compartment. Since the overall volume of the cranial vault cannot change, an increase in the volume of one component, or the presence of pathologic components, necessitates the displacement of other structures, an increase in ICP, or both**
- **Thus, ICP is a function of the volume and compliance of each component of the intracranial compartment.**

Pathophysiology

- **The volume of brain parenchyma is relatively constant in adults, although it can be altered by mass lesions or in the setting of cerebral edema**
- **The volumes of CSF and blood in the intracranial space vary to a greater degree, and abnormal increases in the volume of either component may lead to elevations in ICP.**



Pathophysiology-CSF

- ❑ **CSF is produced by the choroid plexus and elsewhere in the central nervous system (CNS) at a rate of approximately 20 mL/h (500 mL/day) .**
- ❑ **CSF is normally resorbed via the arachnoid granulations into the venous system.**
- ❑ **Problems with CSF regulation generally result from impaired outflow caused by ventricular obstruction or venous congestion**
- ❑ **CSF production can become pathologically increased; this may be seen in the setting of choroid plexus papilloma**

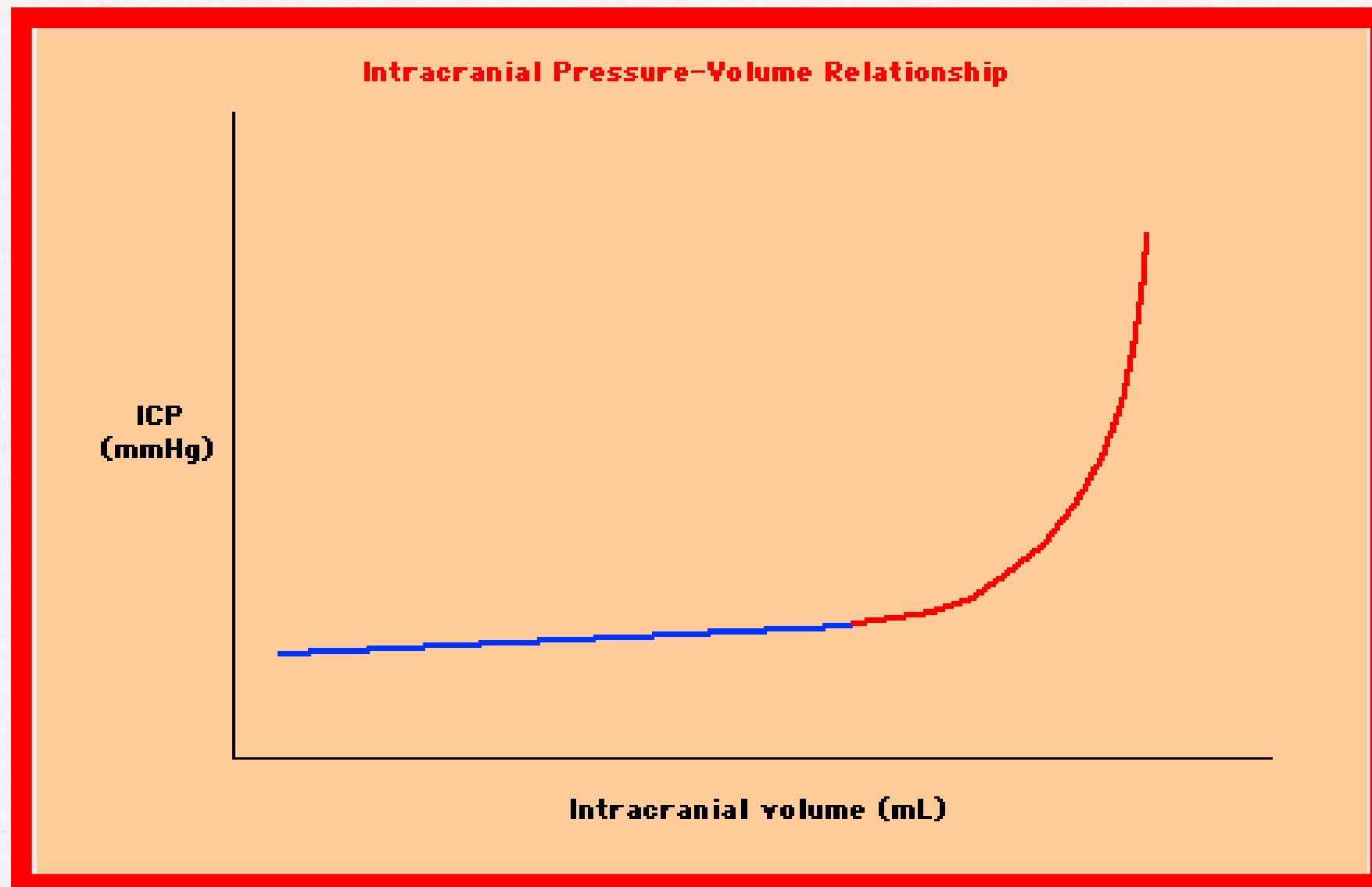
Pathophysiology

- ❑ **Cerebral blood flow (CBF) determines the volume of blood in the intracranial space.**
- ❑ **CBF increases with hypercapnia and hypoxia.**
- ❑ **Autoregulation of CBF may be impaired in the setting of neurologic injury, and may result in rapid and severe brain swelling, especially in children**

Intracranial Compliance

- ❑ **The interrelationship between changes in the volume of intracranial contents and changes in ICP defines the compliance characteristics of the intracranial compartment.**
- ❑ **The compliance relationship is nonlinear, and compliance decreases as the combined volume of the intracranial contents increases. Initially, compensatory mechanisms allow volume to increase with minimal elevation in ICP**
- ❑ **These mechanisms include:**
 - ◇ **Displacement of CSF into the thecal sac**
 - ◇ **Decrease in the volume of the cerebral venous blood via venoconstriction and extracranial drainage**

Intracranial Compliance



Intracranial Compliance

- ❑ Magnitude of the change in volume of an individual structure determines its effect on ICP.**
- ❑ Rate of change in the volume of the intracranial contents influences ICP.**

Cerebral blood flow

- **CBF : function of the pressure drop across the cerebral circulation divided by the cerebrovascular resistance, as predicted by Ohm's law :**

$$\text{CBF} = (\text{CAP} - \text{JVP}) \div \text{CVR}$$

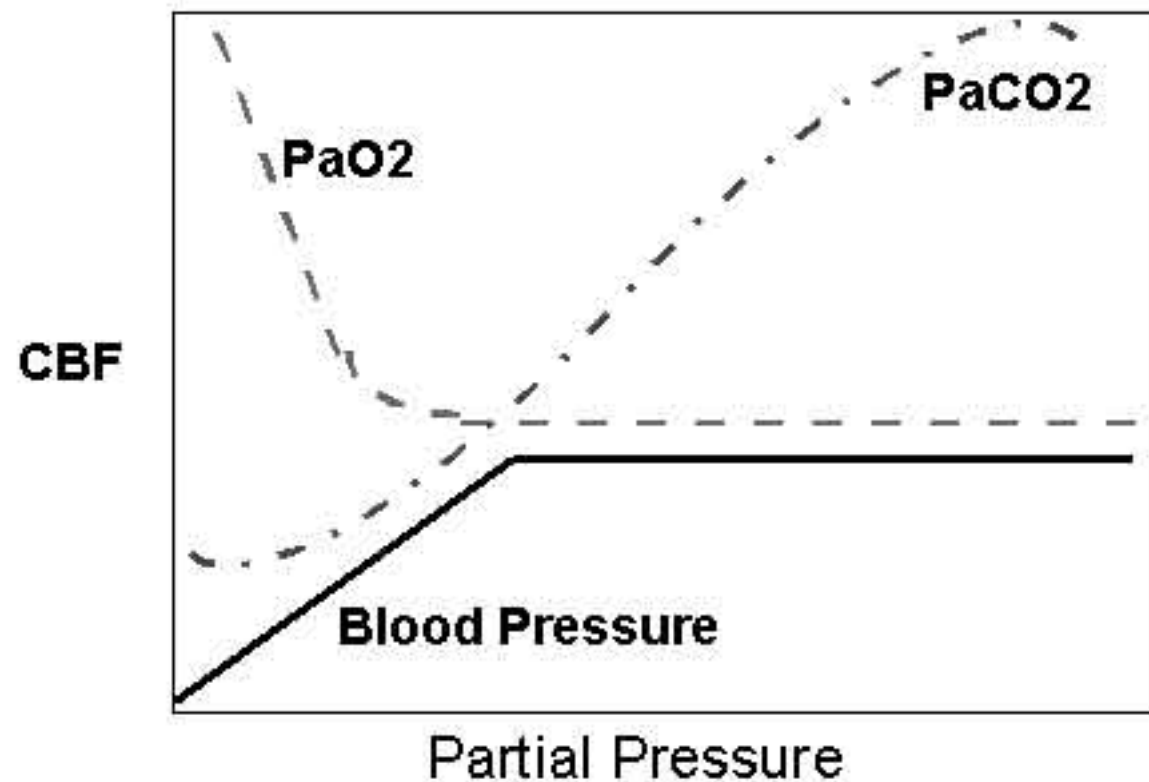
where CAP is carotid arterial pressure, JVP is jugular venous pressure, and CVR is cerebrovascular resistance.

Cerebral Blood Flow

- **CPP is defined as : mean arterial pressure (MAP) minus ICP.**

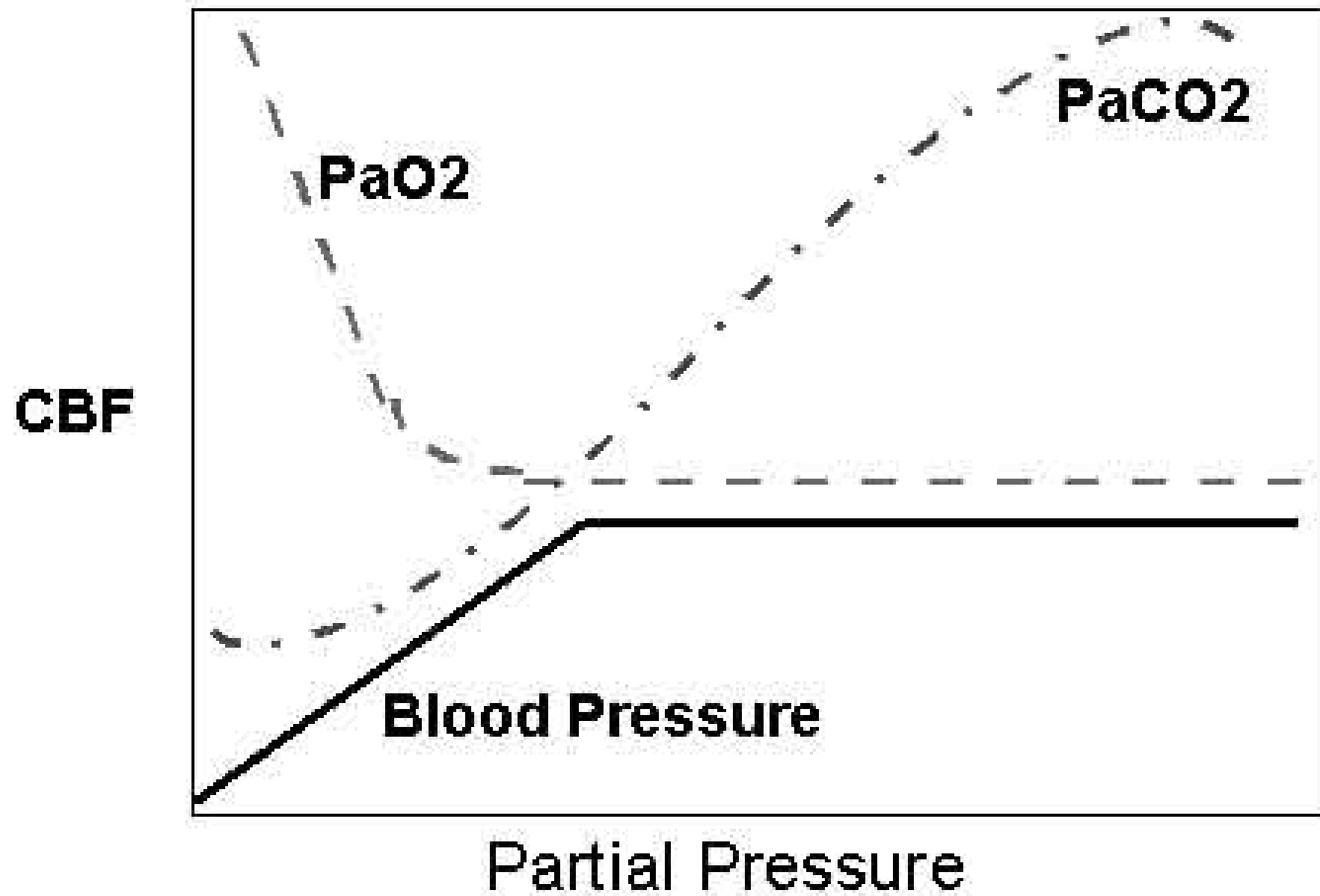
$$\text{CPP} = \text{MAP} - \text{ICP}$$

Effect of Blood Gases on Cerebral Perfusion

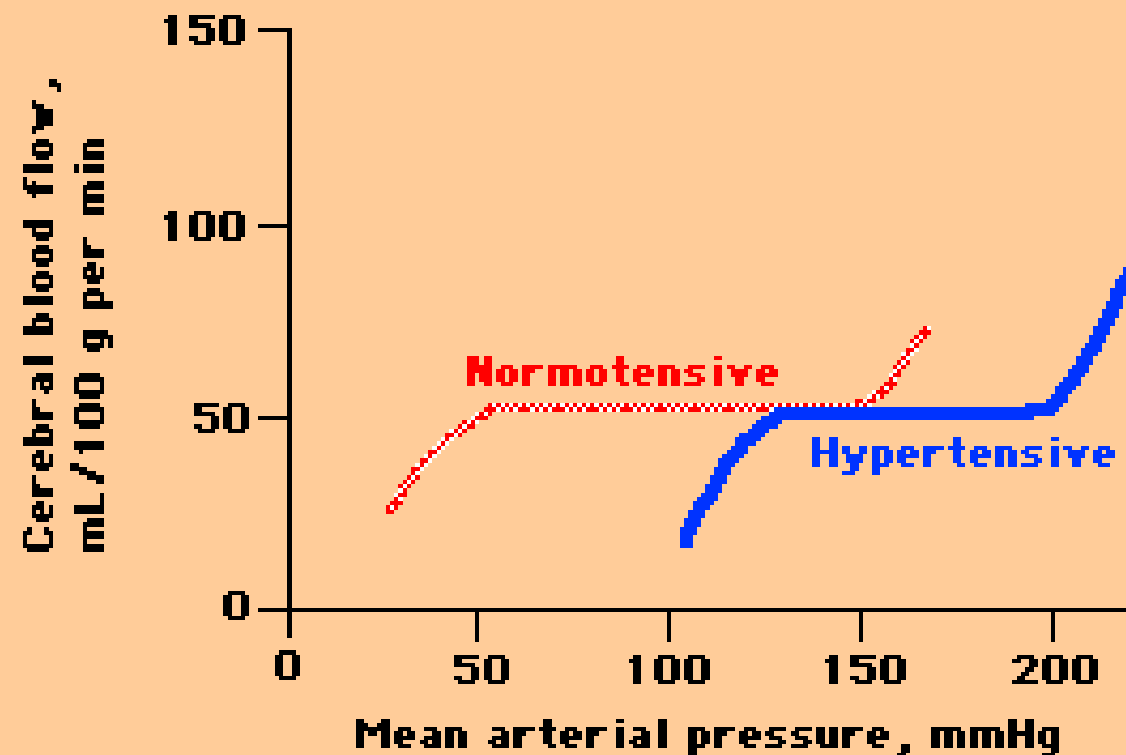


As you can see, with increased $p\text{CO}_2$, cerebral blood flow increases, and hence the volume of the blood in the brain increases, and the intracranial pressure increases. Similarly, with LOW $p\text{O}_2$ (less than 50mmHg), CBF will increase, and this can lead to increased ICP. Also note that with hypocapnia (ie, hyperventilation), CBF decreases--if this is severe or prolonged, ischemia may ensue. Autoregulation may or may not be intact in the injured brain (blood gases or blood pressure). Actually, autoregulation of cerebral blood flow in response to changes in blood gases is a complex phenomenon, and is in part a function of local pH (which is a function of $p\text{CO}_2$), possibly mediated by nitric oxide. Thus, patients who are chronically hypercapneic should not be corrected quickly, as this will lead to alkalosis and cerebral ischemia. Similarly, hyperventilation, and its induced alkalosis, will only reduce CBF for a short time (24-36 hours), until CSF pH normalizes.

Effect of Blood Gases on Cerebral Perfusion

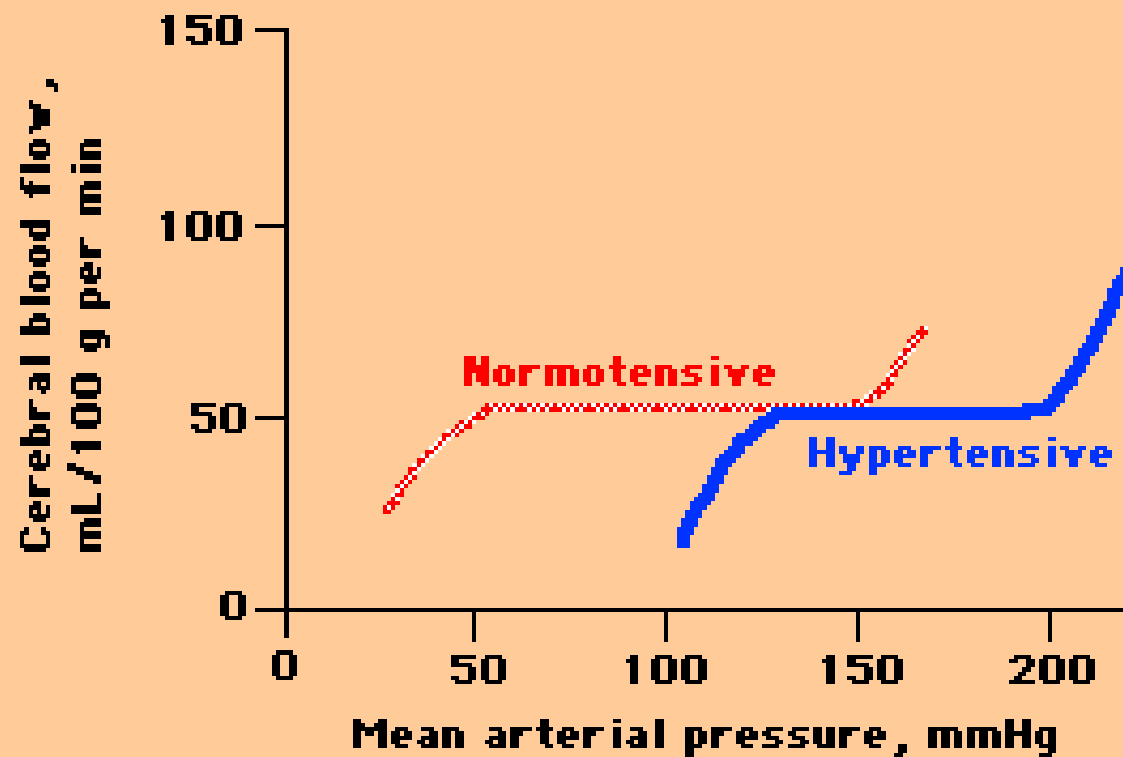


Autoregulation



- CBF is normally maintained at a relatively constant level by cerebrovascular autoregulation of CVR over a wide range of CPP (50 to 100 mmHg) .
- Autoregulation of CVR can become dysfunctional in certain pathologic states, most notably stroke or trauma.

Autoregulation



- In patients with hypertension: the set point is different.
- Acute reductions in blood pressure, even if the final value remains within the normal range, can produce ischemic symptoms in patients with chronic hypertension

Headache

altered level of consciousness

Ipsilateral pupil dilation

oculomotor nerve palsy

Decerebrate posturing

Hemiparesis

Alteration of respiration

Hypertension

Bradycardia

Respiratory arrest

QUIZ



Clinical Manifestations

global or local reductions in CBF are responsible for the clinical manifestations of elevated ICP.

These manifestations can be further divided into:

- ◇ generalized responses to elevated ICP**
- ◇ herniation syndromes**

CLINICAL MANIFESTATIONS

Global symptoms of elevated ICP include

- ❑ **headache**
- ❑ **Depressed global consciousness due to either the local effect of mass lesions or pressure on the midbrain reticular formation**
- ❑ **vomiting.**
- ❑ **Signs include CN VI palsies**
- ❑ **Papilledema**
- ❑ **Spontaneous periorbital bruising**
- ❑ **Triad of bradycardia, respiratory depression, and hypertension (Cushing's triad)**

CLINICAL MANIFESTATIONS

- ❑ **Mass lesions (?example)**
- ❑ **Herniation syndromes: Herniation results when pressure gradients develop between two regions of the cranial vault**

Herniation Syndromes

- **◇subfalcine**
- **◇ central**
- **◇transtentorial**
- **◇uncal transtentorial**
- **◇upward cerebellar**
- **◇cerebellar tonsillar/foramen magnum**
- **◇transcalvarial**

- ☐ **Kernohan's notch phenomenon.**
- ☐ **Combination of contralateral pupillary dilatation and ipsilateral weakness.**
- ☐ **Use of radiologic studies may support the diagnosis.**
- ☐ **the most reliable method of diagnosing elevated ICP is to measure it directly.**

ICP MONITORING

- ❑ **The purpose of monitoring ICP is to improve the clinician's ability to maintain adequate CPP and oxygenation**
- ❑ **Empiric therapy for presumed elevated ICP is unsatisfactory**
- ❑ **Therapies directed at lowering ICP are effective for limited and variable periods of time**
- ❑ **Treatments may have serious side effects**

- ❑ **The only way to reliably determine CPP (defined as the difference between MAP and ICP) is to continuously monitor both ICP and blood pressure (BP)**
- ❑ **Patients are managed in intensive care units (ICUs) with an ICP monitor and arterial line**
- ❑ **The combination of ICP monitoring and concomitant management of CPP may improve patient outcomes, particularly in patients with closed head trauma**

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DESIGN: Study of case records. **METHODS:** The data files from the Ontario Trauma Registry from 1989 to 1995 were examined. Included were all cases with an Injury Severity Score (ISS) greater than 12 from the 14 trauma centres in Ontario.

CONCLUSIONS: ICP monitor insertion rates vary widely in Ontario's trauma hospitals. The insertion of an ICP monitor is associated with a statistically significant decrease in death rate among patients with severe TBI. This finding strongly supports the need for a prospective randomized trial of management protocols, including ICP monitoring, in patients with severe TBI.

Intracranial pressure monitoring and outcomes after traumatic brain injury.

**Lane PL; Skoretz TG; Doig G; Girotti MJ
Can J Surg 2000 Dec;43(6):442-8.**

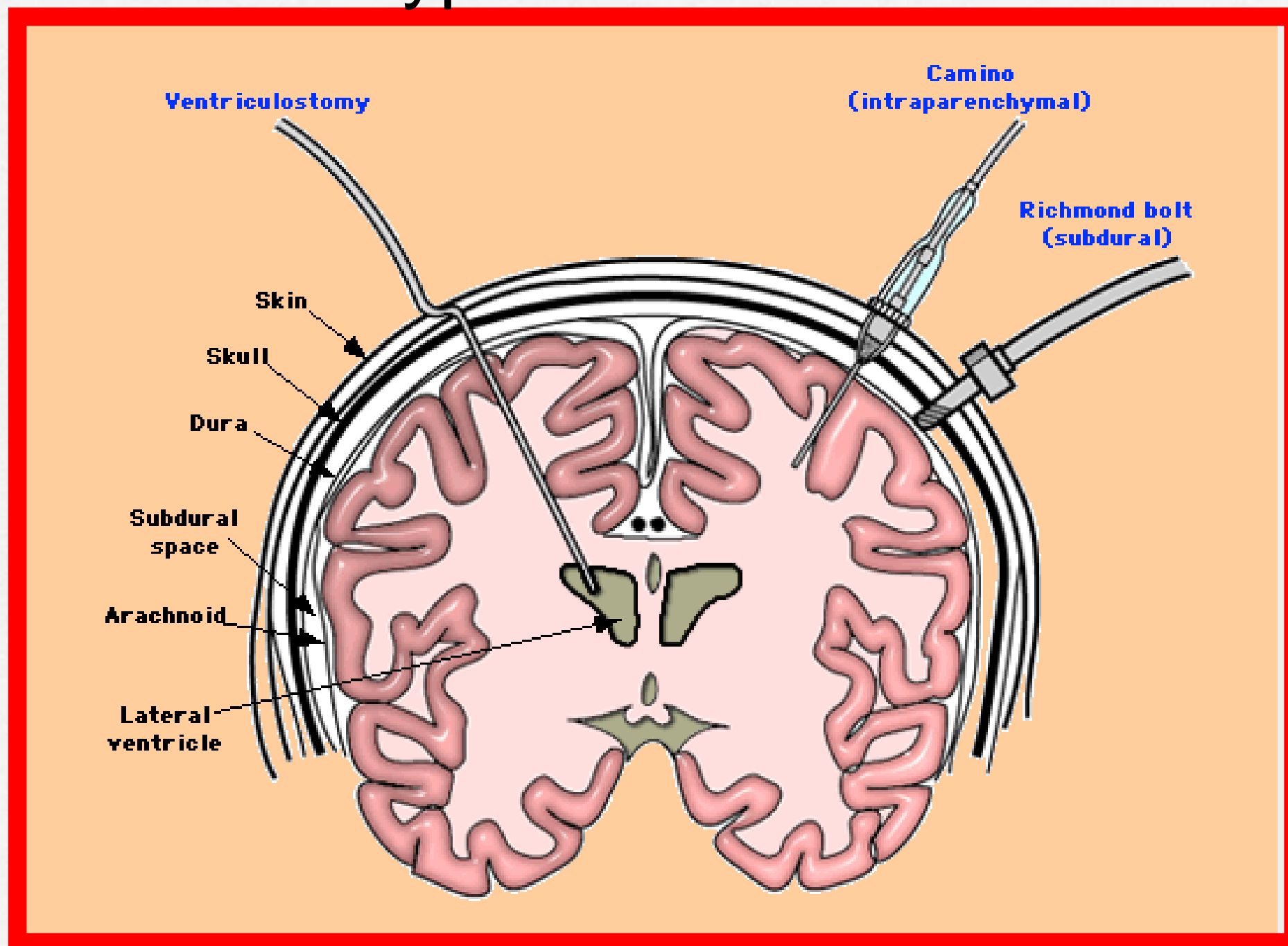
- ❑ **CPP should be kept between 60 and 75 mmHg in patients with elevated ICP, in an attempt to avoid hypoperfusion and ischemic injury.**
- ❑ **One study of 158 patients with head trauma and a Glasgow Coma Scale (GCS) <7 found that ICP monitoring and maintenance of CPP >70 mmHg resulted in improved outcomes when compared with historical controls . This study also demonstrated that ICP generally did not increase with elevations in CPP until a critical level >110 mmHg was reached**

Indications-ICP monitoring

- ❑ **Closed head injury is one of the most frequent and best-studied indications for ICP monitoring.**
- ❑ **Other potential indications for ICP monitoring include: stroke, intracerebral hemorrhage, hydrocephalus, subarachnoid hemorrhage, Reye's syndrome, hepatic encephalopathy, and sagittal sinus thrombosis.**

- ❑ **The Guidelines for the Management of Severe Head Injury suggest that ICP monitoring is indicated in comatose head injury patients with Glasgow Coma Score (GCS) 3 to 8 and with abnormal cranial findings on computed tomographic (CT) scan**
- ❑ **Comatose patients with normal CT scans have a much lower incidence of elevated ICP unless they have the following features at admission:**
 - Age >40 years**
 - Unilateral or bilateral motor posturing**
 - Systolic blood pressure (SBP) <90 mmHg**

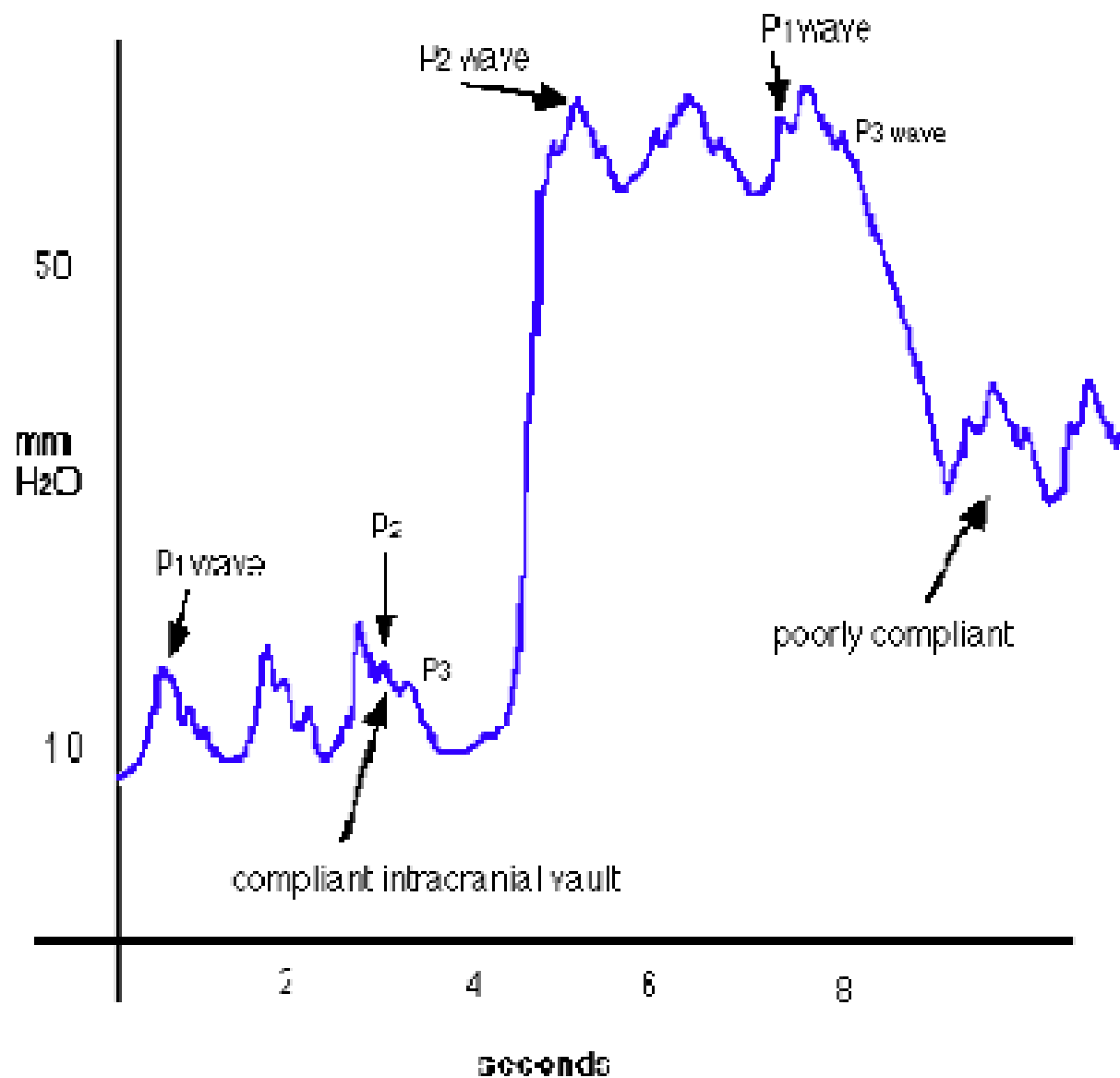
Type of Monitors



In progress..

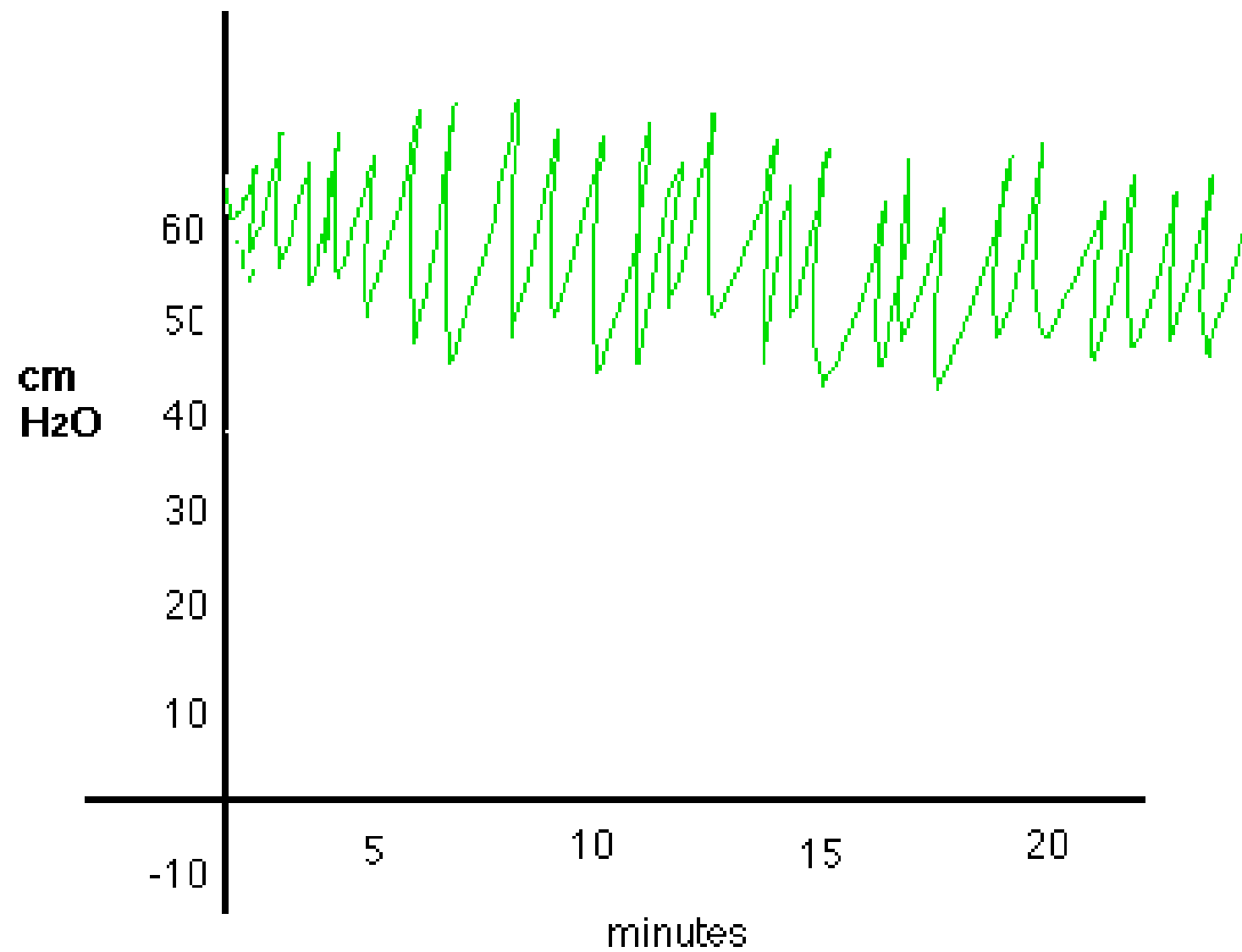
- ❑ **Tissue resonance analysis (TRA)**
an ultrasound-based method.
In one trial 40 patients underwent both invasive and TRA ICP monitoring, with good correlation between concomitant invasive and TRA measurements .
- ❑ **Transcranial Doppler (TCD)**
measures the velocity of blood flow in the proximal cerebral circulation.
- ❑ **Intraocular pressure** (using an ultrasonic handheld optic tonometer)
- ❑ **Tympanic membrane displacement**
(measured using an impedance audiometer)
based on the hypothesis that increased ICP will transmit a pressure wave to the tympanic membrane via the perilymph
- ❑ **jugular venous oxygen saturation monitoring**

Waveform analysis



- P1 (percussion wave) represents arterial pulsations
- P2 (rebound wave) reflects intracranial compliance
- P3 (dichrotic wave) represents venous pulsations

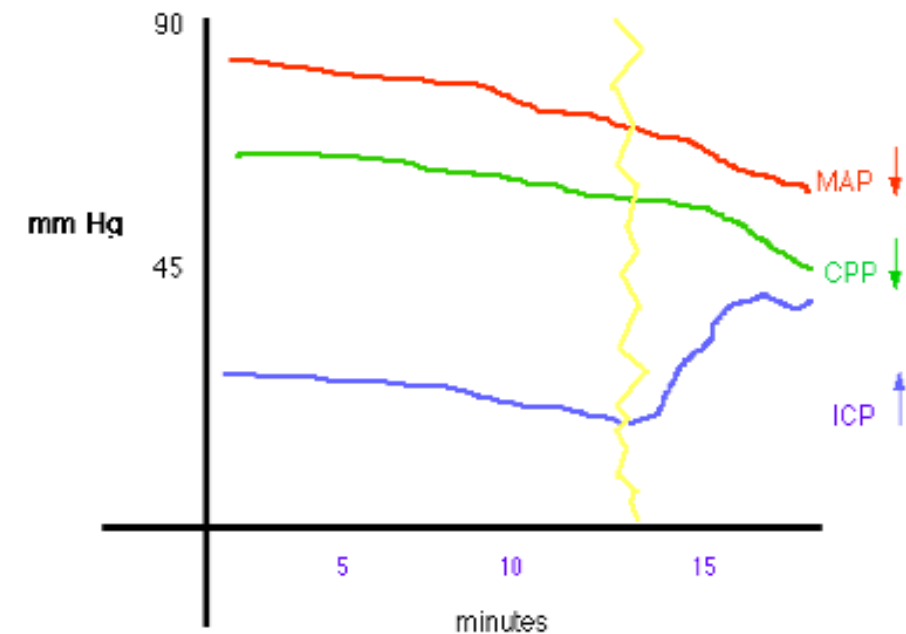
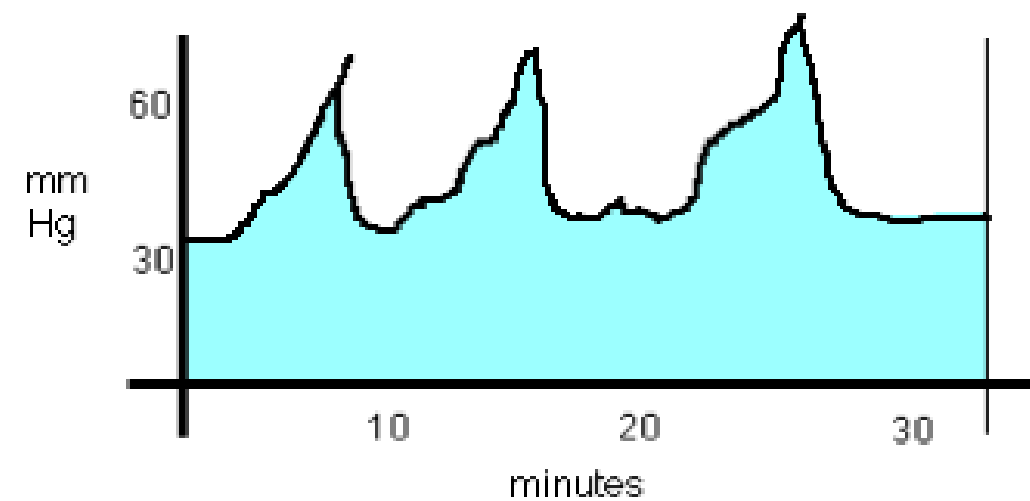
intracranial pressure: 'b' waves

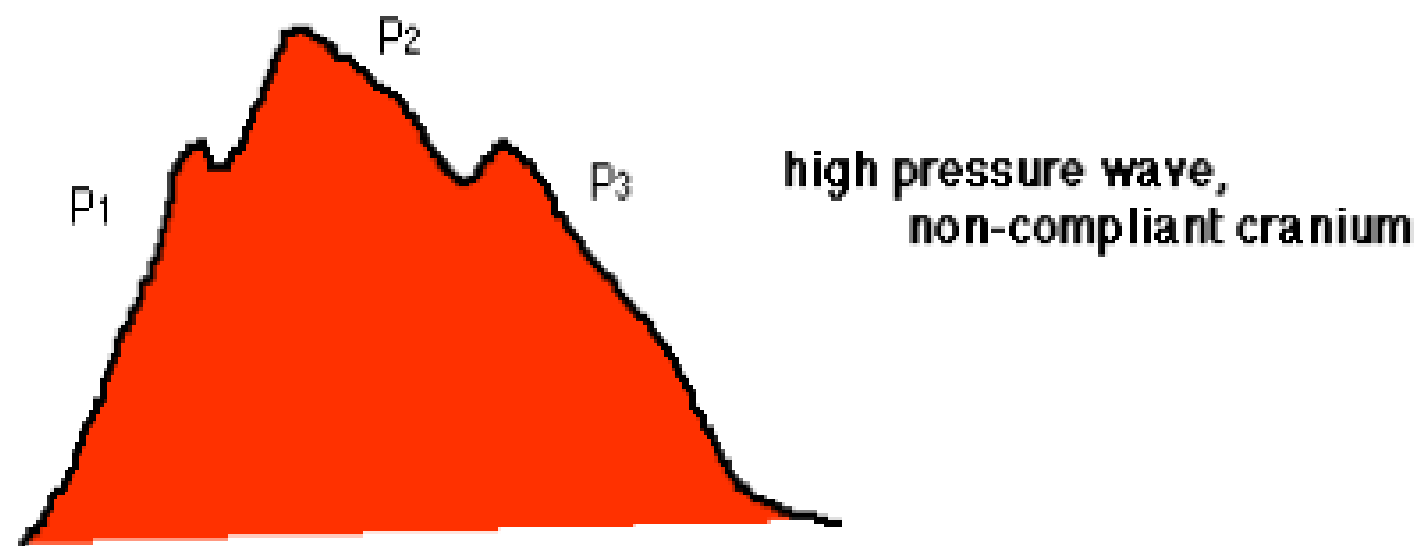
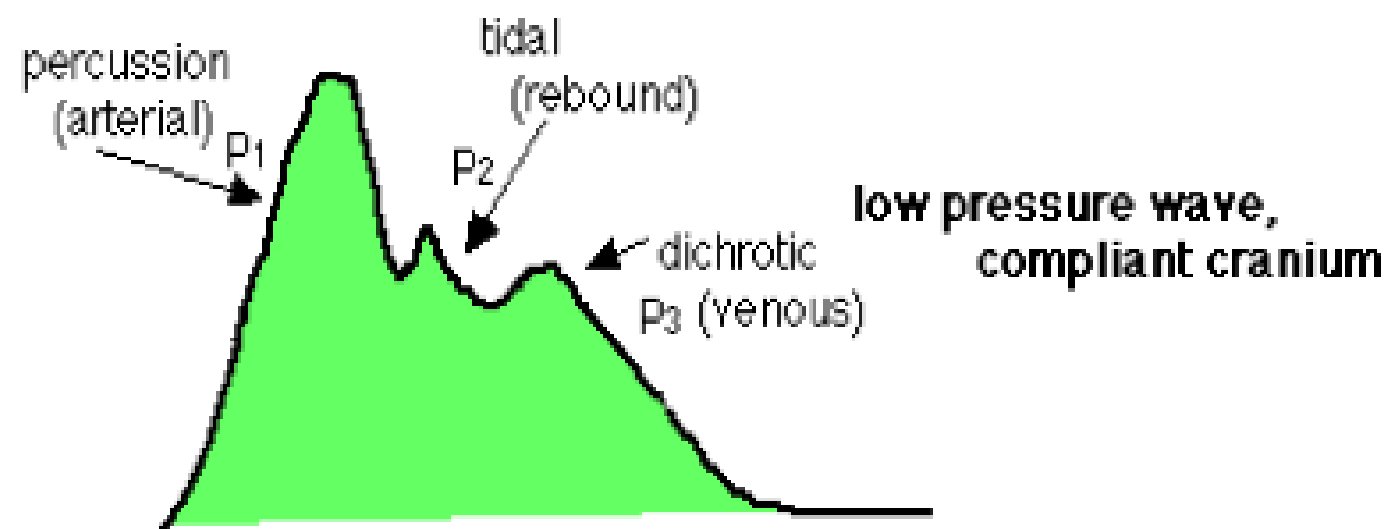


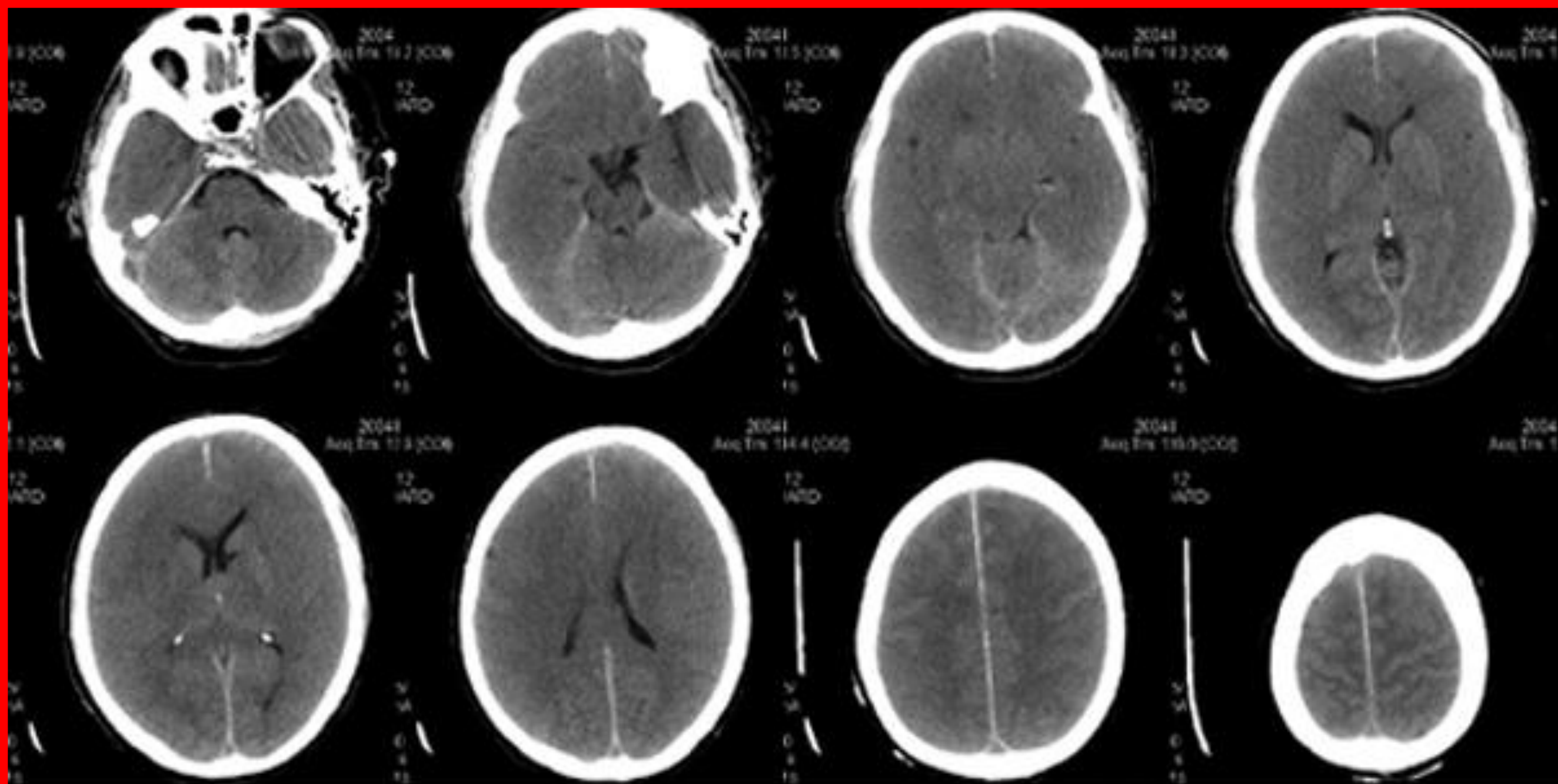
A-waves

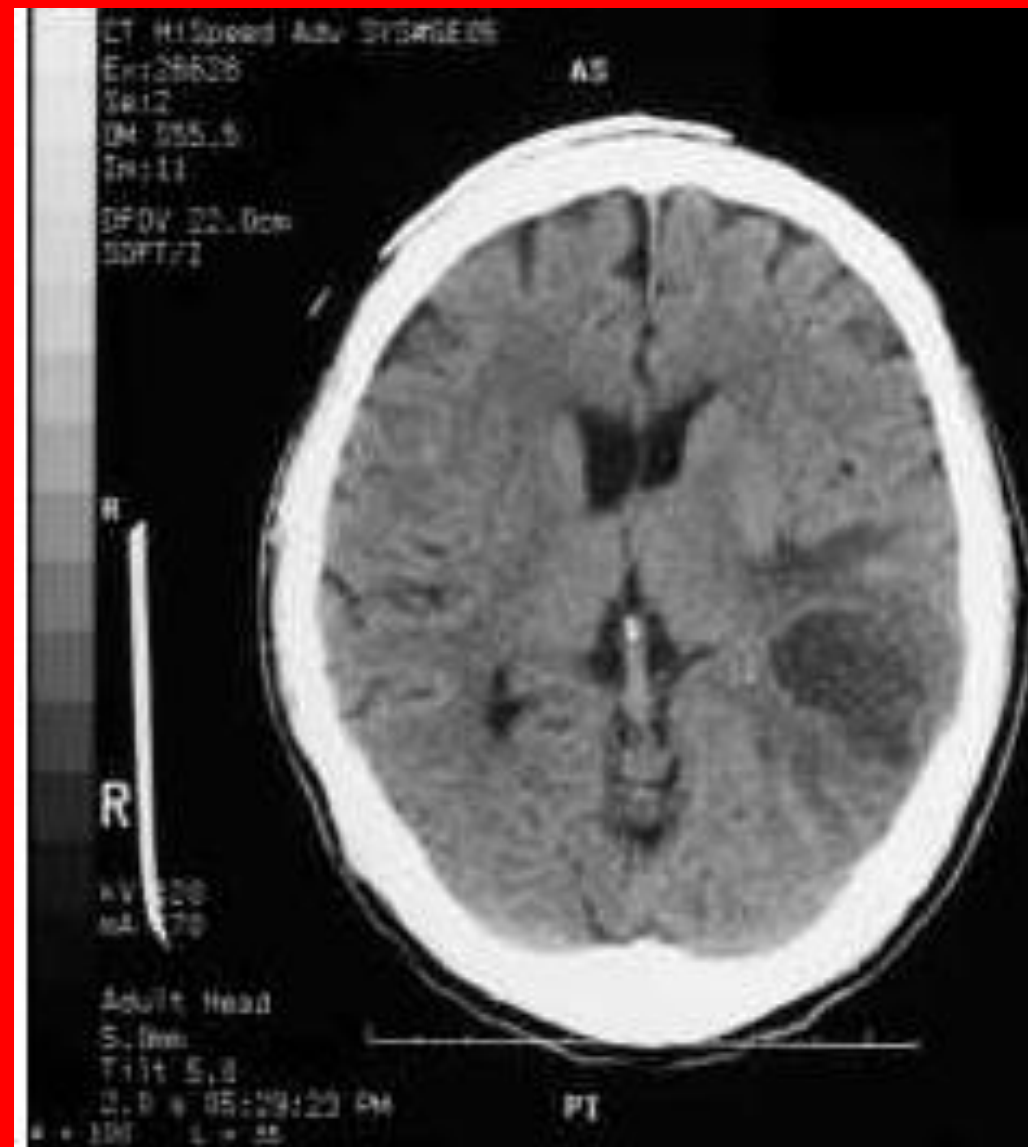
- **Pathological A waves (also called plateau waves) are abrupt, marked elevations in ICP of 50 to 100 mmHg, which usually last for minutes to hours.**
- **signifies a loss of intracranial compliance, and heralds imminent decompensation of autoregulatory mechanisms**

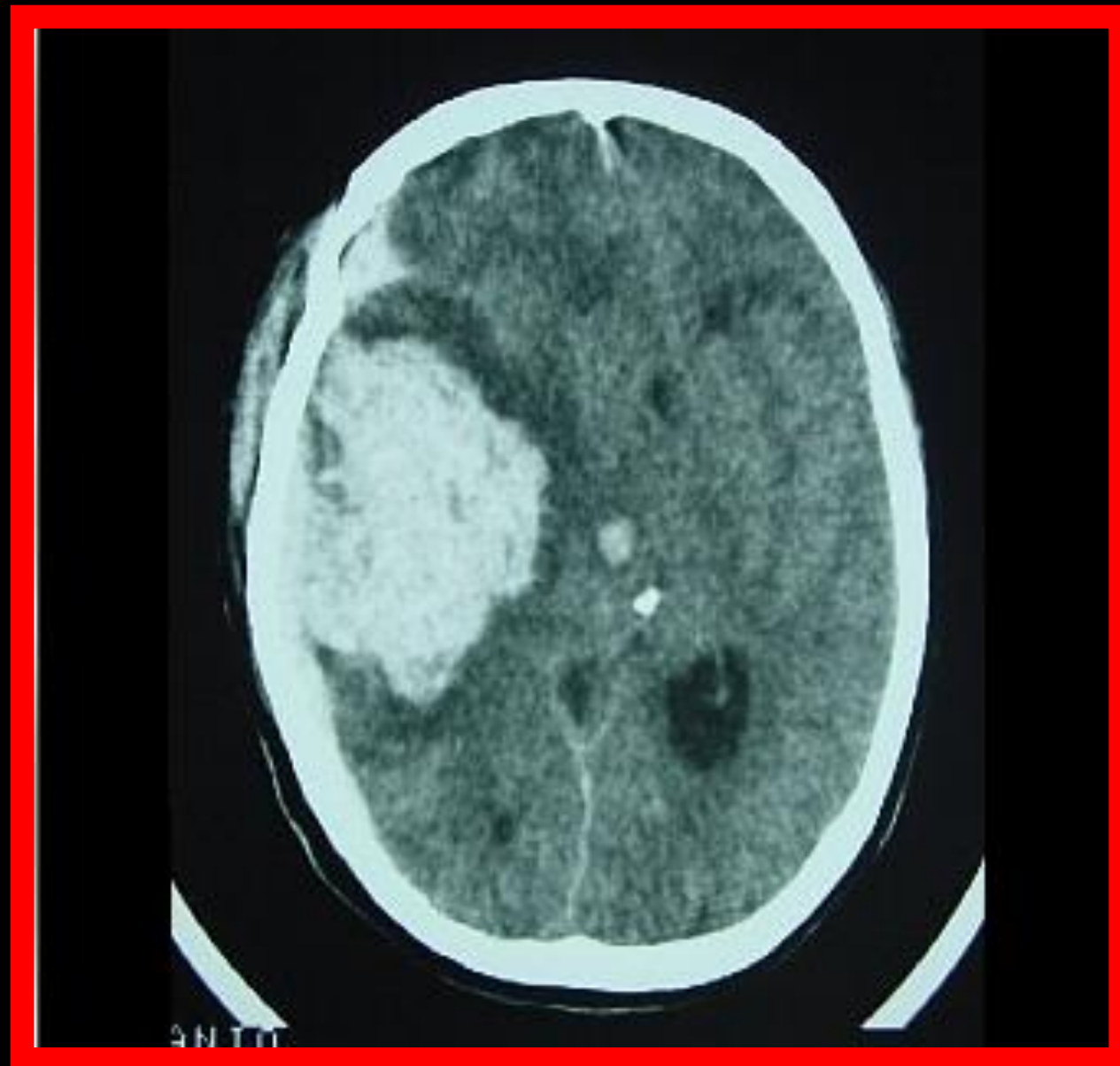
intracranial pressure: 'a' waves











Management

The best therapy for intracranial hypertension (ICH) is resolution of the proximate cause of elevated ICP

- ◇ **evacuation of a blood clot**
- ◇ **resection of a tumor**
- ◇ **CSF diversion in the setting of hydrocephalus**

Management

- **Head elevation**
- **General:** support of oxygenation, blood pressure, and end-organ perfusion
- **BP Control**
 - ◆ BP should be sufficient to maintain CPP >60 mmHg
 - ◆ Large shifts in blood pressure should be minimized, with particular care taken to avoid hypotension
- **Fluid:** Patients should be kept euvolemic and normo- to hyperosmolar
 - ◆ (isotonic fluids such as 0.9 percent (normal) saline). Serum osmolality should be kept >280 mOsm/L.
 - ◆ avoid all free water (including D5W, 0.45 percent (half normal) saline, and enteral free water).
- **Sedation** — Keeping patients appropriately sedated can decrease ICP by reducing metabolic demand, ventilator asynchrony, venous congestion, and the sympathetic responses of hypertension and tachycardia

Metabolic Demand

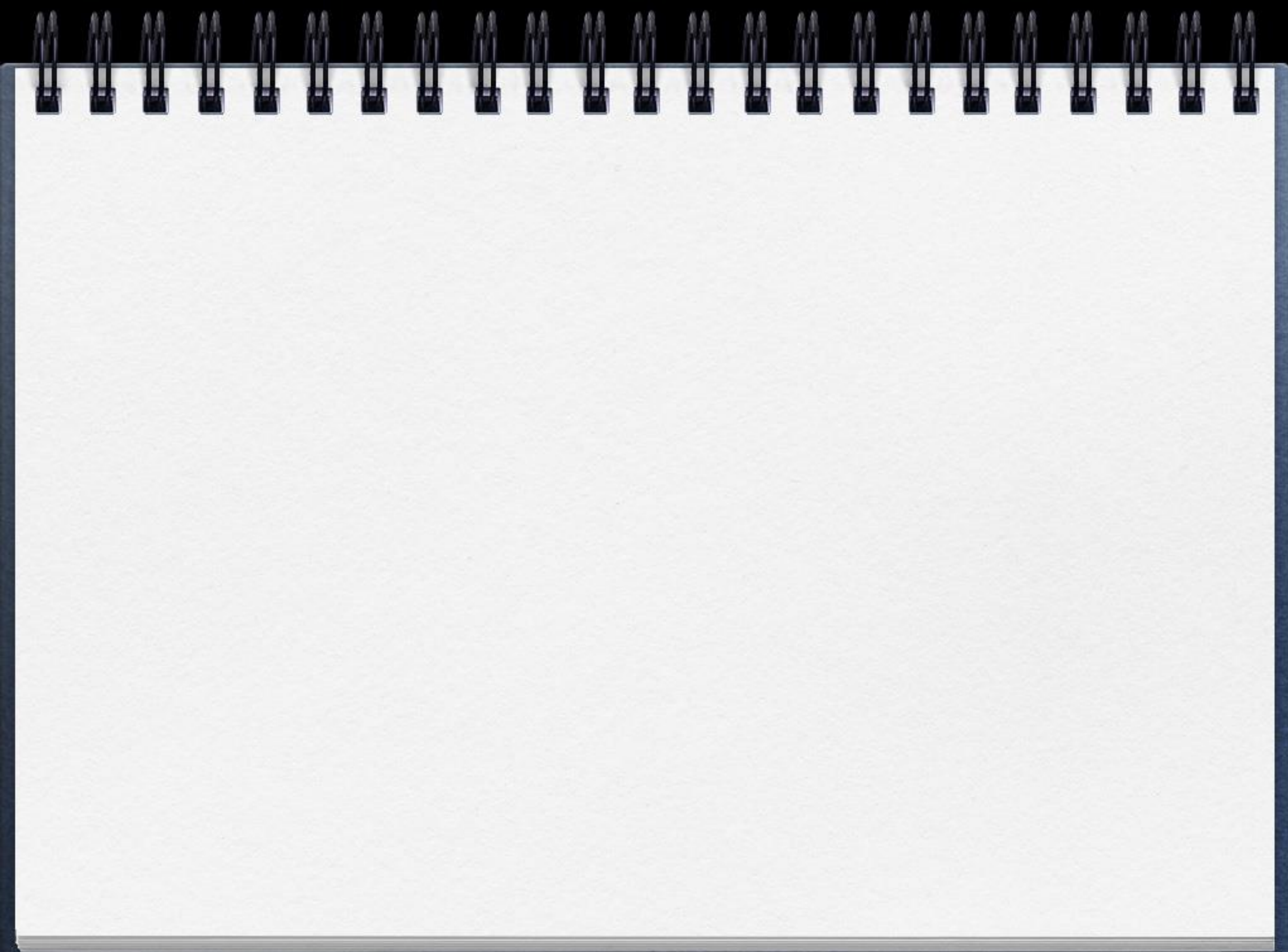
Elevated metabolic demand in the brain results in increased cerebral blood flow (CBF), and can elevate ICP by increasing the volume of blood in the cranial vault. Conversely, decreasing metabolic demand can lower ICP by reducing blood flow.

◇Fever

◇seizure

Mannitol

- ❑ **Osmotic diuretic ; reduce brain volume by drawing free water out of the tissue and into the circulation, where it is excreted by the kidneys, thus dehydrating brain**
- ❑ **Dose: 0.25 to 0.5 g/kg as needed IV Q6H (20% soln)**
- ❑ **The effects are usually present within minutes, peak at about one hour, and last 4 to 24 hours**
- ❑ **Furosemide appears to be synergistic**
- ❑ **Adverse effects?**
- ❑ **Reassess: serum sodium >150 meq, serum osmolality >320 mOsm, or evidence of evolving acute tubular necrosis (ATN)**



Corticosteroids

- ❑ **corticosteroids may have a role in the setting of intracranial hypertension caused by brain tumors and CNS infections**

- **Patients were entered into the trial within eight hours of presentation, and were randomly assigned to therapy with methylprednisolone (20 g over 48 hours) or placebo**
- **Patients treated with methylprednisolone experienced increased all-cause mortality at two weeks (21 versus 18 percent; RR 1.18, 95% CI 1.09-1.27)**

Effect of intravenous corticosteroids on death within 14 days in 10,008 adults with clinically significant head injury (MRC CRASH trial):
randomised placebo-controlled trial.
Lancet 2004 Oct 9;364(9442):1321-1328.

Hyperventilation

PaCO₂ : 26 to 30 mmHg

- ❑ **1 mmHg change in PaCO₂ is associated with a 3 percent change in CBF**
- ❑ **Hyperventilation also results in respiratory alkalosis, which may buffer post-injury acidosis**
- ❑ **The effect of hyperventilation on ICP is short-lived (1 to 24 hours)**

Barbiturates

- ❑ **reduce brain metabolism and cerebral blood flow(neuroprotective)**
- ❑ **Pentobarbital is generally used, with a loading dose of 5 to 20 mg/kg as a bolus, followed by 1 to 4 mg/kg per hr**
- ❑ **Adverse effects:**
 - ◆ **hypotension, possibly requiring vasopressor support.**
 - ◆ **loss of the neurologic examination, requiring accurate ICP, hemodynamic, and often EEG monitoring to guide therapy.**
- ❑ **The therapeutic value is unclear**

Therapeutic hypothermia

Goal :core temperature of 32 to 34°C

- ❑ **decreases cerebral metabolism and may reduce CBF and IC**
- ❑ **lower ICP and improve patient outcomes up to six months after injury**
- ❑ **methods of cooling?**

Removal of CSF

- ❑ **ventriculostomy or CSF diversion**
- ❑ **Therefore, CSF should be removed 3 to 5 mL at a time to gradually reduce ICP and minimize the risk of rebleed**

Decompressive craniectomy

- ❑ **craniectomy alone lowered ICP 15 percent**
- ❑ **opening the dura in addition to the bony skull resulted in an average decrease in ICP of 70 percent**
- ❑ **decompressive craniectomy, improves outcomes in trauma, stroke, and subarachnoid hemorrhage in carefully selected cases**

THANKS