

Circulation in special areas: Brain and Liver

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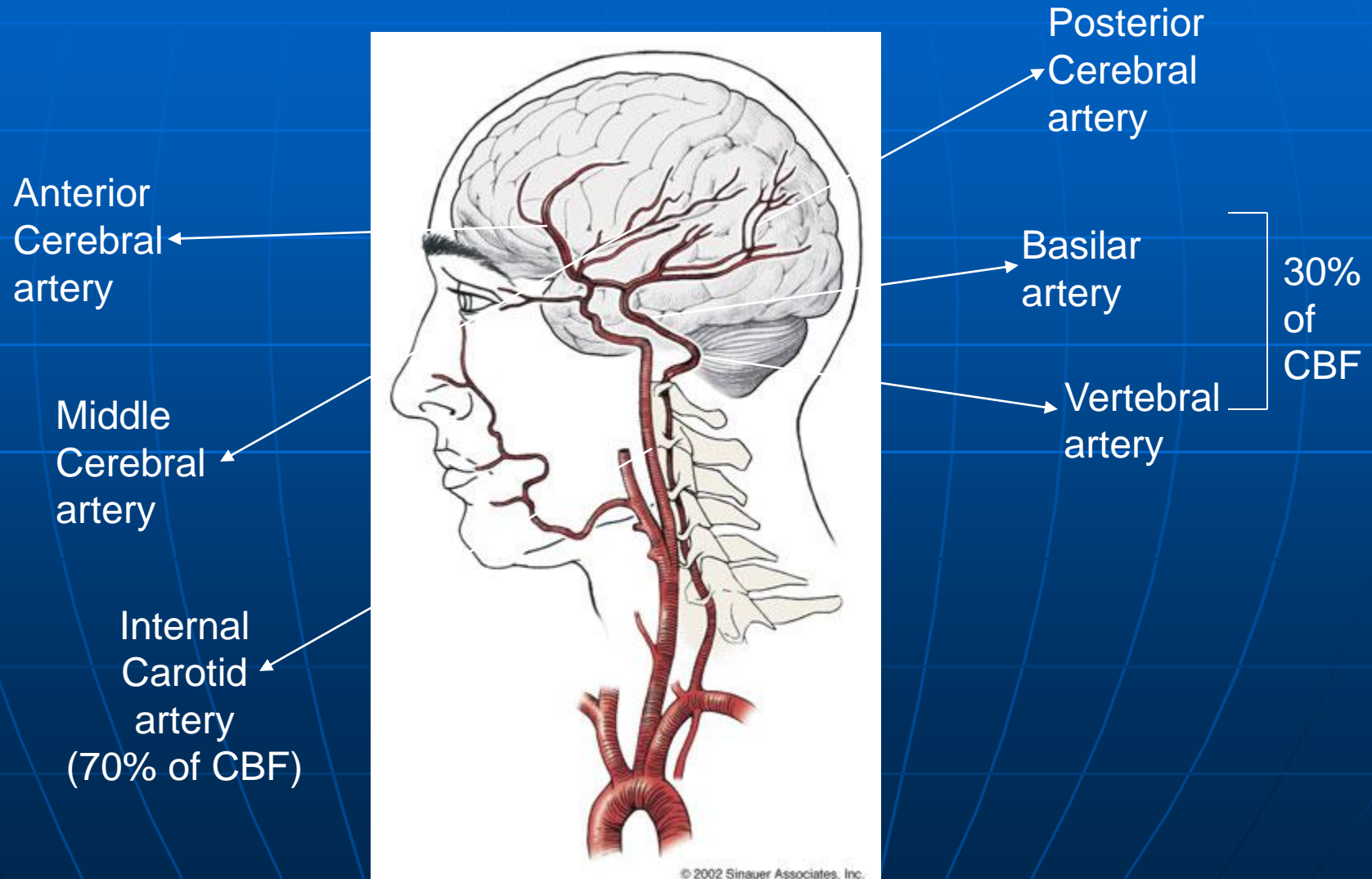
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Objectives

- To understand :
 - Cerebral blood supply (review of anatomy)
 - Cerebral physiology
 - Factors affecting cerebral blood flow
 - Autoregulatory mechanisms
- Review sample questions

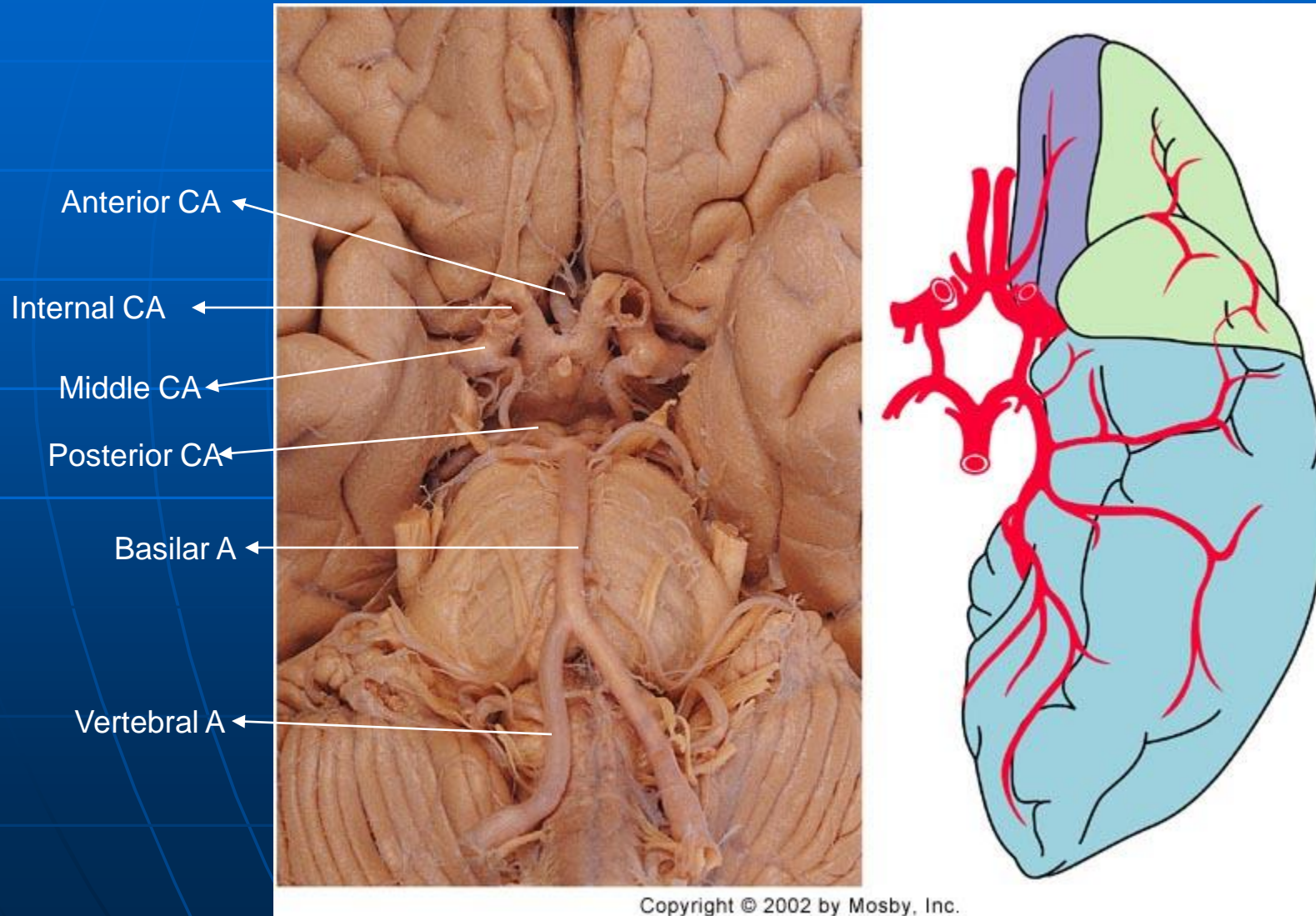
Overview of cerebral circulation

Arterial supply



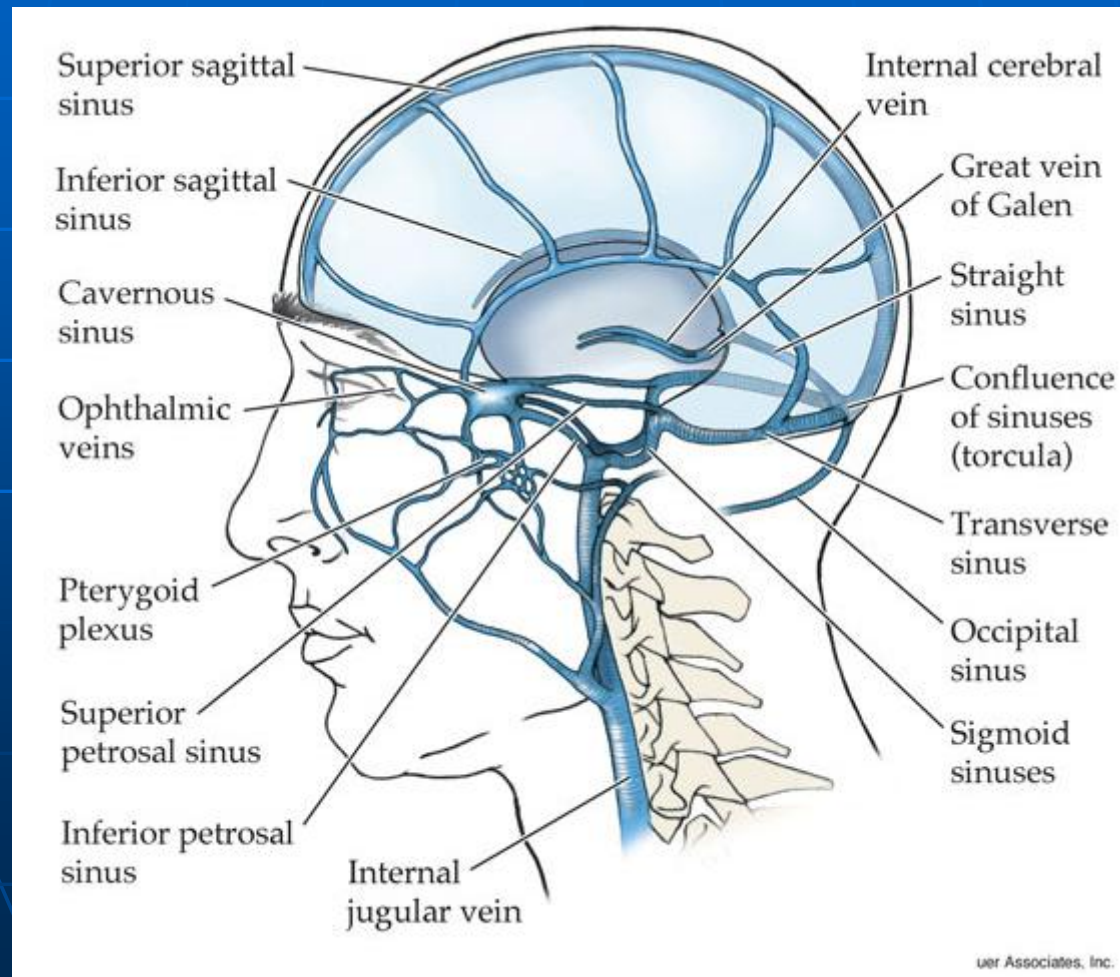
Overview of cerebral circulation

Circle of Willis

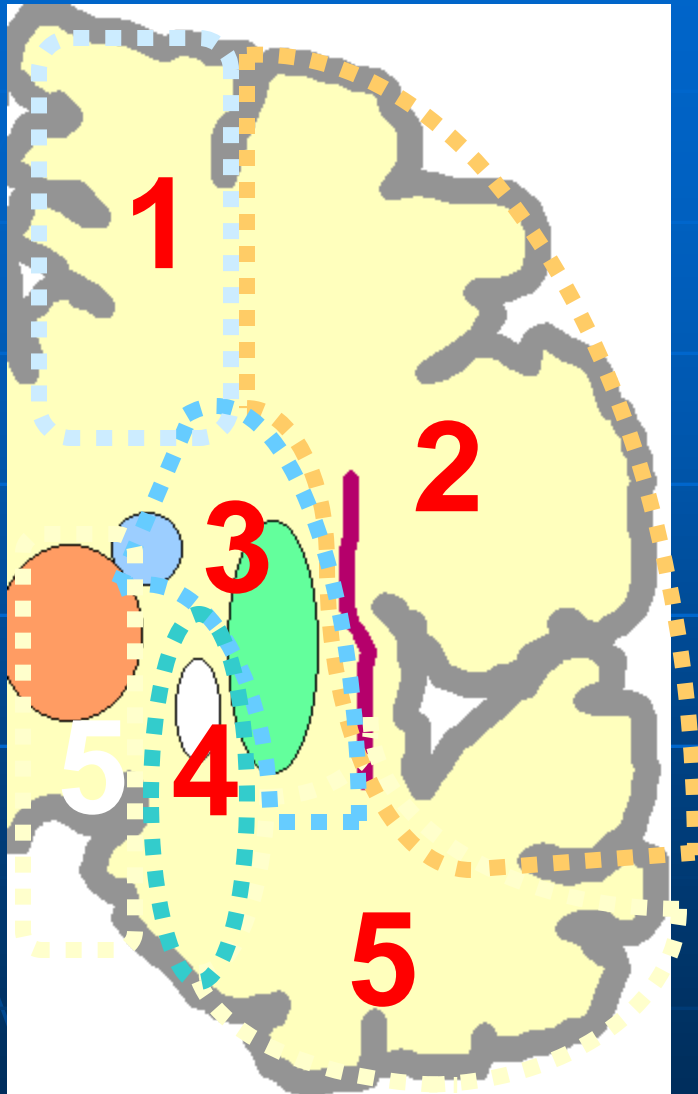


Overview of cerebral circulation

Venous drainage



Cerebral Artery Areas



1. anterior cerebral
2. Middle cerebral
3. Penetrating branches of middle cerebral
4. Anterior choroidal
5. Posterior cerebral

Cerebral physiology I

- 2% of BW (1400 g brain, 75 ml blood
75 ml CSF (*Monro Kellie doctrine*))
- 20% of Total body O₂ consumption
 - (60% used for ATP formation)
- CMR O₂ 3-3.8mL /100 gm/min
 - (50 ml /min in Adult)
- 15% of CO
- Glucose consumption 5mg/100gm/min
 - (25% of total body consumption/min)

CMR , Cerebral Metabolic Rate

Cerebral physiology II

- High oxygen consumption but no reserve
- Grey matter of cerebral cortex consumes more
- Directly proportional to electrical activity
 - (Hippocampus & cerebellum most sensitive to hypoxic injury)

Normal Physiologic Values

CBF	
GLOBAL	45-55ml/100g/min
CORTICAL	75-80ml/100g/min
SUBCORTICAL	20ml/100g/min
CMRO ₂	3-3.5ml/100g/min
CVR	2.1 mmHg/100ml/min/ml
Cerebral venous PO ₂	32-44mmhg
Cerebral venous SO ₂	55%-70%
ICP(supine)	8-12mm Hg

Cerebral physiology III

- Approximately 60 % of the brain's energy consumption is used to support electrophysiological function.
- Remaining 40%-?

Cerebral physiology IV

- Local CBF (I-CBF) and local CMR (I-CMR) within the brain are very heterogeneous, and both are approximately four times greater in gray matter than in white matter.

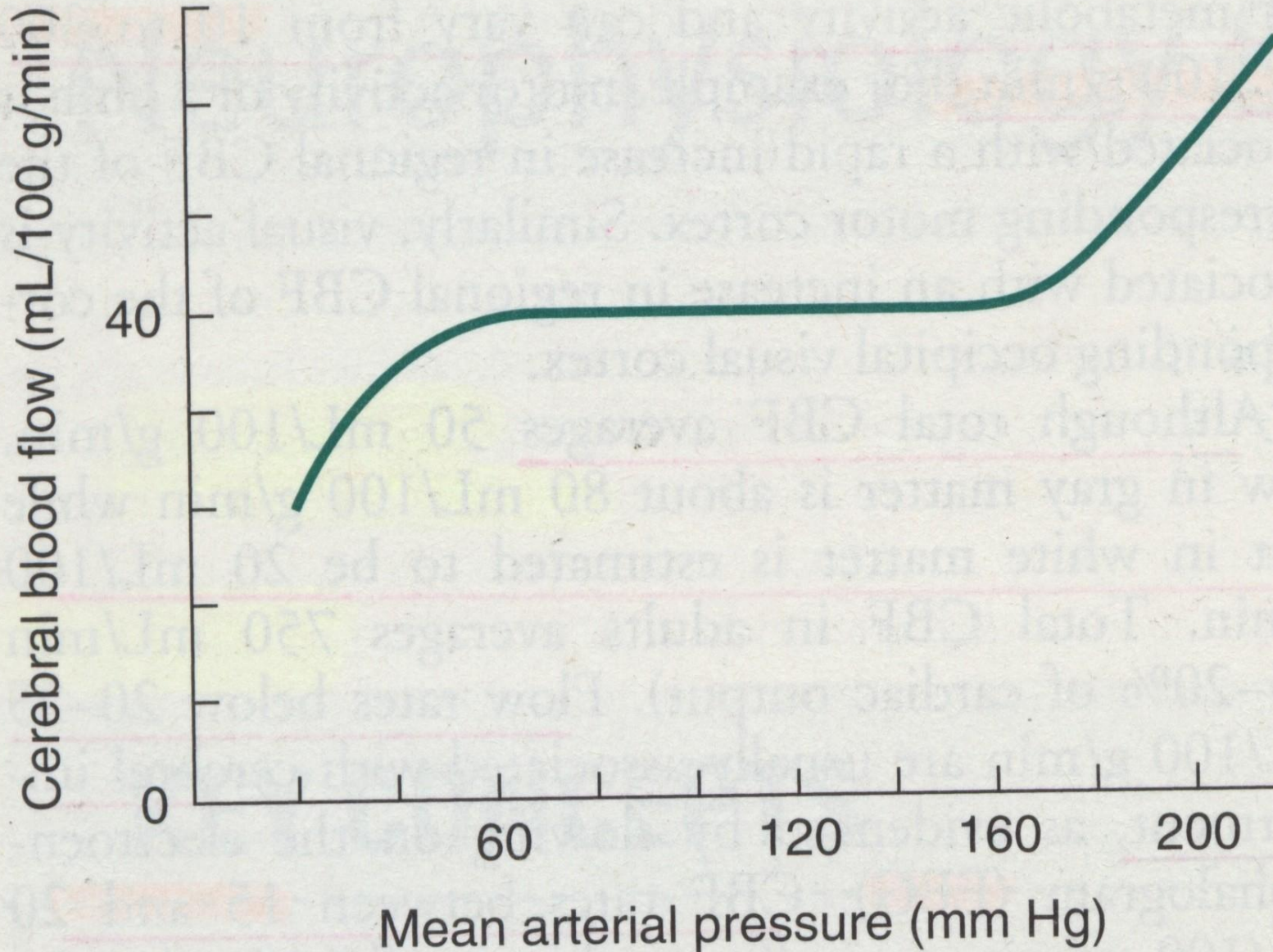
Cerebral physiology V

- The brain's substantial demand for substrate must be met by adequate delivery of O_2 and glucose.
- The space constraints imposed by the noncompliant cranium and meninges require that blood flow not be excessive (**Monro-Kellie doctrine**).
- There are elaborate mechanisms for the regulation of CBF.

Cerebral perfusion pressure (CPP)

- $[MAP - ICP]$ (or CVP whichever is greater)
- Normally 80 to 100mm Hg
- ICP is <10 mmHg so CPP primarily dependent on MAP
- Increase in $ICP > 30 = CPP$ & CBF compromise
- $CPP < 50$ slowing of EEG
 - 25-40 Flat EEG
 - $CPP < 25$ result in Irreversible brain death

CBF: Autoregulation



Factors influencing CBF

- Chemical/metabolic
- Myogenic
- Rheologic
- Neurologic

Chemical/Metabolic

CEREBRAL METABOLIC RATE

Arousal/seizures
mental tasks

Anaesthetics

Temperature

PaCO_2

PaO_2

Vasoactive drugs

Anaesthetics

Vasodilators

Vasopressors

Cerebral Metabolic Rate

- Increased neuronal activity results in increased local brain metabolism
- Although it is clear that local metabolic factors play a major role in these adjustments in CBF, the complete mechanism of flow metabolism coupling remains undefined.

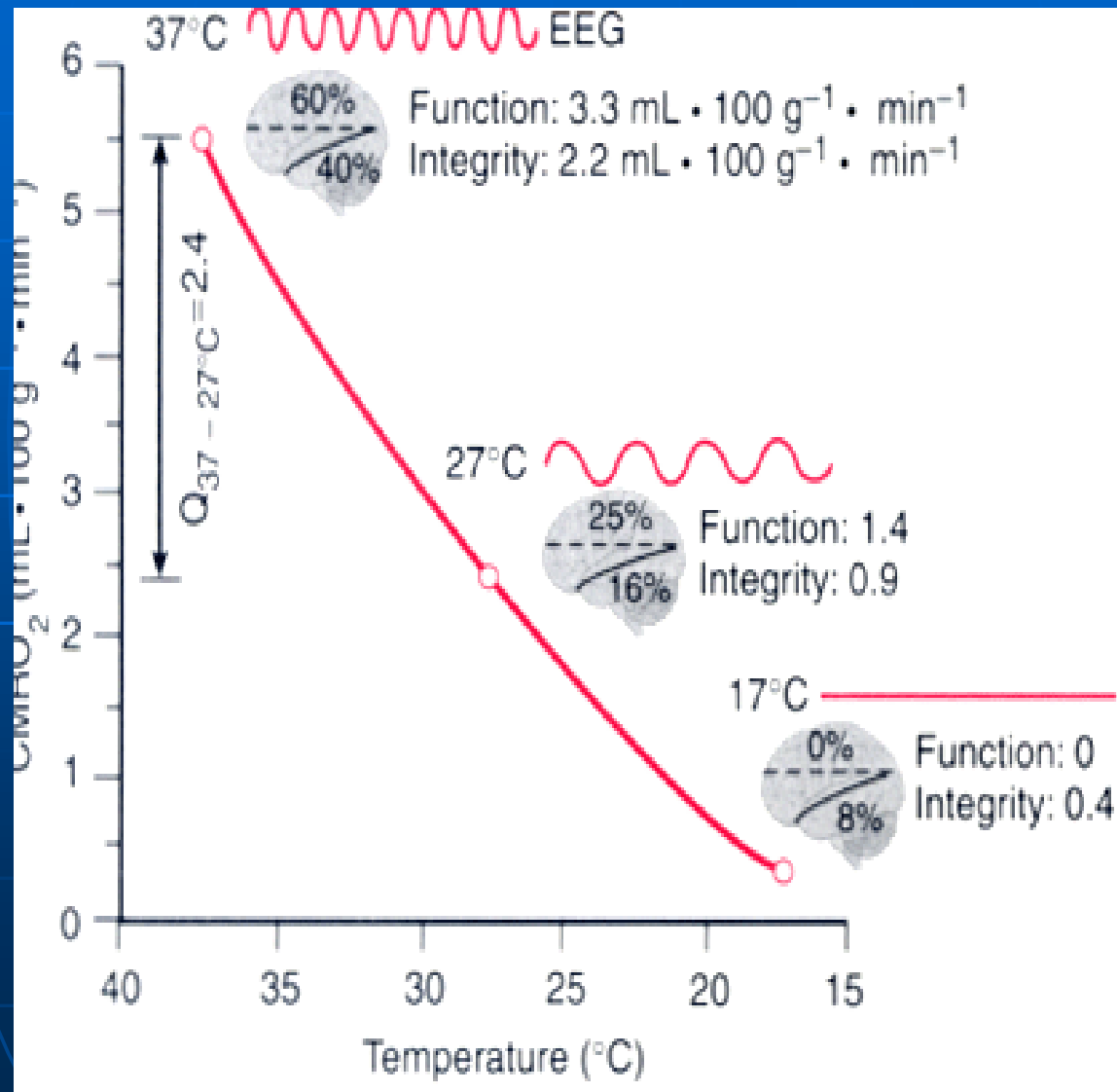
Functional State

- CMR decreases during sleep and increases during sensory stimulation, mental tasks, or arousal of any cause.
- During epileptoid activity, CMR increases may be extreme, whereas CMR may be substantially reduced in coma.



Temperature

- CMR decreases by 6 to 7 % per Celsius degree of temperature reduction.
- However, in contrast to anesthetic agents, temperature reduction beyond that at which EEG suppression first occurs does produce a further decrease in CMR

The effect of temperature reduction on the cerebral metabolic rate of oxygen



Temperature on CBF

- 6-7 % decrease /°C FALL IN TEMP.
- 37-42 °C -  CBF & CMRO₂
- >42 °C -  CMRO₂
- 20 °C - ISOELECTRICITY

Partial Pressure of Carbon Dioxide

- CBF varies directly with PaCO_2
- The effect is greatest within the range of physiologic PaCO_2 variation.
- CBF changes 1 to 2 mL/100 g/min for each 1 mmHg of change in PaCO_2 around normal PaCO_2 values.
- This response is attenuated below a Pa CO_2 of 25 mm Hg.

Effect of PH on CBF I

- Changes in CBF caused by PaCO₂ are dependent on pH alterations in the ECF of the brain
- In contrast to respiratory acidosis, acute systemic metabolic acidosis has little immediate effect on CBF because the blood-brain barrier (BBB) excludes the H⁺ ion from the perivascular space.

Effect of PH on CBF II

- Although the CBF changes in response to Pa CO₂ alteration occur rapidly, they are not sustained.
- In spite of the maintenance of an elevated arterial pH, CBF returns to normal over 6 to 8 hours because cerebrospinal fluid (CSF) pH gradually normalizes as a result of the extrusion of bicarbonate.

Homework

- Read about ...
 - >> Steal Phenomenon
 - >> Inverse Steal or Robin Hood Phenomenon

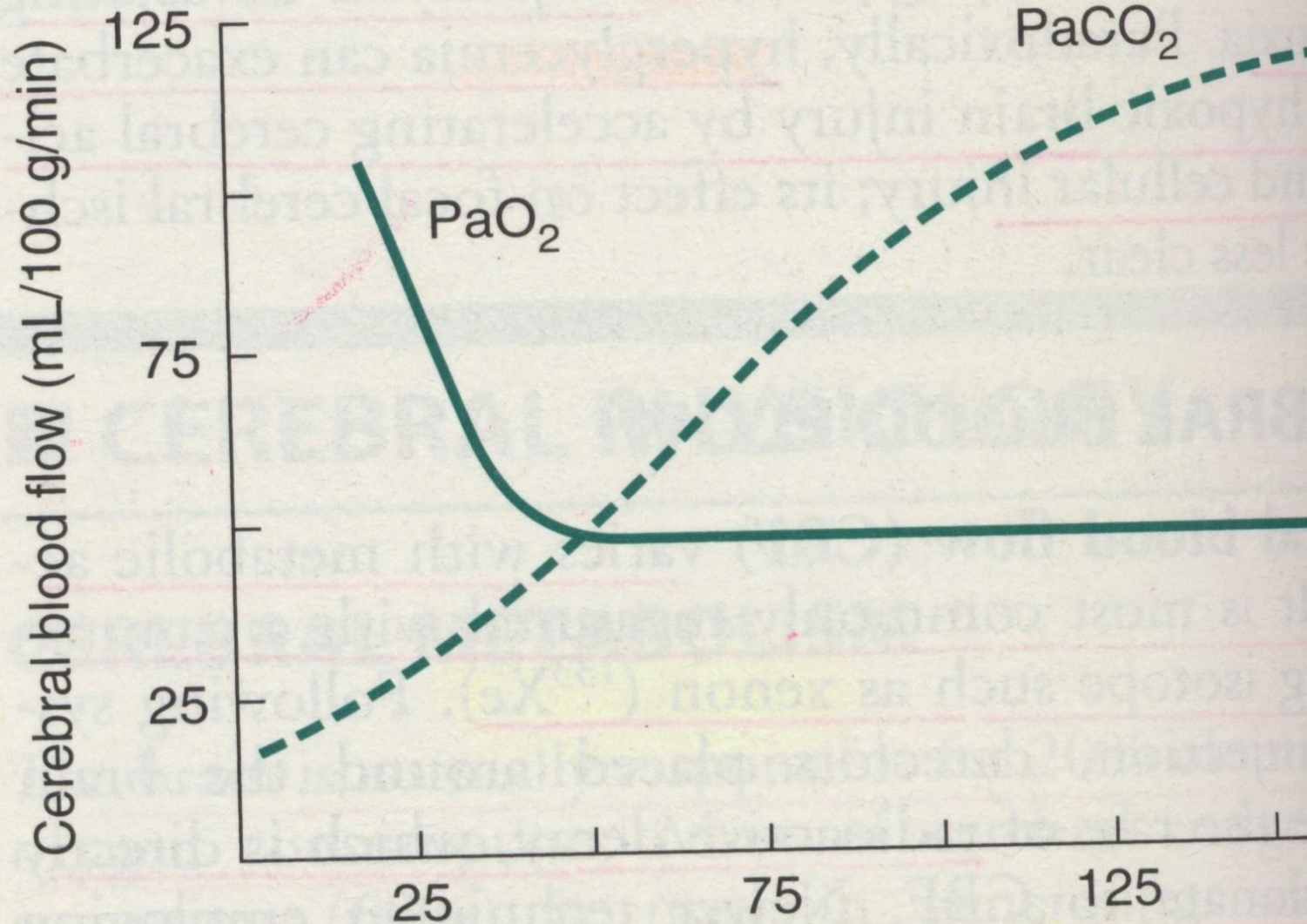
Partial Pressure of Oxygen

- Changes in PaO_2 from 60 to more than 300 mmHg have little influence on CBF.
- When the PaO_2 is less than 60 mmHg, CBF increases rapidly
- At high PaO_2 values, CBF decreases modestly.

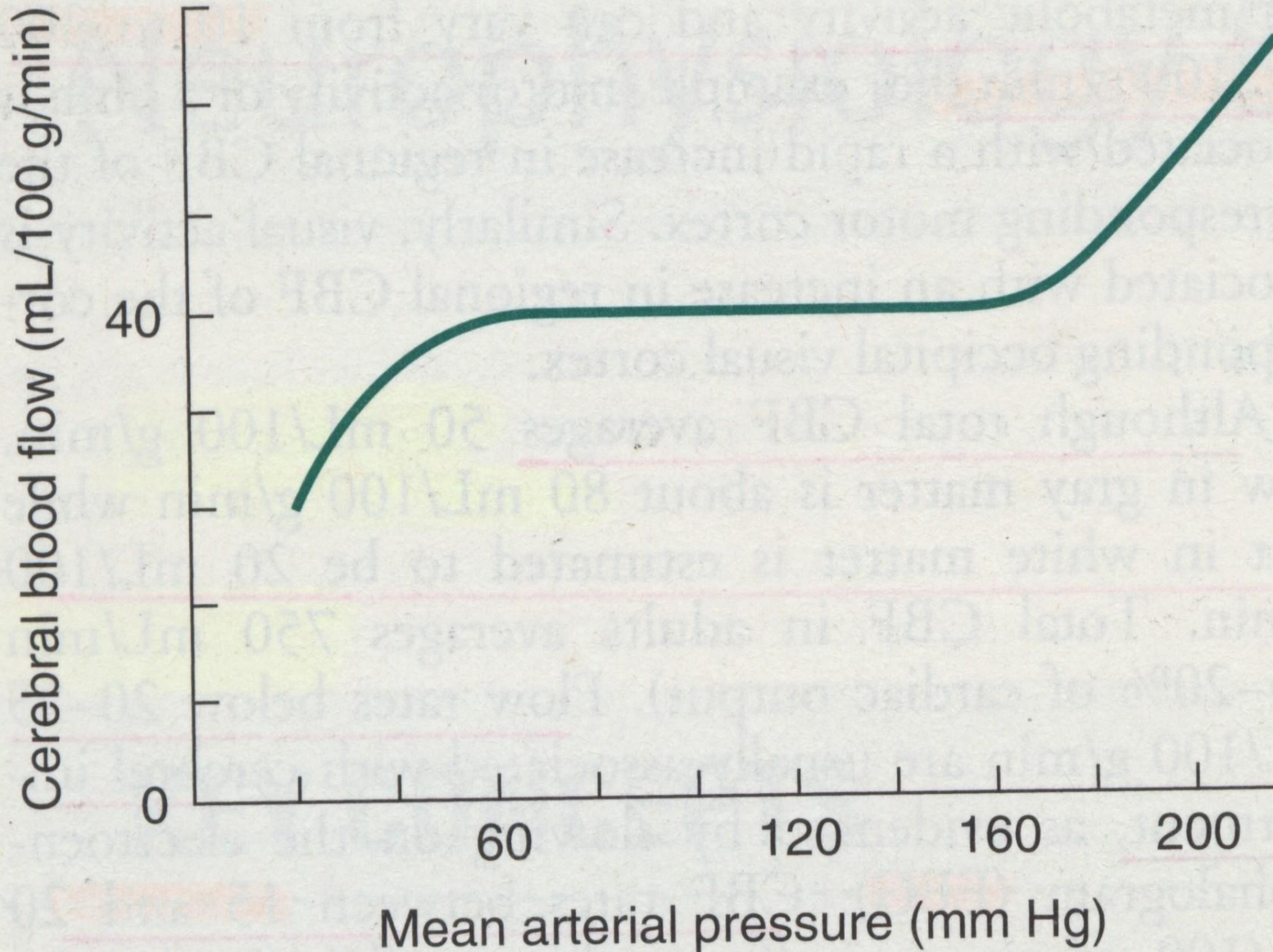
Effects of cerebral hypoxia

- The mechanisms mediating the cerebral **vasodilation** during hypoxia are not fully understood, but they may include **neurogenic** effects initiated by peripheral and/or neuraxial chemoreceptors as well as local humoral influences
- At 1 atm O_2 , CBF is reduced by 12 percent.

CBF vs. PO_2 and PCO_2



CBF: Autoregulation



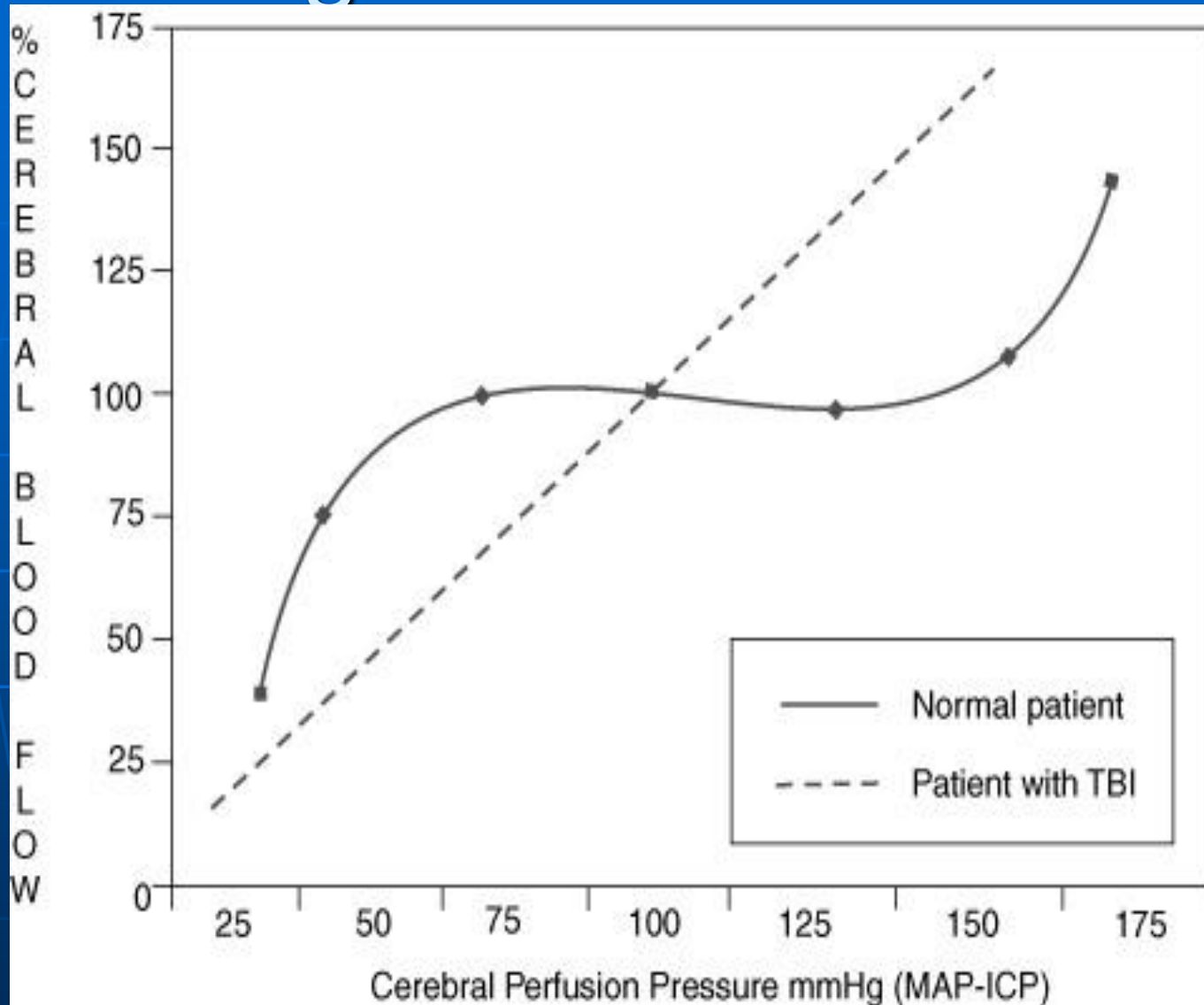
Myogenic Regulation (Autoregulation)

- Autoregulation refers to the capacity of the cerebral circulation to adjust its resistance in order to maintain CBF constant over a wide range of mean arterial pressure (MAP).

Autoregulation in humans I

- In normal human subjects, the limits of autoregulation occur at MAPs of approximately 70 and 150 mm Hg
- Above and below the autoregulatory plateau, CBF is pressure dependent (pressure passive) and varies linearly with CPP.

Autoregulation in humans II



TBI, Traumatic Brain Injury

Autoregulation in humans III

- Autoregulation Curve shift to Right in Chronic hypertension
- Decreased CPP leads to vasodilation
- Increased CPP leads to vasoconstriction

Autoregulation in humans IV

- The precise mechanism by which autoregulation is accomplished is not known
- Nitric Oxide (NO) may participate in the vasodilation associated with hypotension in some species, but not, according to a single study, in primates

Neurogenic Regulation I

- There is considerable evidence of extensive innervation of the cerebral vasculature.
- The density of innervation declines with vessel size, and the greatest neurogenic influence appears to be exerted on larger cerebral arteries

Neurogenic Regulation II

- This innervation includes **autonomic**, serotonergic, and vasoactive intestinal peptide-ergic (**VIPergic**) systems of extra-axial and intra-axial origin.

Viscosity Effects

- Blood viscosity can influence CBF.
- Haematocrit is the single most important determinant of blood viscosity
- In healthy subjects, haematocrit variation within the normal range (33-45%) results in only trivial alteration of CBF.
- Beyond this range, changes are more substantial.

**THANK YOU FOR YOUR
ATTENTION**

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