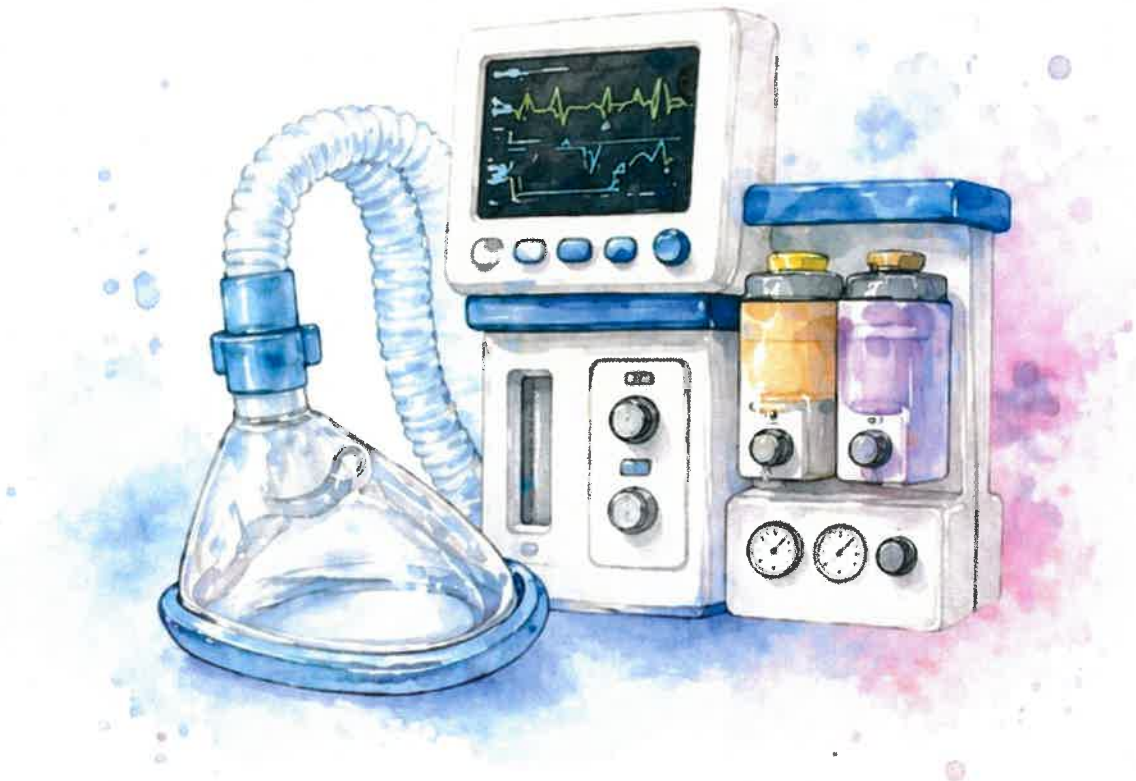
NEET SS ANESTHESIA

Updated Notes 2026



NEURO ANAESTHESIA

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PHYSIOLOGY FOR NEUROANAESTHESIA

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Energy

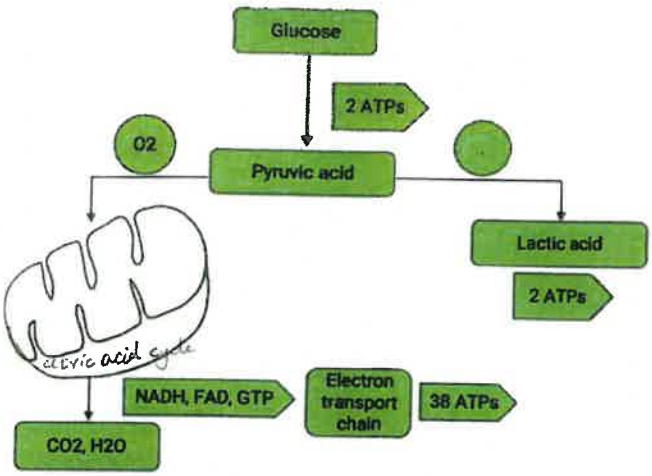
00:03:48

Features :

- Supply (Cerebral blood flow) vs demand (Cerebral metabolic rate).
- To maintain function of ion channels, resting membrane potential (-94 mV), neuronal function.
- To maintain cellular structure and integrity.
- For production of neurotransmitters.

Aerobic & anaerobic metabolism :

- Glucose is the main energy substrate of the brain.
- Energy = ATP.
- Glucose is not freely permeable across the blood brain barrier and requires a transporter to enter the brain, which is not energy dependent.
- The glucose transporters move glucose only down its concentration gradient.
- Glucose uptake into cells occurs via :
  - GLUT1 into astrocytes.
  - GLUT3 into neurons.
  - GLUT5 into microglial cells.



Citric acid cycle

Energy distribution in brain :

- 60% Of the energy produced is utilized for the functioning of the neurons (i.e. Their chemical and electrical activity) : Reduced by burst suppression.
- 40% To maintain the integrity and homeostasis of the neuronal cells : Reduced by hypothermia.
- Glucose metabolism :
  - 70% Of glucose entering the cells undergoes oxidation using the glycolytic and citric acid cycle.
  - 30% Converted to amino acids, proteins and lipids.
  - Lactic acid generated acts as a key energy substrate during periods of high metabolic activity and stress.

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Note : To reduce metabolic activity of a part of brain, induce burst suppression.

Changes during stress :

- Metabolic reserves are very limited in brain.
- Glycogen stores within the brain are exhausted after 2–3 min.
- Blood sugar levels < 4 mmol/L (72 mg/dL) result in glycogenolysis and gluconeogenesis.
- < 3 mmol/L (54 mg/dL) : These compensatory mechanisms fail.
- Clinical manifestations :
  - Altered level of consciousness.
  - Impairment of cognition.
  - During prolonged fasting, the brain adapts to utilize ketone bodies.

Cerebral metabolic rate (CMR) 00:14:29

Features :

- Refers to the rate at which the brain utilizes metabolic substrates, such as oxygen ($CMRO_2$) and glucose (CMR glu), or generates by products like lactate (CMR lact).
- The brain has the highest metabolic requirements of any organ in the body, which is reflected by its high blood flow.
- Brain metabolism and oxygen consumption :
  - The brain is a remarkably complex organ that requires a continuous supply of oxygen and nutrients to function optimally.
  - It consumes approximately 20% of the total oxygen.
  - Loss of consciousness occurs within seconds if there is insufficient blood flow (Ischemia) to the brain, leading to potential brain damage within 3 to 8 minutes.

Cerebral Blood Flow (CBF) :

- Although the brain constitutes only 2% of body mass, it receives a substantial proportion (12–15%) of the resting cardiac output in adults.
- Brain's blood supply comes : Internal carotid and vertebral arteries.
- Grey matter (Composed of neuronal cell bodies) requires a larger share of arterial blood supply due to its involvement in complex functions.
- White matter (Composed of axons) transmits impulses and needs a smaller fraction of blood supply.

Note : During stroke, infarcts occur more commonly in grey matter.

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Parameter	Normal range
Cerebral Blood Flow (CBF)	Approximately 50 ml/100 g/min
Cerebral Oxygen Delivery (Cerebral DO_2)	150–300 ml/min (Assuming Hb level of 150 g/L
CMRO (Cerebral metabolic Rate of Oxygen)	Approximately 3.8 ml/100 g/min
Cerebral Oxygen Extraction Ratio (CO ER)	35% – 25%
Jugular Bulb Venous Saturation ($SjvO_2$)	55% – 75%
Cerebral glucose consumption	6.3 mg/100 g/min

CMR and temperature :

- For each 1°C decrease in body temperature, $CMRO_2$ drops by approximately 7%.
- CBF is nearly halved at a temperature of 27°C and the CMRO2 is as low as 10% of normal at 18°C, allowing preservation of brain function during episodes of DHCA.
- Cooling to 32–34°C is recommended in post cardiac arrest patients and as a treatment of raised ICP refractory to other treatment modalities.
- Major suppression of neuronal function occurs between 17 ° and 27 °C.
- Hyperthermia, on the other hand, increases CMR and CBF between 37° and 42°C, after which protein degradation occurs with a resultant decrease in $CMRO_2$.
- metabolic temperature coefficient (Q10) :
  - Defined as the ratio of $CMRO_2$ at temperature T, divided by the $CMRO_2$ at a temperature that is 10°C lower (T – 10).
  - Normal Q10 = 2.0 and 3.0.
  - Below 27°C, Q10 increases to near 4.5.

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Study	Objective	Findings	Conclusion
meta analysis of therapeutic hypothermia in adult TBI patients	Evaluate risks and benefits of therapeutic hypothermia management in TBI patients	Increased mortality in the therapeutic hypothermia group Reduced risk of unfavorable functional outcome with hypothermia. Increased risk of pneumonia with hypothermia.	Hypothermia did not reduce overall mortality but might benefit TBI patients with elevated intracranial hypertension when initiated within 24 hours
Prophylactic hypothermia after Severe TBI	Assess prophylactic hypothermia after severe TBI	Low grade recommendation for using prophyiactic hypothermia Largest randomized controlled trial showed no benefit Hypothermia for more than 48 hours and slow rewarming improved survival	-
Hypothermia in traumatic brain injury surgery	Investigate hypothermia in TBI surgery	NABIS : H II Study : Hypothermia induced early after TBI does not generally lead to improved outcomes might be beneficial in a subgroup of patients undergoing surgery to treat large traumatic hematomas	-
Mild therapeutic hypothermia in animal models	Explore mild hypothermia's impact on TBI in animal models	Reduced mortality, improved behavioral outcomes, and diminished blood-brain barrier disruption in animals subjected to mild therapeutic hypothermia after TBI	-
Early prophylactic hypothermia for neuroprotection	Investigate early prophylactic hypothermia for neuroprotection	Laboratory data shown compelling benefit at a wide range of target temperatures delivered after TBI	-

Flow metabolism coupling :

- Described by Roy and Sherrington in 1890.
- Increase in activity, either regional or general, causes an increase in the CMR which in turn results in proportional increases in blood flow.
- This method of matching oxygen or glucose delivery to metabolic requirements is termed as 'flow metabolism coupling'.
- The change occurs within seconds of increased functional cerebral activity.
- Vasoactive metabolites are released in areas with increase in neuronal activity
- Neural and glial tissue : Production of metabolic by products such as adenosine, nitric oxide (NO), H^+, K^+, Ca^{2+} and lactate which act locally to cause cerebral vasodilatation and hyperemia.

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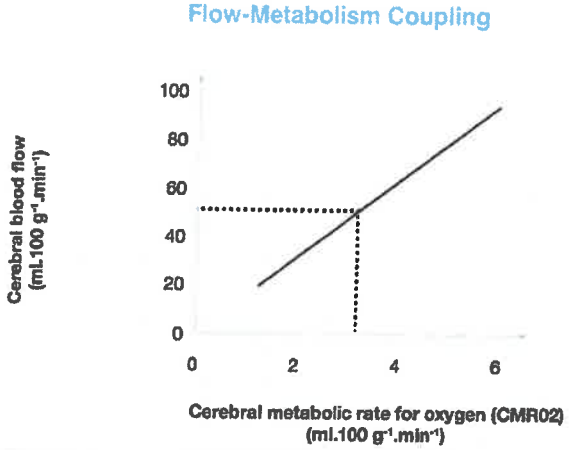
- Astrocytes :
  - Abundant and located surrounding cerebral blood vessels.
  - Ca^{2+} dependent release of neurotransmitters.



Cerebral flow-metabolism coupling. Areas of brain tissue with increased $CMRO_2$ produce increased amounts of vasoactive metabolites, causing local vasodilatation and hyperemia, leading to increased CBF. The dotted line demonstrates normal values for $CMRO_2$ (3.3 mL/100 g/min) and CBF (50 mL/100 g/min).

Dotted line indicates normal values :

- $CMRO_a$ = 3.3 ml/100 g/min.
- CBF = 50 ml/100 g/min.

Cerebral blood flow (CBF) 00:29:00

Basics :

- Receives 15% of cardiac output (700 mL/min or 50 mL/100 g/min).
- Grey matter, composed of the cell bodies of the neurons which are involved with the complex functions of the human body, has higher metabolic requirements, and receives a higher proportion of the arterial blood supply (70 mL/100 g/min).
- White matter, composed of axons which transmit impulses in between the neurons and involved with less complicated functions (20 mL/100 g/min).
- CBF can be described by the Hagen-Poiseuille equation for laminar flow :

$$CBF = \frac{\Delta P \pi r^4}{8 \mu l}$$

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CBF can therefore be affected by :

- Changing the driving pressure (ΔP : The cerebral perfusion pressure (CPP)).
- Altering the cerebral blood vessel radius (r) : This occurs through autoregulation, neurohumoral effects, respiratory gas effects, and cerebral flow metabolism coupling.

Measurement of CBF :

1. PET scan.
2. Single-Photon Emission Computed Tomography (SPECT).
3. Magnetic Resonance Imaging (MR angiography).
4. Thermal clearance.
5. Doppler techniques.
6. Optical methods for clinical assessment of CBF :
   - Jugular venous oximetery, near infrared spectroscopy.
7. Optical methods for preclinical research :
   - Intra Vital Microscopy.
   - Laser Doppler blood flow.
   - Laser Doppler perfusion imaging.
   - Speckled laser Doppler flow mapping.
   - Infrared thermal imaging.
   - Photo Acoustic tomography and functional brain imaging.
   - Two photon microscopy.
   - Optical Coherence Tomography.

Cerebral autoregulation 00:33:46

Cerebral perfusion pressure (CPP) :

- CPP is the difference in the pressures between the arterial and venous circulation which dictates blood flow to the brain.
- mean cerebral venous pressure is hard to measure, and therefore ICP is used as a surrogate.
- Cerebral Perfusion Pressure (CPP) = mean Arterial Pressure (MAP) – Intracranial Pressure (ICP).
- CBF remains constant with CPP in the range of approximately 50 to 150 mm Hg.
- CPP values of < 50 mm Hg lead to cerebral hypoperfusion and ischemia.
- Current guidelines recommends targeting a CPP of 60–70 mmHg in the management of TBI.

Cerebral Perfusion Pressure

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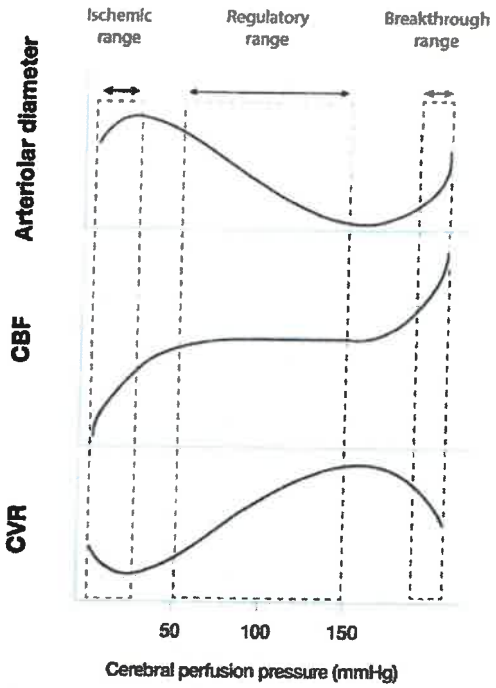
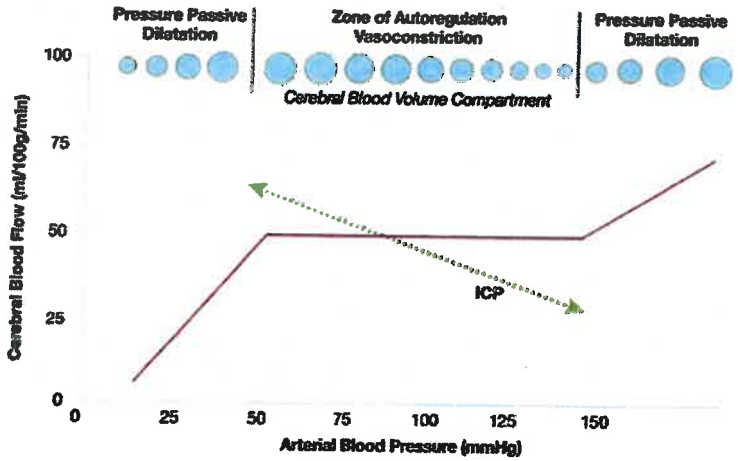


Cerebral autoregulation :

- The process by which the cerebral vasculature maintains a constant CBF across a range of systemic blood pressures or CPPs.
- Introduced initially by Lassen in the 1950.

Intact cerebral autoregulation



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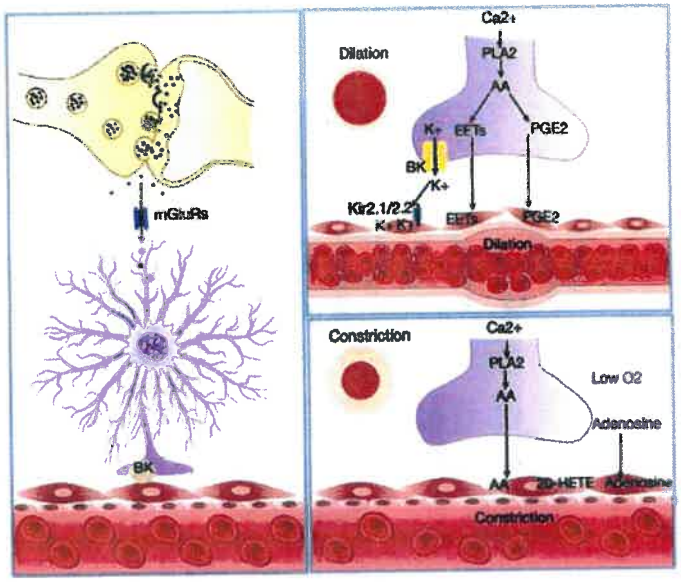
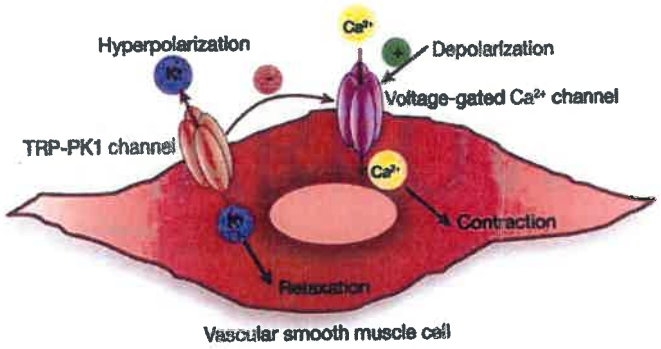
Autoregulation – Neurogenic :

- Autonomic factors do not appear to control the cerebral circulation.
- The cerebral blood vessels are under both sympathetic and parasympathetic control.
- The innervation of the cerebral vasculature is extensive, involving serotonergic, adrenergic, and cholinergic systems of both intracranial and extracranial origin.
- Parasympathetic fibers surround the vessels of the circle of Willis and the cortical pial vessels.
- Input from neurons and glial cells, particularly astrocytes, regulates local blood flow directly by a "feed forward mechanism".
- Whether astrocytes ultimately mediate prorelaxant or procontractile effects may depend on the existing vascular tone and local O_2 concentration.

Mechanisms underlying astrocyte-mediated vascular responses



Mechanism of the TRP-PK1 channel



Autoregulation – myogenic :

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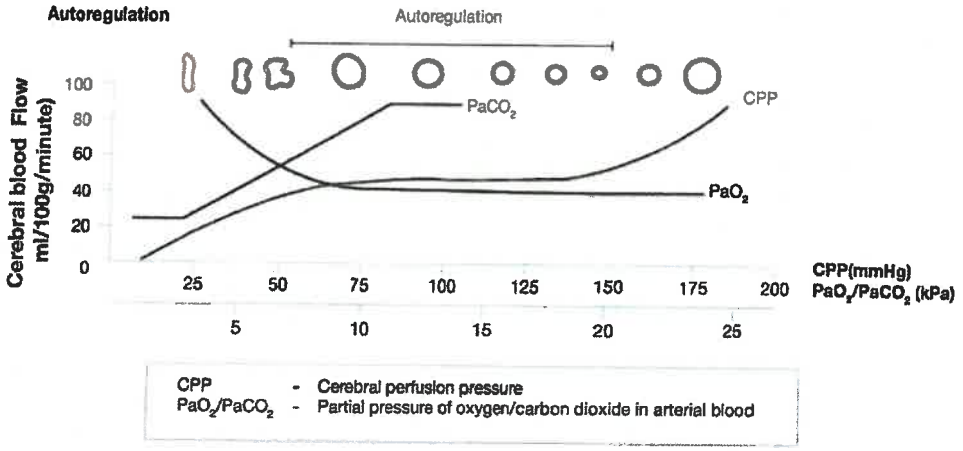
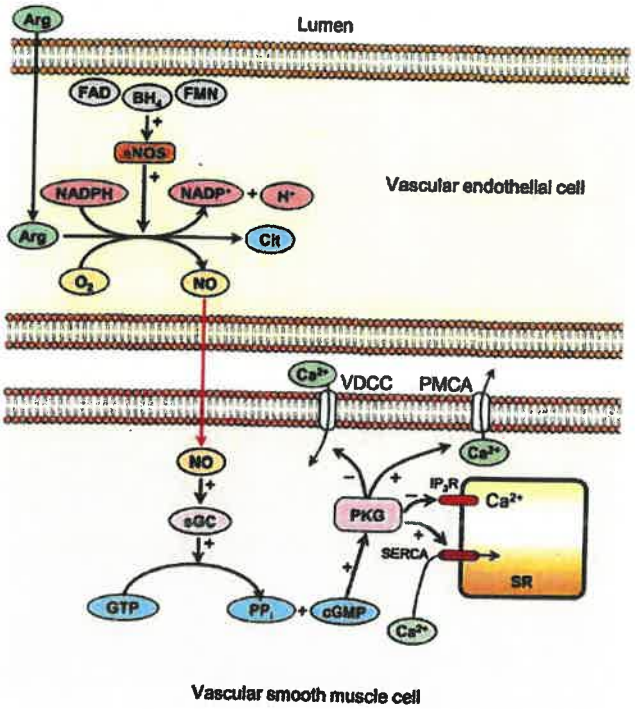
- Mediated by K^+ channels.
- Loss of cerebral metabolic autoregulation is seen in :
  - Traumatic brain injury.
  - Diabetes mellitus, hypertension.
  - Increased age.
  - Dementia.

Autoregulation curves



Autoregulation – Endothelial :

- NO from endothelium cause vasodilatation, as an "endothelium derived relaxing factor".
- NO appears to be formed on demand and is not stored in vesicles.
- The endothelium also produces the vasodilators endothelial derived hyperpolarizing factor (EDHF) and prostacyclin (PGI2).



Autoregulation : Endothelial

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- Prostaglandins in cerebral circulation :
  - PGE2 and PGI2 are vasodilators.
  - Thromboxane A2 and PGF2α are vasoconstrictors.
- Endothelin most likely acts through influx of extracellular calcium, which is probably mediated by protein kinases.
- Endothelin has been implicated in vascular spasm after SAH

Autoregulation (metabolic) :

CO_2 reactivity :

- CBF is extremely sensitive to changes in CO_2.
- For each 1 mm Hg change in $PaCO_2$, CBF changes by 4%.
- Rapid diffusion across the blood-brain barrier (BBB) allows CO_2 to modulate extracellular fluid pH and affect arteriolar resistance.
- In general, doubling PaCO2 doubles CBF and vice versa.

	Hypercapnia	Hypocapnia
Cerebral blood vessels	Vasodilatation	Vasoconstriction
Plateau of autoregulation	Upward shift	Lowers
Lower limit of autoregulation	Rightward shift	Small change
Upper limit of autoregulation	Leftward shift	No evidence to suggest any effect
ODC	Shift to right	Shift to left

Relationship between hyperventilation and ICP :

- Hyperventilation leads to a relative hypocapnia.
- Subsequent vasoconstriction.
- Temporary measure in the management of acutely raised ICP.

Cerebrospinal fluid (CSF) 00:38:47

Physical properties :

- Clear aqueous solution.
- Produced by the ependymal cells of the choroid plexus in the lateral, third and fourth ventricles.
- Produced at a rate of 0.35 - 0.40 mL/min (500-600 mL/day).
- Total volume : 140-150 mL in adults.
- Turnover time for total CSF volume is 5 to 7 hours.
- Turnover rate of about 4 times per day.

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CSF production :

- Within the choroid plexus occurs 40% from by ultrafiltration of plasma through fenestrated capillaries, with the addition of water and other dissolved substances by active transport across the blood : CSF barrier.

Biochemical properties :

- The lower specific gravity of CSF (1.007) relative to brain tissue (1.040) reduces the effective mass of the brain from 1400 g to only 47 g, enabling it to support the brain and protect against acceleration and deceleration forces against the skull.
- The acid–base characteristics of CSF influence respiration, CBF, autoregulation of CBF and cerebral metabolism.
- CSF calcium, potassium, and magnesium levels influence heart rate, blood pressure, vasomotor and other autonomic reflexes, respiration, muscle tone, and emotional states.

Substance	CSF	Plasma
Sodium (Na^+)	144–152 mmol/L	135–145 mmol/L
Potassium (K^+)	2.0–3.0 mmol/L	3.8–5.0 mmol/L
Glucose (Fasting)	2.5–4.5 mmol/L	3.0–5.0 mmol/L
Calcium (Ca^{2+})	1.1–1.3 mmol/L	2.2–2.6 mmol/L
Magnesium (Mg^{2+})	1.3–1.5 mmol/L	0.8–1.0 mmol/L
Chloride (Cl^-)	123–128 mmol/L	100–110 mmol/L
Phosphate (PO_4^{3-})	0.4–0.7 mmol/L	0.81–1.45 mmol/L
Urea	2.0–7.0 mmol/L	2.5–6.5 mmol/L
Bicarbonate ($HCO3^-$)	24–32 mmol/L	24–32 mmol/L
Protein	200–400 mg/L	60–80 g/L
pH	7.28–7.32	7.35–7.45
Osmolality	280–300 mmol/kg	275–295 mmol/kg
Specific gravity	1.006–1.008	1.010–1.020

CSF production :

- Osmotic forces appear to play a major role in water movement.
- Pericapillary spaces provide less restricted passage of water and electrolytes than most of the cerebral vasculature.
- This glucose rich and protein poor "lymph" diffuses through the ECF space toward the macroscopic CSF spaces.
- 60% of extrachoroidal CSF formation results from oxidation of glucose (into water and carbon dioxide) by the brain.

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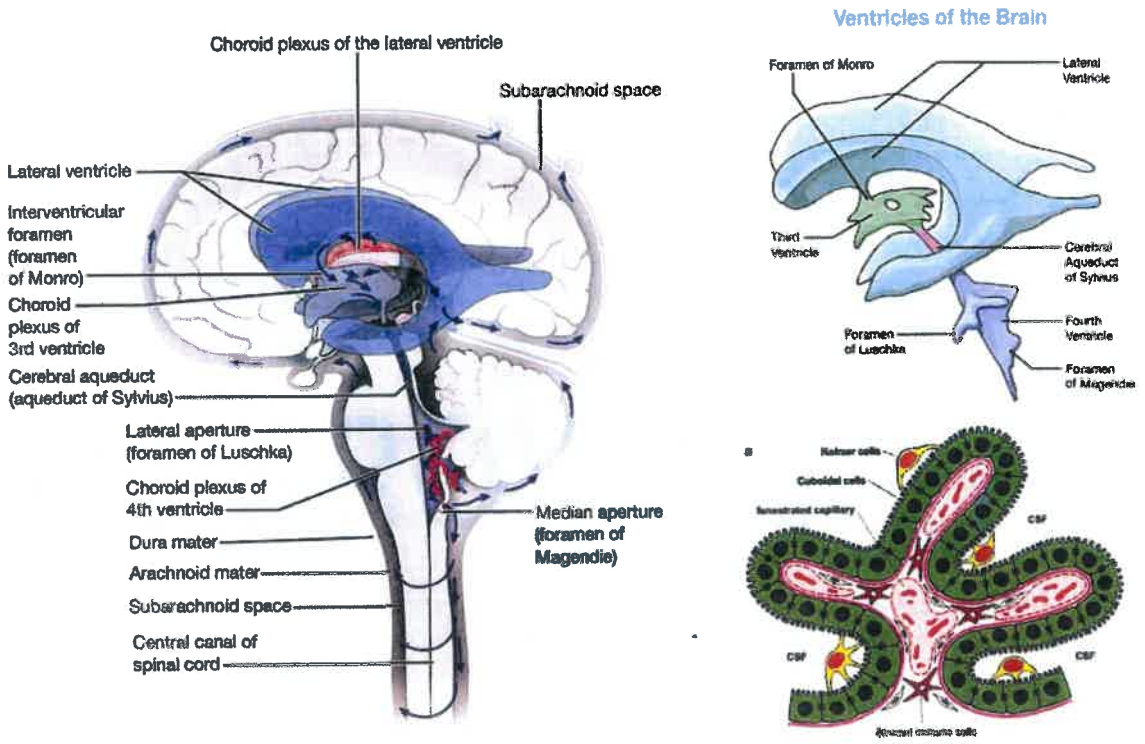
- Production is partly dependent on CPP, with a pressure below 70 mm Hg causing a reduction in CSF production due to the reduction in cerebral and choroid plexus blood flow.

CSF drainage :

- The hydrostatic pressure of CSF formation, 15 cm H_2O, produces CSF flow.
- Cilia on ependymal cells generate currents that propel CSF toward the fourth ventricle and its foramina into the subarachnoid spaces.
- CSF reabsorption occurs across microscopic arachnoid villi and macroscopic arachnoid granulations, down a pressure gradient of 6 cm H_2O between the CSF (mean pressure : 15 cm H_2O) and superior sagittal sinus (mean pressure : 9 cm H_2O).
- 85% to 90% of CSF is reabsorbed at intracranial sites, and 10% to 15% at spinal sites.
- Newer studies add the role of CSF drainage into lymphatic pathways and CSF reabsorption throughout the entire CSF-interstitial fluid interface.



CSF drainage

Changes in CSF formation :

- Hypothermia decreases rate of CSF formation, probably by reducing the activity of active secretory and transport processes and by decreasing CBF.
- Each 1°C reduction in temperature between 41° and 31°C decreases rate by 11%.

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- Reduced osmolarity of ventricular CSF or increased osmolarity of serum decreased rate of CSF formation, and vice versa.
- Prolonged hypercapnia or hypocapnia does not significantly change the rate of CSF formation.
- metabolic acidosis does not change rate of CSF formation, but metabolic alkalosis decreases rate of CSF formation, presumably as a result of a pH effect unrelated to ion or substrate availability.

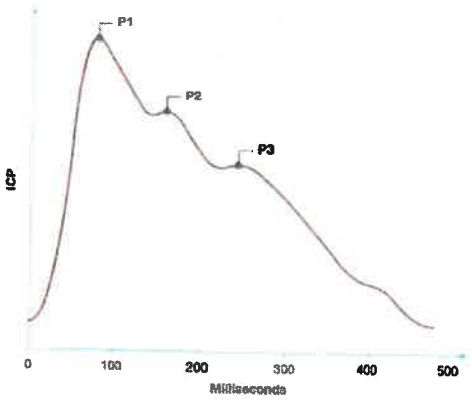
Intracranial pressure (ICP) :

- Pressure within the intracranial cavity relative to atmospheric pressure.
- Normal ICP ranges from 5 to 15 mm Hg.
- Varies significantly between individuals and with posture.
- ICP is a dynamic pressure waveform, with variation in amplitude due to cardiac and respiratory cycles.

Intracranial pressure waveform :

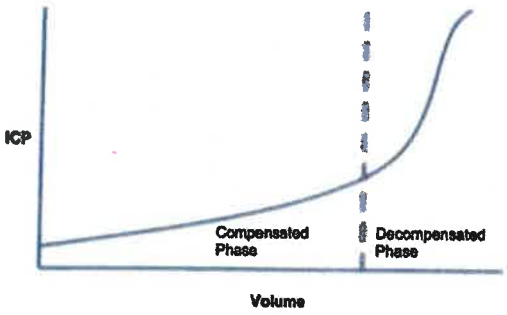
- Has three distinct phases, P1, P2, and P3, together known as the "vascular pulse".
- P1 : Percussion wave, represents transmitted cerebral arterial pulsation from the choroid plexus.
- P2 : Tidal wave, represents intracranial compliance.
- P3 : Dicrotic wave, represents aortic valve closure.
- During the respiratory cycle, there is variation in amplitude between consecutive waves, known as the "Respiratory pulse".

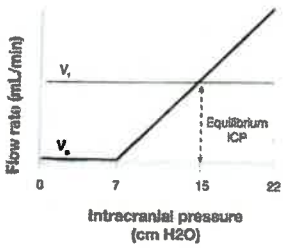
ICP waveform

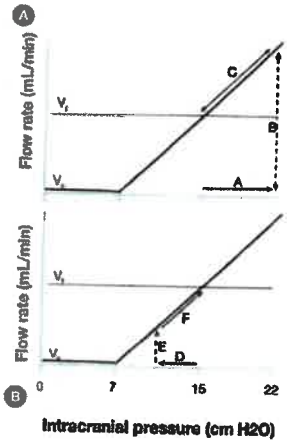


Intracranial pressure volume relationship :

ICP vs Flow rate curves







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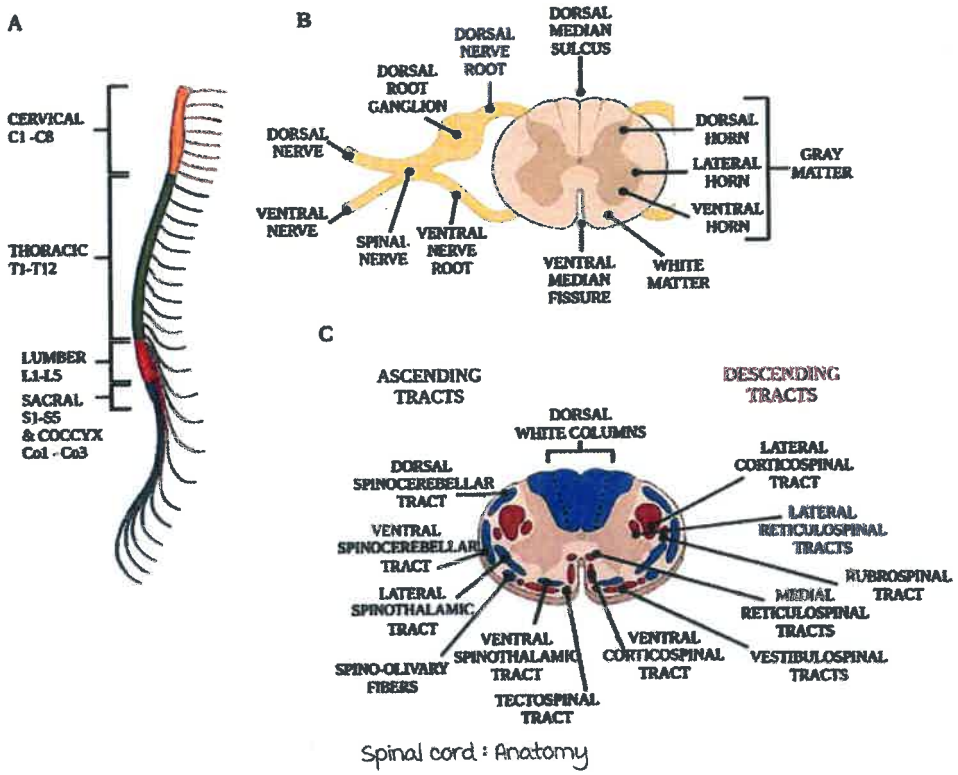
Spinal cord

00:45:29

Anatomy :

- Spinal cord extends from the medulla oblongata at the foramen magnum to the conus medullaris and cauda equina at the level of L1 or L2 in an adult (L2 or L3 in a neonate).
- 31 Pairs of spinal nerves exit the spinal cord, 8 cervical, 12 thoracic, 5 lumbar, 5 sacral, and 1 coccygeal.



Spinal cord : Anatomy

myotomes :

Nerve root	muscle groups innervated
C1-C4	Diaphragm (Via the phrenic nerve) Neck flexors (E.g., sternocleidomastoid, scalenes) Shoulder muscles (Trapezius, levator scapulae) Some intrinsic muscles of the hand
C5	Deltoid Biceps brachii Brachialis Supinator
C6	Wrist extensors (Extensor carpi radialis longus, extensor carpi radialis brevis) Biceps brachii (Continuation) Brachialis (Continuation)