Conduction Velocity of Nerve fibers:

Fiber type	Function	Fiber diameter(μm)	Conduction velocity(m/s)	Spike duration(ms)
Aα	Proprioception, somatic motor	12-20	70-120	
Aβ	Touch, pressure	5-12	30-70	0.4-0.5
Aγ	Motor to muscle spindles	3-6	15-30	
Aδ	Pain, temperature	2-5	12-30	
B	Preganglionic autonomic	<3	3-15	1.2
C Dorsal root	Pain, temperature	0.4-1.2	0.5-2	2
C Sympathetic	Post ganglionic sympathetic	0.3-1.3	0.7-2.3	2

Aα : Motor Nerve, Proprioception

Mnemonic : Alpha male love to ride Motor bike

Aβ : Touch & pressure

↓

बेटा → Naughty

↓

Parents Slap ∴ touch & pressure

Aγ : Motor to muscle spindle

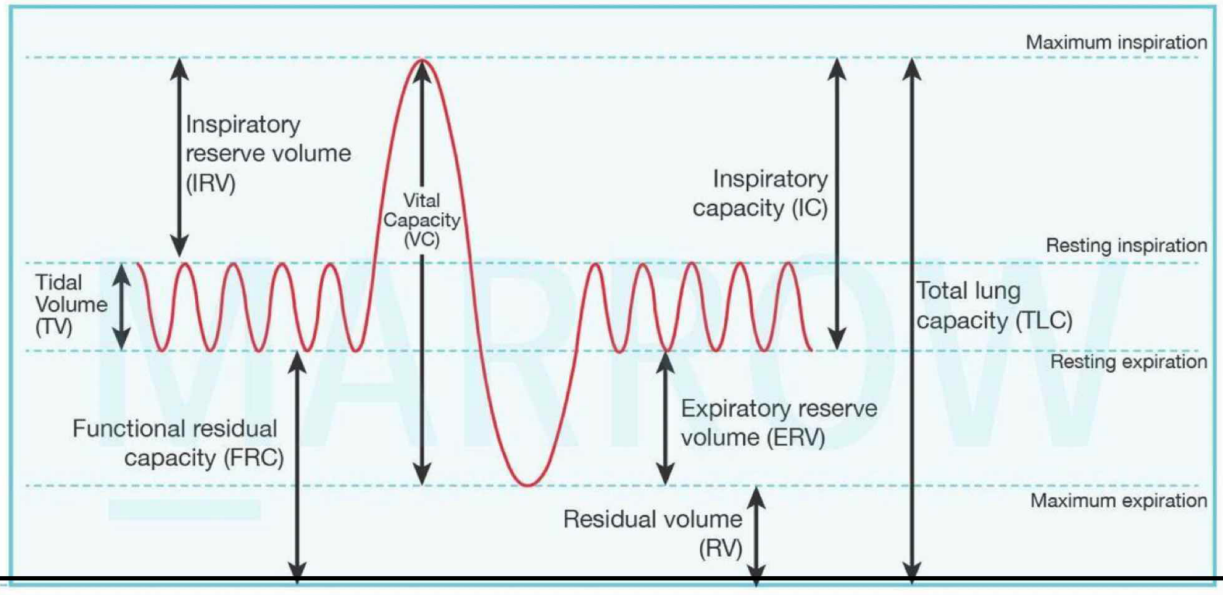
↳ Gamma

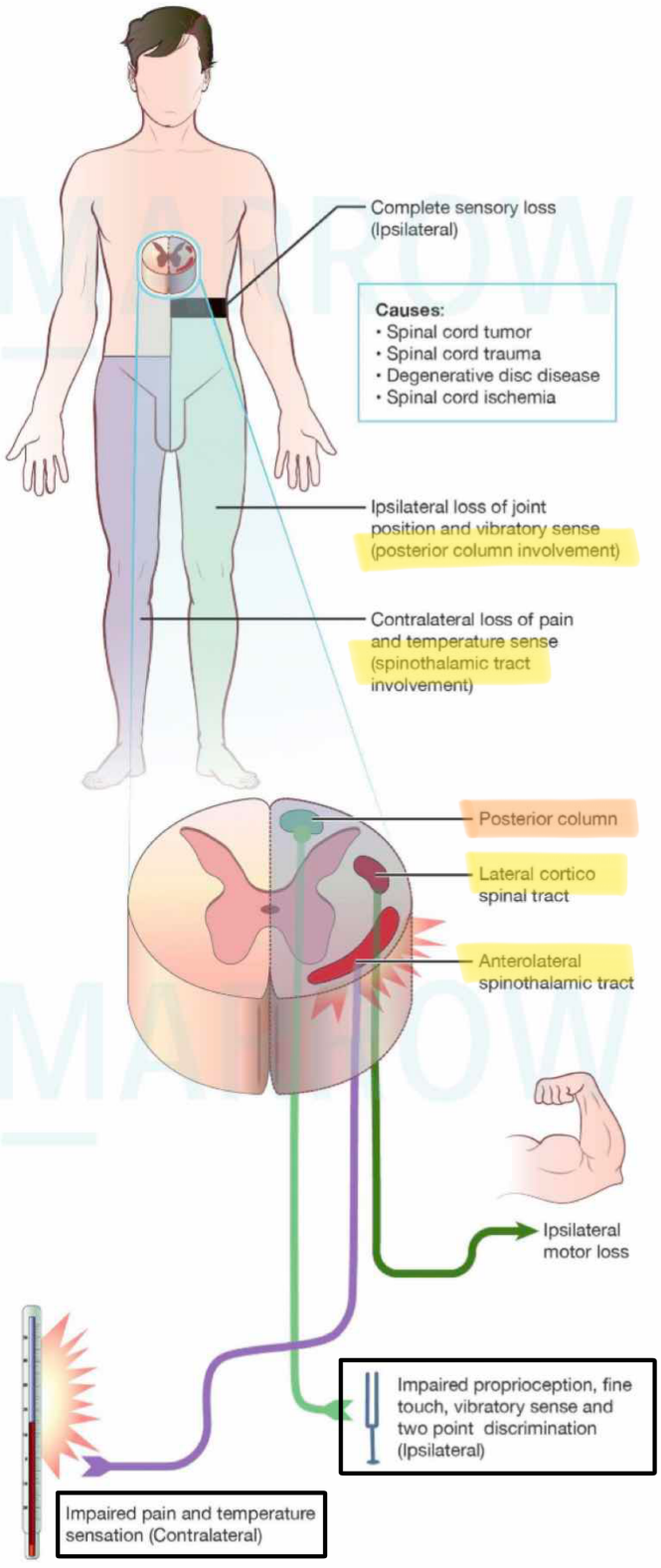
↳ gym grow muscles

Aδ : Pain & Temprature Sensation

Measurement	Description	Normal Value
Tidal volume (TV)	The amount of air that moves into the lungs with each inspiration (or the amount that moves out with each expiration) during quiet breathing	500–750 mL.
Inspiratory reserve volume (IRV)	The air inspired with a maximal inspiratory effort in excess of the TV	~3 L
Expiratory reserve volume (ERV)	The volume expelled by an active expiratory effort after passive expiration	~1.1 L
Residual volume (RV)	The air left in the lungs after a maximal expiratory effort	~1.2 L
Total lung capacity (TLC)	IRV+TV+ERV+RV	~5.8 L
Vital capacity (VC)	IRV+TV+ERV	~4.5 L
Inspiratory capacity (IC)	TV+IRV	~3.5 L
Expiratory capacity (EC)	TV+ERV	~1.6 L
Functional residual capacity (FRC)	ERV+RV	~2.3 L

Lung volumes and Capacities





Brown Sequard Syndrome: is a condition that results from damage (resection) to one half of the brain on either side.

- This hemisection causes damage of the spinal cord tracts which are pathways for transfer of information to and from the brain.
- This syndrome results in the weakness or paralysis (Hemiparaplegia) on one side of the body & loss of sensation (Hemi--anesthesia) on the opposite side.

Caused by: Penetrating trauma such as a puncture wound to the neck or back.

- ischemia (obstruction of a blood vessel)
- Infectious or inflammatory disease such as tuberculosis or multiple sclerosis.

Symptom: Loss of pain & temp. contralaterally for body regions from affected dermatome & down.

- Motor effects: ipsilateral spasticity & weakness.
- loss of fine discrimination touch, vibration, & position sense ipsilaterally for body regions from affected dermato--me

IOC: MRI

Glucose Transporters

→ Glucose is a Hydrophilic compound. It must be transported through the Hydrophobic plasma membrane. Hence Transporter is required.

- SGLT - Sodium dependent Glucose Transporter
- GLUT - Sodium independent Glucose Transporter

SGLT → 2° Active transport

Types	Location	Function
SGLT-1	• intestine : Luminal side • Proximal Renal Tubule	• Absorption of Glucose
SGLT-2	• Proximal Renal Tubule	• Absorption of Glucose

inhibitors are used as OHA's called Gliflozins

GLUT - Facilitated Carrier mediated transport

- **GLUT 1**
  - k - kidney
  - B - Brain
  - C - Colon
  - Par - Placenta
  - Red - RBC
- **GLUT 2**
  - Small - Small intestine
  - kid - kidney
  - Live in - Liver
  - Punjab - Pancreatic β-cells
- **GLUT 3**
  - BP - Brain
  - Kill - kidney
  - People - Placenta
- **GLUT 4** - Insulin stimulated Glucose uptake.
  - H - Heart
  - A - Adipose tissue
  - S - Skeletal muscles
- **GLUT 5**
  - Five - Small intestine for Fructose

High Altitude Physiology:

- Atmospheric or barometric pressure → Low
- The density of air ↓ ∴ high altitude.
- Hypoxic Hypoxia occurs.

We live at sea level:
Barometric pressure: 760 mmHg (1 atm pressure)
Composition: 21% of O_2
Partial pressure of O_2: 160 mmHg (in environmental air)

Changes of acclimatisation:

- ↑ in pulmonary ventilation → Leading to Respiratory alkalosis
- ↑ diffusing capacity of the lungs.
- ↑ numbers of RBCs. (↑ erythropoietin Release)
- ↑ vascularity of peripheral tissues. (Angeogenesis)
- ↑ ability of tissue cells to use O_2, despite low P_{O_2}.

Acclimatisation	Adaptation
Temporary change	Permanent change
Happens over a short time	Happens over generations
Genetic makeup is not affected	Changes are inherited genetically

- Right shift in O_2 Hemoglobin dissociation Curve.

High altitude illness

1. AMS - (Acute mountain Sickness) → Rx:- Decent to lower altitude
   - with in 6-12 hours of ascent to high altitude.
   - C/F: Nausea, vomiting, confusion.
   - may become severe → Edema
     - O_2 therapy
     - CCB like nifedipine
     - Dexamethasone (for edema)

   HAPE: High altitude Pulmonary edema.
   HACE: High altitude cerebral edema.

2. Chronic moutain sickness (Monge's disease):
   - Excessive erythrocytosis
   - Pulmonary HTN
   - Corpulmonale (if untreated)
   - Rx: Venesection, Acetazalamide.

Sodium Deficit calculation

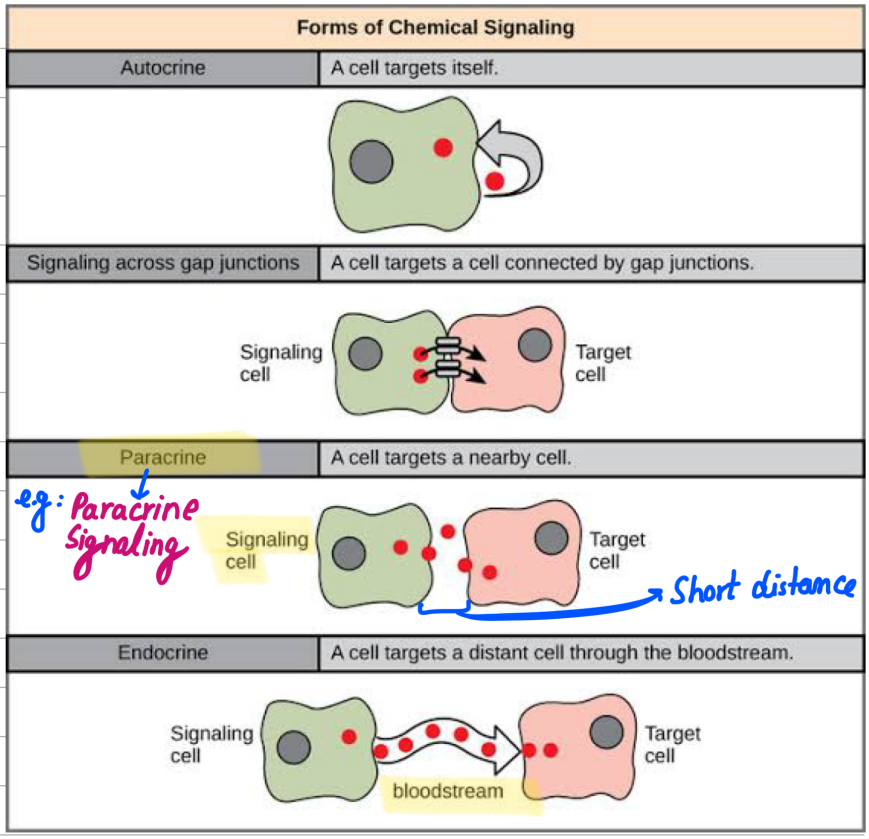
→ 0.6 × (wt in kg) × (desired sodium − measured sodium)
(0.5 for females)

Cell signaling pathways

Basics: There are two types of cells

- Signalling cells → Ligand or Signals are released
- Target cells → Recieves the signals

The are 4 types of cell signalling
1. Paracrine
2. Autocrine
3. Endocrine
4. Signalling by direct contact



eg: Paracrine Signaling → Short distance

Feedback Mechanisms

Homeostasis: Concept of Constancy.

The degree of effectiveness of the negative feedback is determine by → Gain = correction/error

- Claude Bernard said despite of changes in the external environment, the body will always try to to maintain stable internal environment (milieu interior)

Control System

Negative feedback
- Baroreflex: ↑BP → ↓BP
- Cortisol → inhibits ACTH

Positive feedback

Amplification reaction
- C- Coagulation cascade
- C- Calcium entery in SR (Relaxⁿ of m/s)
- L- LH Surge
- A- Action potential
- P- Parturition
- S- Shock

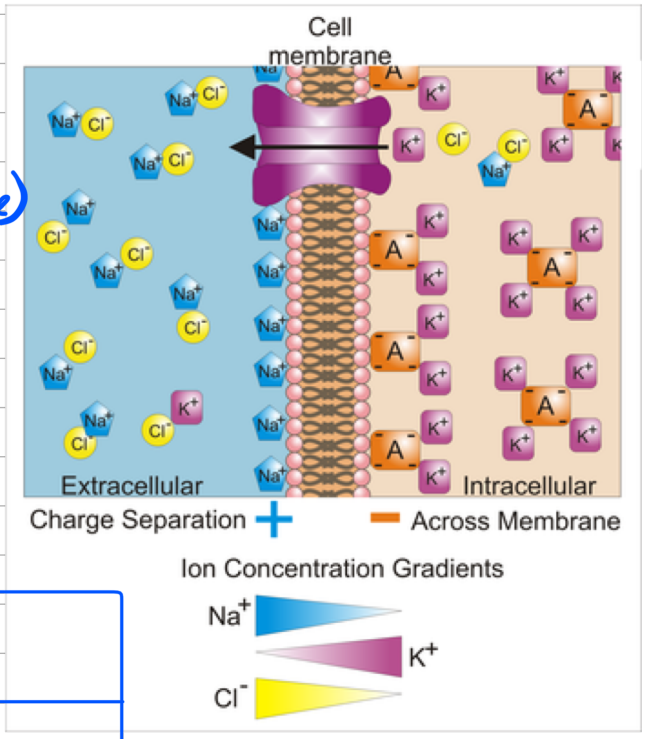
Feedforward control system
- Anticipatory tachycardia & tachypnea.
- Temperature regulation

Cellular fluids

- Membrane is permeable to Na^+, K^+, Cl^-
- membrane is impermeable to proteins (Negative charge)

Gibbs-Donnan effect:

- The precence of negativity inside the cell is due to accumulation of protein anions as a result of impermeability of the membrane to proteins



Total body water (60% of body weight), 42 liters (70kgm)

ICF	ECF	
2/3rd of TBW i.e. 40% Body weight (28 Liters)	1/3rd of TBW i.e. 20% Body weight (14 Liters)	
	Interstitial: 75% or 3/4 of ECF or 15% of body weight.	Plasma: 25% or 1/4th of ECF or 5% of body weight.

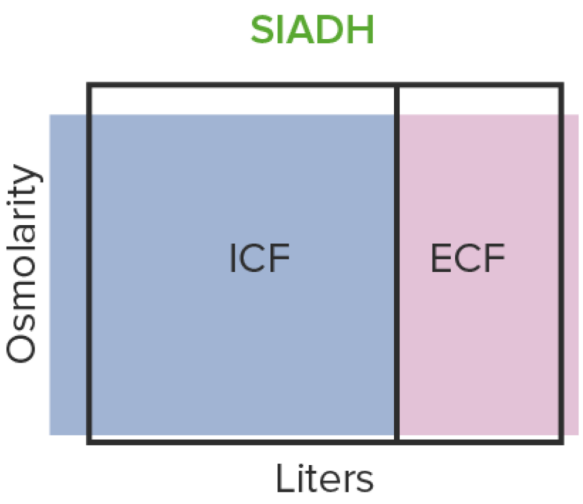
Volume	Indicators
• Total Body water	• Deuterium, Tritium
• Extracellular fluid	• Insulin, Sucrose, mannitol, Na^+ thiosulphate
• Plasma volume	• Radiolabelled-albumin, Evan's bluedye
• Blood volume	• Chromium labelled RBC, Blood volume = $\frac{\text{Plasma volume}}{1-\text{Hematocrit}}$
• Interstitial fluid	• IF = ECF - PV
• Intracellular fluid	• ICF = TBW - ECF

SIADH : Syndrome of inappropriate antidiuretic Hormone

↓

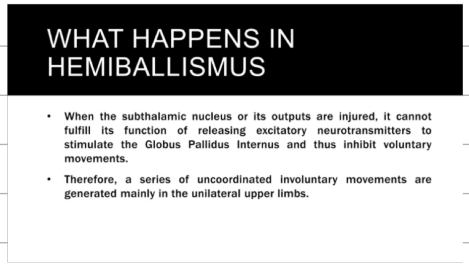
Gain of water / water excess

- ECF volume : increase
- ECF osmolarity : Decrease
- ICF volume : increase
- ICF osmolarity : Decrease

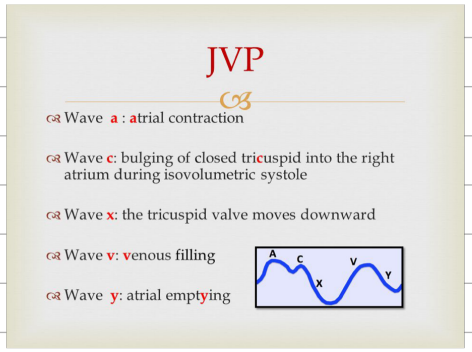


Hemiballismus : Characterised by the menifestation of abrup and irregular involuntary movements on one side of the one side of the body.

- Caused by lesion in the subthalamic nucleus (part of the basal ganglia) or its connections with Globus pallidus internus.



Waves of JVP



a - atrial contraction

c - isovolumetric systole (Bulding tricuspid valve)

x - atrial relaxation

v - venous filling of right atrium

y - Passive emptying of the RA into RV

Regulation of body temperature

Lesions causes Hyperthermia

Hot environment

Controlled by anterior Hypothalamic nucleus

to dissipate Heat

- Sweating
- ↑ thirst
- Vasodilation

Due to presence of osmoreceptors (found in anterior Hypothalamus)

Stimulated by dehydration, hemorrhage and prandial drinking (drinking water while eating)

Cold environment

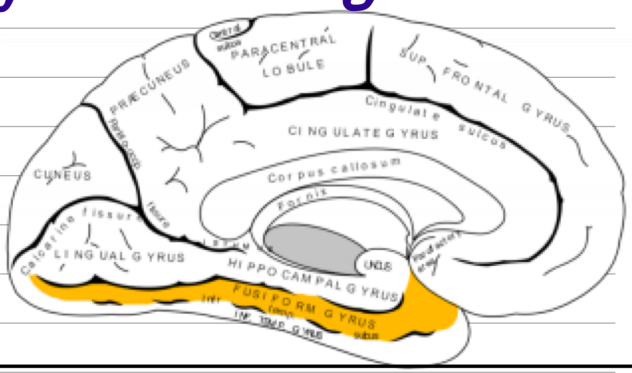
Controlled by posterior Hypothalamic nucleus

Lesion causes: Hypothermia

- Shivering
- Vasoconstriction
- ↑ urine frequency

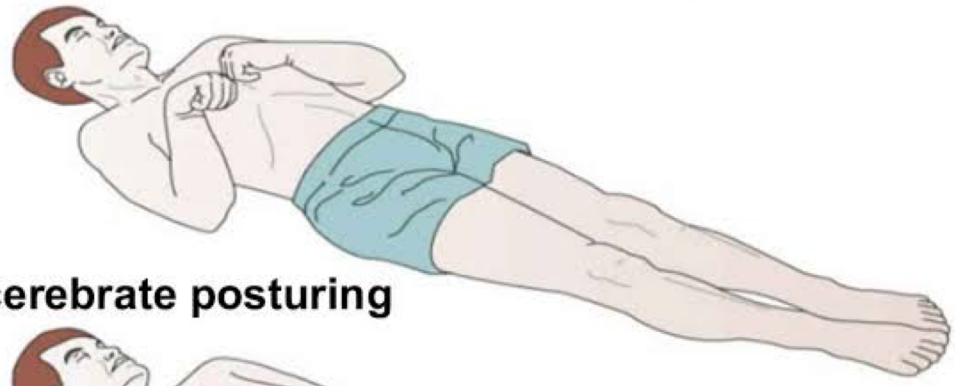
Prosopagonasia :

- Facial blindness
- Prosopagnasia is a neurological disorder characterized by the inability to recognize faces. It is a known face blindness or facial agnosia.
- Can Result from stroke, traumatic brain injury, or certain neurodegenerative disease.

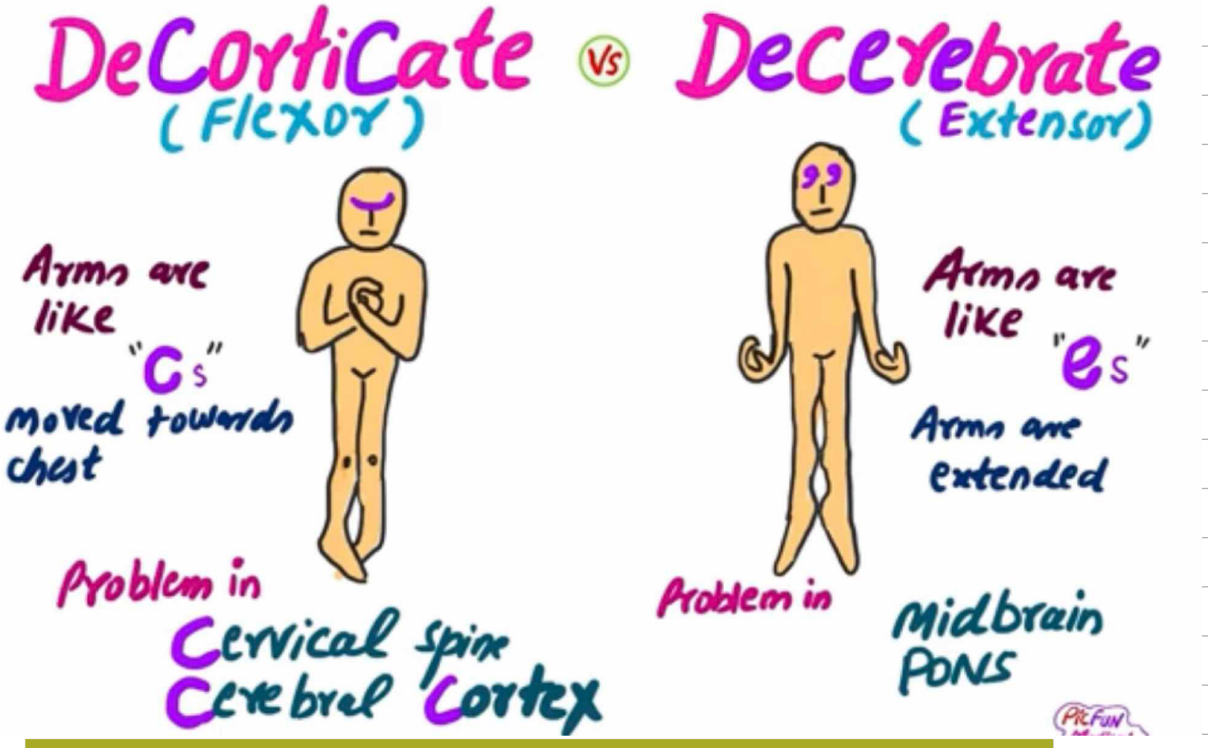


Manifestations of Brain Injury

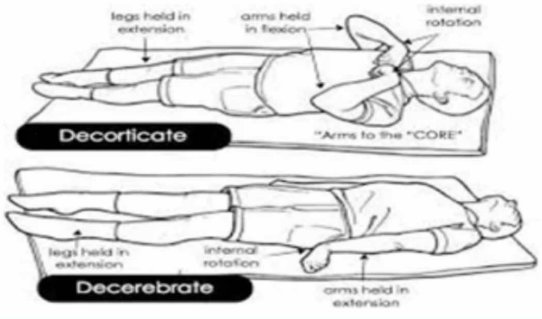
Decorticate posturing



Decerebrate posturing



DECEREBRATE RIGIDITY	DECORTICATE RIGIDITY
Upper and lower limb extended	Upper limb flexed and lower limb extended
Lesion below midbrain	Lesion above midbrain
Temperature regulation absent	Temperature regulation present
Not better outcome	Better outcome



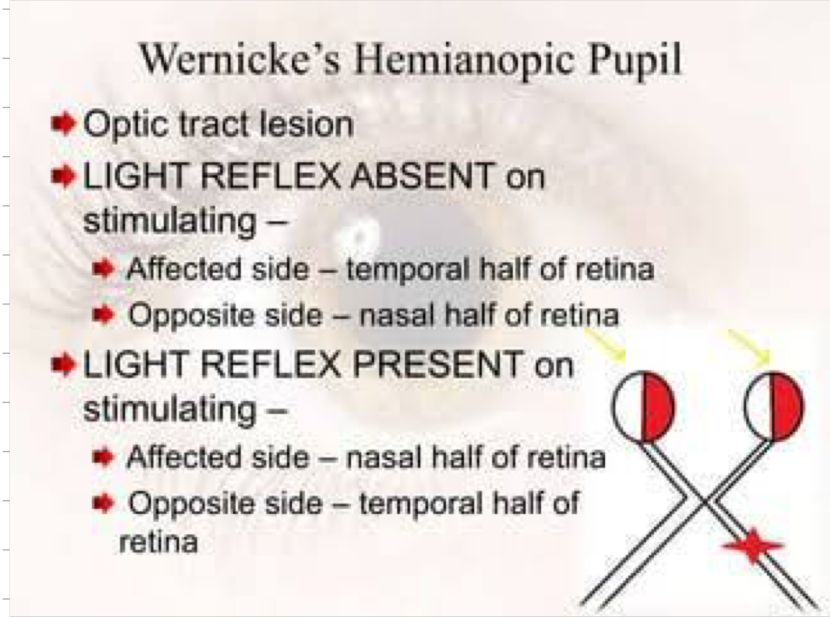
Decerebrate rigidity

γ motor neuron discharge rate increased.

Motor neuron pool excitability increased.

- Decerebration produces no phenomenon similar to spinal shock.
- Antigravity extensor muscles are involved in decerebrate injury.

Wenicke's hemianopic pupil lesion



Renal Autoregulation: GFR remains constant in BP range of 80-180 mmHg.

- **Mechanism 1 (Myogenic Mechanism)**: ↑ in blood flow → Stretch vascular smooth muscles (VSM) → Ca^{2+} channel open → Decreased blood flow

(Myogenic mechanism abolished in paralysis of VSM)

2. **Mechanism 2 (Tubulo-Glomerular feedback)**: ↑ in GFR → More NaCl filtered → Activates macula densa (GFR Sensor) → Adenosine release

(macula Densa is found in Tubules)

↓ Afferent arteriole constriction

↓ Decrease in GFR.

3. High protein intake : Increase in amino acid

↓ more amino acids and Na^+ reabsorbed in PCT

↓ macula densa recieves less Na^+

↓ Activates RAAS (Renin angiotensin aldosterone system)

↓ Increases GFR

Counter Current Mechanism :

↓ Responsible for development and maintenance of the medullary gradient and hyperosmolarity of interstitial fluid in the kidney.

2 components :

1. Counter Current multipleir → Loop of Henle

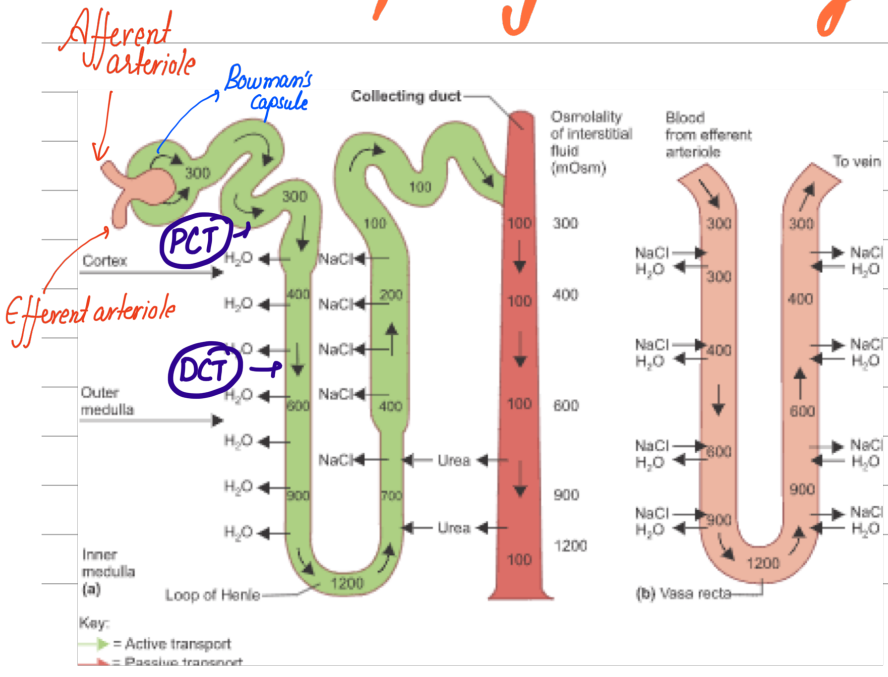
↓ Responsible for development of Hyperosmolar Medullary interstitial

2. Counter current exchanger : made by Vasa Recta

↓ Continuation of efferent arteriole.

↓ Responsible for maintenance of Medullary Gradient

↓ developed by Counter Current gradient



Countercurrent Mechanism

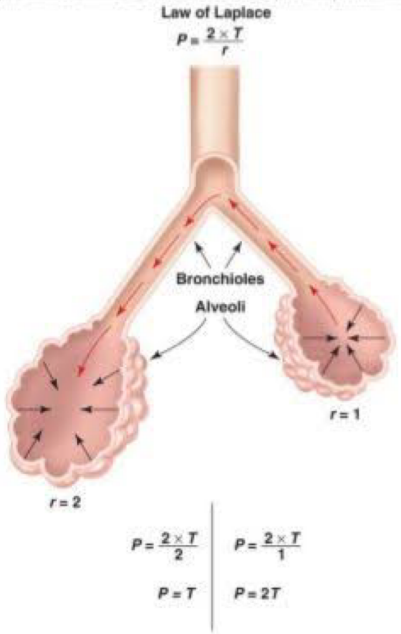
Interaction between the flow of filtrate through the loop of Henle (countercurrent multiplier) and the flow of blood through the vasa recta blood vessels (countercurrent exchanger)

Countercurrent multiplication expends energy to create a concentration gradient.

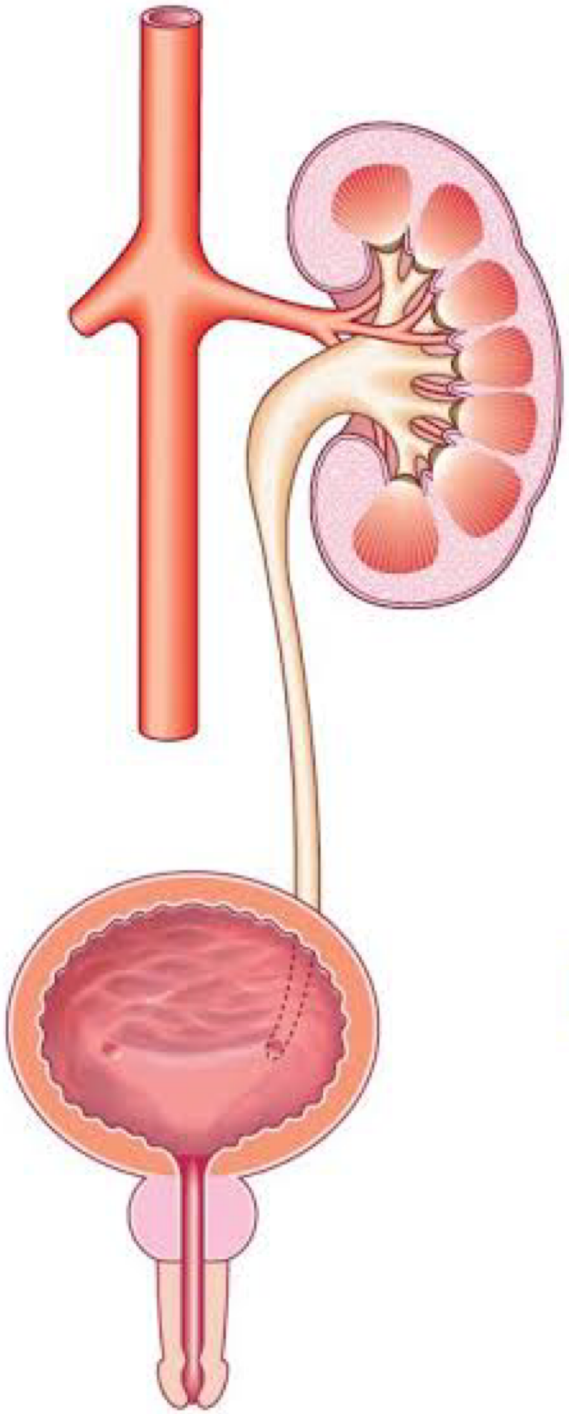
Countercurrent exchanger is similar, with different mechanism, where gradients are maintained but not established.

Law of Laplace

- Pressure is directly proportional to surface tension and inversely proportional to radius of alveolus.
  - Small alveoli would be at greater risk of collapse without surfactant.



Causes of Acute Renal Failure



PRE-RENAL

Impaired perfusion:
- Cardiac failure
- Sepsis
- Blood loss
- Dehydration
- Vascular occlusion

RENAL

Glomerulonephritis
Small-vessel vasculitis
Acute tubular necrosis
- Drugs
- Toxins
- Prolonged hypotension

Interstitial nephritis
- Drugs
- Toxins
- Inflammatory disease
- Infection

POST-RENAL

Urinary calculi
Retroperitoneal fibrosis
Benign prostatic enlargement
Prostate cancer
Cervical cancer
Urethral stricture/valves
Meatal stenosis/phimosis

Causes of acute kidney injury.

Source : Davidsons Essentials of Medicine, 2e

Fetal circulation

Concept 1:

Oxygenated blood from placenta to the fetus via the left umbilical vein.

Concept 2:

Most of the umbilical venous blood shunts via the **ductus venosus** to the inferior vena cava and gets mixed with deoxygenated blood from the lower part of the body and liver.

Concept 3:

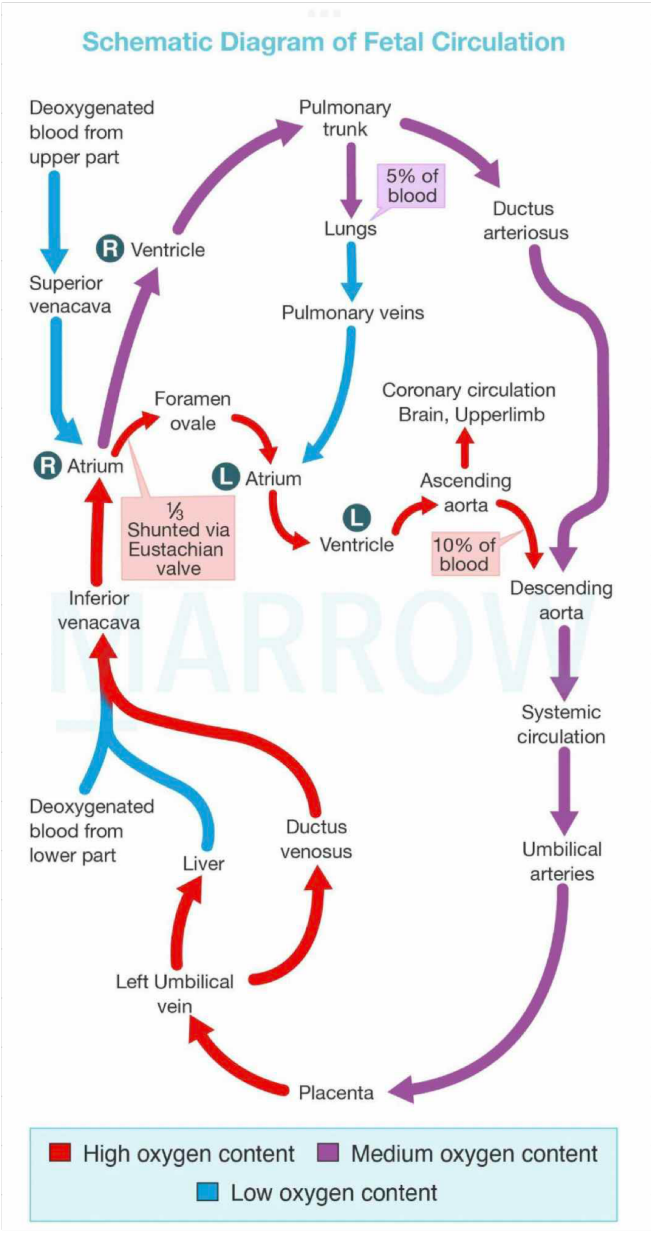
One-third of blood from the inferior vena cava is directly pumped to the left atrium across the **foramen ovale**; then to the left ventricle; and ejected from there into the ascending aorta, where it supplies the upper body and the brain.

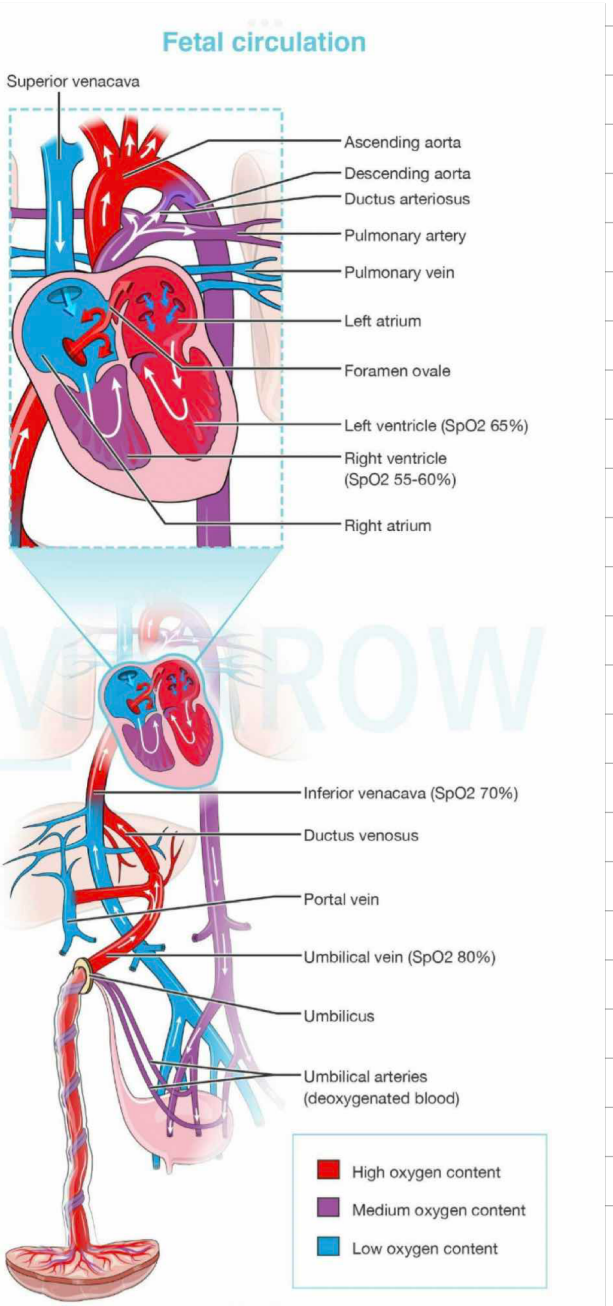
Concept 4:

A major portion of blood enters the right ventricle, mixes with blood from the superior vena cava, and ejects into the pulmonary trunk.

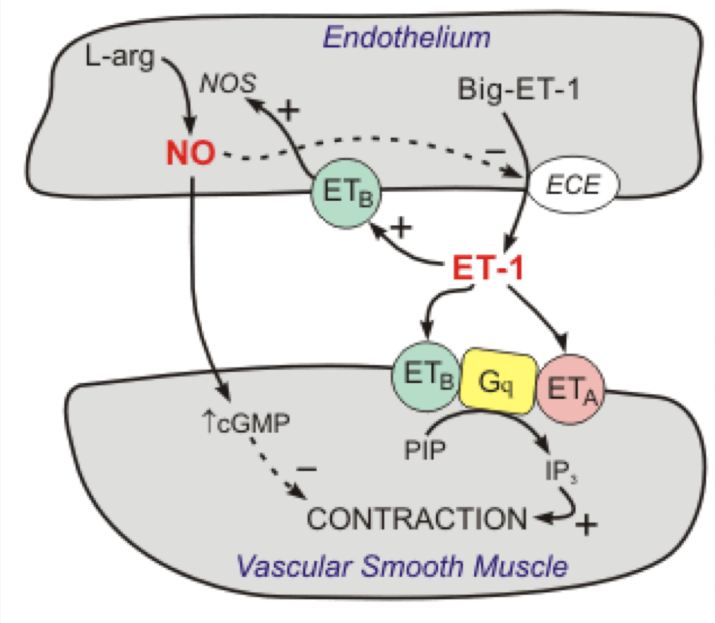
Concept 5:

Ductus arteriosus bypasses the lungs, and hence, a major portion pumps into the descending aorta to perfuse the lower part of the body and the umbilical arteries carry blood to the placenta for oxygenation.



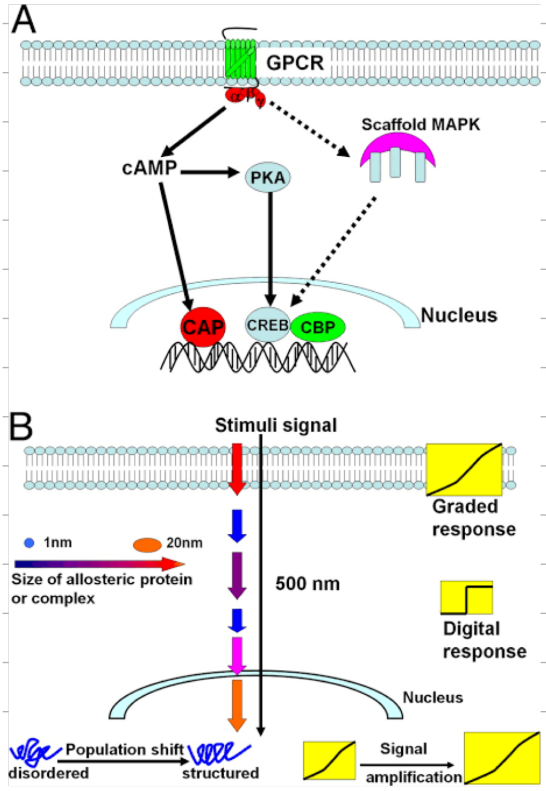


Endothelin-1 (ET-1) is a multifunctional hormone which regulates the physiology of the cardiovascular and renal systems. ET-1 modulates cardiac contractility, systemic and renal vascular resistance, salt and water renal reabsorption, and glomerular function. ET-1 is responsible for a variety of cellular events: contraction, proliferation, apoptosis, etc. These effects take place after the activation of the two endothelin receptors ET_A and ET_B, which are present – among others – on cardiomyocytes, fibroblasts, smooth muscle and endothelial cells, glomerular and tubular cells of the kidney.



Nuclear Signaling pathway

Nuclear receptors are activated by lipid-soluble signals (e.g., steroid hormones) that cross the plasma membrane. Once activated, most function as transcription factors to control gene expression for numerous biological processes.



ABO BLOOD GROUP SYSTEM

GROUP	A	B	AB	O
RED BLOOD CELL TYPE				
ANTIGENS PRESENT	 Antigen A	 Antigen B	 Antigen AB	 None
ANTIBODIES PRESENT	 Anti - B	 Anti - A	 None	 Anti - A + Anti - B

Ebb and flow Response to Trauma

	Ebb phase	Flow phase
Duration	24-48 h	- Catabolic Phase : 3-10 days - Anabolic Phase : weeks
Characteristics	**Shock** features *(decrease)* • Hypovolemia • Low BMR • Reduced CO • Hypothermia • *Lactic acidosis*	**SIRS** features *(increase)* • Tissue edema • High BMR • Increased CO • Hyperthermia • *Leukocytosis* • **Increased O2 consumption** • **Increased Gluconeogenesis**
Hormonal regulations	Catecholamine, cortisol, aldosterone. (stress hormones)	Catecholamines, cortisol, **insulin** and glucagon.
Main role	**Conserve** both circulating volume and energy stores for recovery and repair.	**Mobilization** of body energy stores for recovery and repair.

CPDA and ACD in Blood storage

Anticoagulant/Preservative solutions

- Acid Citrate Dextrose (ACD): 21 day storage
- Citrate-phosphate-dextrose (CPD) & Citrate-phosphate-dextrose-dextrose (CP2D): 21 day storage
- Citrate-phosphate-dextrose-adenine (CPDA-1): 35 day storage
  - Similar to CPD but + 17.3 mg adenine

The storage period when adding phosphate alone to ACD would be **21 days** and when adding adenine and phosphate together would be **35 days**.

When adding phosphate to ACD, Citrate Phosphate Dextrose (**CPD**) is formed – It has a storage period of **21** days. There is no change in the shelf life as phosphate only increases the 2,3 DPG levels of the stored blood.

When adding both phosphate and adenine to ACD, Citrate Phosphate Dextrose Adenine (**CPDA-1**) is formed. It has a storage period of **35** days. It increases the shelf life by improving ATP synthesis of RBCs.

Using additive solutions containing saline, adenine, and glucose, with or without mannitol (**SAGM**) decreases RBC hemolysis, thus increasing the storage period to **42** days.