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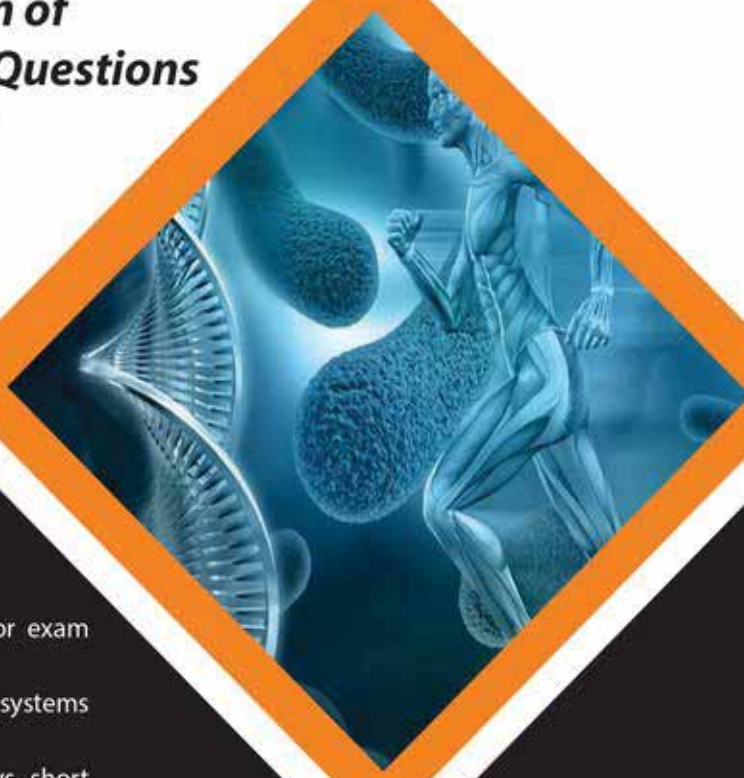
# Uniquest Series

# PHYSIOLOGY

***A Compilation of All India University Questions 2011–2021***



2nd Edition

**Highlights**

- A need of the hour Physiology book for exam preparation
- Important questions and answers from all systems are discussed
- Clear and to the point answers for essays, short notes and short answers type questions
- Answers made easy with flowcharts and diagrams.

**V Suganthi**

*Foreword*
**Milind V Bhutkar**

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# Answers to 2014 Question Paper

## ANSWER ALL QUESTIONS

**I. Essay Questions (15 Marks each)**

1. Describe the physiological roles of the different types of granulocytes circulating in blood.
2. Define glomerular filtration rate (GFR). What are its determinants? Discuss the phenomenon of autoregulation of GFR. Describe the best test for estimation of GFR. What is the routinely used clinical test to assess renal function?
3. Define the terms cardiac output and total peripheral resistance and discuss their determinants.
4. What are the neural mechanisms involved in spontaneous breathing? Discuss chemical regulation of respiration. Distinguish between the two types of respiratory failure.
5. What is the composition of gastric juice? Describe the mechanism of HCl secretion. Give a detailed account on the regulation of gastric secretion.
6. Define blood pressure. Discuss in brief the various factors which influence the pressure. Add a note on hypertension.
7. Define anemia. Classify them. List the important investigations to confirm the various types of anemia.
8. Define cardiac cycle. Describe the sequence of events during cardiac cycle in detail with suitable diagrams.

**II. Short Notes (2.5 Marks each)**

1. cAMP signaling pathway, with an example.
2. Colloid oncotic pressure and its importance.
3. Excitation-contraction coupling in skeletal muscle.
4. Types of polycythemia and complications due to this condition.
5. Findings of 'tests of hemostasis' in hemophilia.
6. Functions of macrophages.
7. Physiological role of corticosteroids.
8. Function of any one hormone of posterior pituitary.
9. Composition of bile and the physiological role (if any) of the components.
10. Pathophysiology of peptic ulcer.
11. Describe the 3 bipolar limb leads of ECG. What is the significance of (a) PR interval (b) ST segment in an ECG?
12. Discuss the changes in ventricular volume during different phases of the cardiac cycle with a diagram.
13. Discuss any two pulmonary function tests which can detect obstructive lung disease.
14. Trace the pathway for perception of fine touch.
15. Operant conditioning.
16. Clinical features of cerebellar lesions.
17. Define muscle tone and discuss the phenomenon responsible for it. What conditions lead to alterations of tone?
18. Endogenous opioid peptides.
19. Refractory errors of the eye.
20. Discuss the phenomena by which sound waves in air induce action potentials in the cochlear nerve.
21. Hypersecretion of growth hormone.
22. Tissue macrophage system.
23. Neural regulation of respiration.
24. Functions and tests of cerebellum.

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25. Transport across cell membrane.
26. Ovarian and endometrial changes of menstrual cycle.
27. Brown-Séquard syndrome.
28. Oxygen dissociation curve.

**III. Short Answers (1 Mark Each)**

1. Membrane transporters involved in clearance of calcium from cytoplasm.
2. Concentrations of sodium and potassium in intra- and extracellular fluids.
3. Phenomena involved in the act of swallowing.
4. Role of ATP in relaxation of muscle.
5. Draw a schematic diagram of the sarcomere and label its components.
6. Opsonins.
7. Cells which express major histocompatibility complex II.
8. Significance of glycosylated hemoglobin.
9. Name 4 enzymes in pancreatic secretion.
10. Hormonal imbalance causing (a) acromegaly (b) cretinism.
11. List the types of shock.
12. Define preload and state its effect on cardiac function.
13. Baroreceptor reflex.
14. What is myocardial infarction? State one ECG change in this condition.
15. Role of myelin sheath in conduction of nerve impulse.
16. Conditions where plantar response is 'extensor'.
17. Finding in Weber's test in conduction deafness of the left side.
18. Muscle actions responsible for (a) normal expiration (b) forced expiration.
19. Oxygen carrying capacity of blood.
20. Hypoxic vasoconstriction—where does it occur and what are its complications?
21. Permissive action of hormone.
22. Role of vitamin D in calcium homeostasis.
23. Contraception in males.
24. Corpus luteum.
25. Vitamin-K dependent clotting factors.
26. Atonic bladder.
27. Functions of skin.
28. Secondary active transport.
29. Motor unit.
30. Refractory period.
31. Heart sounds.
32. Waves of ECG in Lead II.
33. Different types of hypoxia.
34. Aphasia.
35. Stages of sleep.
36. Optic pathway.
37. Functions of ascending reticular activating system.
38. Components of vestibular apparatus.
39. Features of Parkinson's disease.
40. Functions of middle ear.
41. Functions of plasma proteins.
42. Nonexcretory functions of kidney.
43. Myasthenia gravis.
44. Stages of spermatogenesis.
45. Cystometrogram and its significance.
46. Hormones regulating calcium homeostasis.
47. Enterohepatic circulation.
48. Enzymes involved in digestion of fat.
49. Structure of platelets.
50. Functions of saliva.
51. Dead space.
52. Hering Breuer reflex.
53. Korotkoff sounds.
54. Draw a diagram of the pathway of crude touch and label it.
55. Functions of CSF.
56. Fluent aphasia.
57. Receptor potential.
58. Motor homunculus.
59. Attenuation reflex.
60. Taste pathway.

## I. ESSAY QUESTIONS

**1. Describe the physiological roles of the different types of granulocytes circulating in blood.**

**Granulocytes in blood are:**

- ❒ Neutrophils
- ❒ Eosinophils
- ❒ Basophils (*refer* **Fig. 1**)

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**Fig. 1:** Granulocytes.

### Physiological Roles of Neutrophils

- Neutrophils are the first line of defense
- It does the defense mechanism by phagocytosis.

### Phagocytosis

- Neutrophils are the first ones to reach the site of infection followed by macrophages.
- They come out of the blood vessels by a process called **emigration**.
- Adhesion molecules like *selectins* and *integrins* help in emigration.
- Phagocytes are attracted to sites of infection or inflammation by **chemotaxis**.
- Substances inducing chemotaxis are called **chemotaxins.**
- On reaching the pathogens or tissue debris, phagocytes adhere to them—**adherence**.
- Then they put out processes—Pseudopods and ingest the particles—**ingestion**.
- The ingested particle is enclosed in a vesicle—**phagosome** (*refer* **Fig. 2**).
- Phagosome attaches to lysosomes in the neutrophil to form— **phagolysosome**.
- The lysosomes put out hydrolytic enzymes like ***lysozymes***.
- The phagocytes have oxidative enzymes in them which form lethal oxidative metabolites like—**$O_2^-$, $H_2O_2$ and hypochlorites**.
- **Antimicrobials: Defensins** in phagocytes kill the microorganisms.
- By all these killing mechanisms the microbes are killed and degraded or form **residual bodies**.

### Other Functions

- Neutrophils release many chemical mediators like leukotrienes, thromboxane A2 which mediate inflammatory reaction.
- It mediates febrile response by releasing endogenous pyrogens.

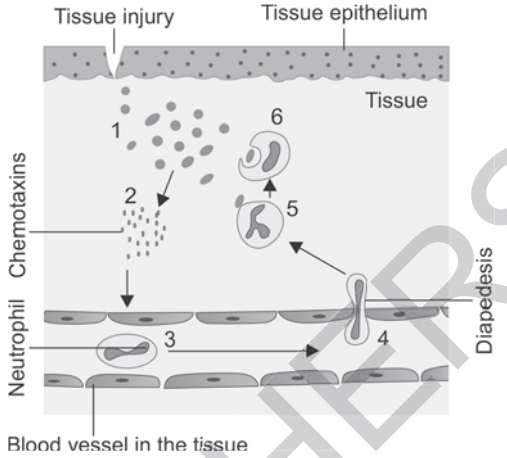


**Fig. 2:** Phagocytosis—Neutrophils marginate towards the wall of capillary followed by diapedesis, chemotaxis and engulfing the microbes: (1) Entry of microorganisms; (2) Chemotaxis; (3) Margination; (4) Diapedesis; (5) Fusion of microbe with lysosome; (6) Formation of phagolysosome.

### Eosinophils

#### *Structure of Eosinophils*

- Size is same as that of neutrophils (10–14 μm)
- Bilobed nucleus and lobes are connected by thick chromatin strand—gives a spectacle-shaped appearance.
- Cytoplasm has coarse, orange pink granules.
- **Granules contain:** Eosinophil peroxidase, eosinophilic cationic protein, major basic protein, histaminase, catalase, etc.
- Mostly present in tissues.

#### *Physiological Roles*

- Leave the capillaries and enter tissue spaces where allergic reactions occur.
- Releases an enzyme, histaminase that combats the effects of histamine released by basophils.
- They inhibit mast cell degranulation
- They are weakly phagocytic and phagocytose Ag-Ab complexes
- Eosinophils also release substances (Major basic protein) which kill the parasitic worms.
- So eosinophil counts increase in allergy and worm infestation.

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**Basophils**

- They are similar to mast cells.
- They are mildly phagocytic
- They enter tissues at sites of infection or tissue damage and release histamine, heparin and serotonin.
- These chemicals are responsible for the inflammatory reactions.
- They also release eosinophilic chemotactic factor which attracts eosinophils to combat the inflammatory reactions by removing Ag-Ab complexes.
- Mast cells and basophils release the inflammatory mediators and are responsible for allergic reactions.
- Basophils release heparin which is an anti- coagulant and thereby removes minute clots and prevents clotting of blood.

**2. Define glomerular filtration rate (GFR). What are its determinants? Discuss the phenomenon of auto-regulation of GFR. Describe the best test for estimation of GFR. What is the routinely used clinical test to assess renal function?**

- **Glomerular filtration rate is defined as the volume of plasma filtered by all the nephrons of both the kidneys per unit of time.**
- **Normal value is 125 mL/min or 180 L/day.**

**The determinants of GFR are:**

- **Filtration co-efficient (Kf):** It is the product of permeability of the glomerular membrane and its surface area. As the Kf increases GFR increases and as Kf decreases GFR decreases.
- **Glomerular capillary hydrostatic pressure ($P_{GC}$):** It is dependent on arterial blood pressure, renal blood flow and afferent and efferent arteriolar resistance. $P_{GC}$ is directly proportional to GFR.
- **Capillary oncotic pressure ($\pi_{GC}$):** GFR is inversely proportional to $\pi_{GC}$.
- **Hydrostatic pressure in Bowman's space:** It opposes filtration and GFR is inversely related to GFR.
- **Sympathetic stimulation:** Stimulation leads to vasoconstriction and decreased RBF and GFR.

**Autoregulation of GFR**

- The autoregulatory mechanism of GFR is similar to autoregulation of renal blood flow.
- Autoregulation of GFR maintains a constant rate of filtration in spite of change in arterial pressure within the range of 80–180 mm Hg (MAP).
- There are two autoregulatory mechanisms of GFR—Myogenic mechanism and tubuloglomerular feedback.

*Myogenic Mechanism*

- As the renal blood pressure increases, the afferent arteriole is stretched and the stretch induces reflex contraction smooth muscles lining the wall of the afferent arteriole.
- This result in vasoconstriction of afferent arteriole and thereby decreases renal blood flow and thereby decreases GFR.

*Tubuloglomerular (TG) Feedback*

- TG feedback is an autoregulatory mechanism in the kidneys to regulate the renal blood flow (RBF) and thereby the GFR.
- This feedback mechanism is dependant on the NaCl content in the tubular fluid.
- The NaCl content in the tubular fluid is sensed by the macula densa cells and signals are sent to the afferent arterioles to regulate the RBF and GFR.
- Increased renal arterial pressure increases glomerular capillary pressure and thereby increases filtration.
- The increased GFR leads to increased NaCl content in the tubular fluid.
- This is sensed by the macula densa cells and they send signals to cause vasoconstriction of the afferent arterioles and thereby decrease RBF and GFR and NaCl content is brought back to normal.
- If NaCl content is less, it is sensed by the macula densa cells and signals are sent to the afferent arterioles resulting in

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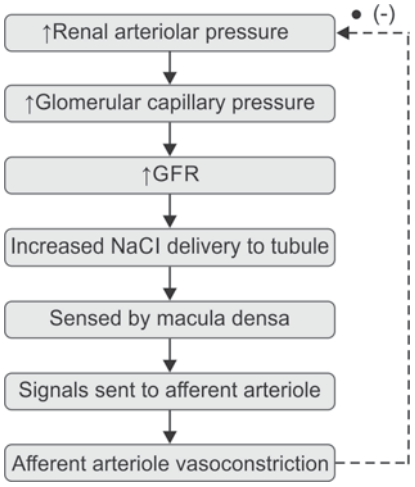


**Fig. 3:** Tubuloglomerular feedback.

vasodilation followed by increased RBF and GFR (*refer* **Fig. 3**).

- The mediating chemicals may be Thromboxane A2 or Adenosine for vasoconstriction and NO for vasodilation and they are released by the macula densa cells.

***The Best Test to Estimate GFR***

- It is done by assessing renal clearance of Inulin.
- Renal clearance is defined as the volume of plasma that is cleared of substance in a minute by excretion of the substance in urine.

***It is Calculated by using the Formula***

**C = UV/P**

- **C** = Clearance of the substance
- **U** = Urinary concentration of the substance
- **V** = Rate of urine flow
- **P** = Plasma concentration of substance in plasma
  - Inulin is used to measure GFR as it is freely filtered and neither reabsorbed or secreted by the tubules.
  - It is biologically inert and nontoxic and is neither metabolized nor stored.

***Methodology***

- A loading dose of Inulin is administered intravenously followed by a continuous infusion to keep the plasma level a constant.
- After the inulin has equilibrated in the body fluids, an accurately timed urine sample of urinary specimen is collected and half way through plasma sample is obtained.
- Then with the plasma and urinary concentrations the clearance is calculated by using the above formula.

$$U_{IN} = 35 \text{ mg/mL}$$
$$V = 0.9 \text{ mL/min}$$
$$P_{IN} = 0.25 \text{ mg/mL}$$
$$C_{IN} = U_{IN} V/P_{IN}$$
$$= 35 \times 0.9/0.25$$
$$= 126 \text{ mL/min}$$

***Routinely used Clinical Test used to Assess Renal Function***

- A fall in GFR is the first and only clinical sign of kidney disease.
- So measurement of GFR is used to assess kidney diseases.
- Since measurement of GFR by inulin clearance is cumbersome in clinical setting, plasma creatinine is measured and its level is inversely proportional to GFR.
- But the fall in GFR should be substantial for the rise in plasma creatinine

---

**3. Define the terms cardiac output and total peripheral resistance and discuss their determinants.**

---

**Cardiac output is defined** as the amount of blood ejected by each ventricle per minute. Cardiac output = Stroke volume × Heart rate
Normal value = 5–6 L/min.

**Determinants of Cardiac Output**

- CO = Stroke volume (SV) × Heart rate (HR)
- So factors affecting either of them will alter CO.

***Factors Affecting SV***

- Preload
- After load
- Contractility

***Factors Affecting HR***

HR is affected by nerves supplying heart (Sympathetic and parasympathetic nerves) and cardiac centers

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### Preload

- The initial muscle length is the **preload** (the extent to which the muscle is stretched before contraction).
- Preload is decided by the **end-diastolic volume (EDV)**.
- EDV is decided by the **venous return (VR)**.
- **Preload affecting stroke volume is based on the Frank-Starling's law:** It states that within physiological limits, the force of contraction is directly proportional to the initial length of the muscle.

#### *Causes for Application of Frank-Starling's Law*

- Increase in EDV →↑stretch of muscle fiber → Interaction of thick and thin filament increases →↑ force of contraction.
- Stretch of muscle fibers → Opens stretch sensitive $Ca^{2+}$ channels on sarcolemma →↑ $Ca^{2+}$ influx →↑ force of contraction.
- ↑$Ca^{2+}$ influx →↑ $Ca^{2+}$ release from SR (CICR) →↑ force of contraction.
- ↑stretch of muscle fiber →↑ Affinity of troponin for $Ca^{2+}$

**EDV is dependent on:**

- **Venous return, which is dependent on:**
  - *Skeletal muscle pump:* Contraction of limb muscles press on the veins and increases forward movement of blood in the veins
  - *Thoracic pump:* Increase in respiration depresses the diaphragm and decreases intrathoracic pressure and thereby, it acts like a suction force to increase VR
  - *Abdominal pump:* During respiration compression of abdominal muscles press on the veins and favors venous emptying
  - *Cardiac pump:* Vis A Tergo (Force from behind), Vis A Fronte (Force from front)
  - *Total blood volume:* As the blood volume increases VR increases and vice versa
  - *Capacity of venous system:* Sympathetic stimulation to the veins causes venoconstriction and thereby increases venous emptying
  - *Body position:* On standing, there is venous pooling due to gravity and it may decrease the VR.
- **Atrial pump activity:** Contributes 20% ventricular filling and stimulated by sympathetic stimulation.
- **Ventricular compliance:** Affected by damage to myocardium as in MI, pericardial effusion and cardiac tamponade.

This type of regulation of stroke volume in relation to change in initial length of muscle fiber is said to be **heterometric regulation**.

### Contractility

Contractility is increase in force of contraction and thereby increase in stroke volume without increase in initial muscle length.

- Ventricles are able to do more work per stroke at a given EDV.
- Factors which affect contractility—inotropic agents.
- There are positive and negative inotropic agents.

**Contractility is increased by:**

- Autonomic activity (Sympathetics are positively inotropic)
- **Muscle mass:** Increase in myocardial mass increases contractility as after regular exercise
- Concentration of hormones and chemicals
- **Heart rate:** Force frequency relationship

**Factors increasing contractility, positive inotropic agents:**

- Sympathetic stimulation
- Circulating catecholamines
- Force frequency relationship
- Digitalis
- Glucagon
- Insulin
- Thyroxine
- Chemicals like xanthine, theophylline

**Factors decreasing contractility, negative inotropic agents:**

- Loss of myocardium as in myocardial infarction
- Hypoxia
- Hypercapnia
- Acidosis
- Acetylcholine

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Regulation of stroke volume by affecting contractility of myocardium is **homometric regulation**. Here the force of contraction is affected without much change in muscle length.

**Afterload**

- It is the force against which the heart muscle shortens
- Cardiac output is inversely proportional to afterload
- **Cardiac output α 1/afterload (anrep effect)**
- It is decided by peripheral resistance
- **Peripheral resistance is decided by:**
  - Vessel diameter
  - Viscosity of blood
- **This is also included in homometric regulation.**

**Determinants of Peripheral Resistance**

- **Total peripheral resistance is defined** as the resistance offered to the blood flow in the blood vessels.
- There is a resistance to blood flow in the peripheral circulatory system and it is said to be the peripheral resistance.
- Arterioles are the site of major resistance to blood flow in the vascular system. Therefore, they are called as the 'resistance vessels.'
- The walls of the arterioles are having more of smooth muscles than elastic tissue and these muscles are supplied by noradrenergic fibers and they are in partial contraction at rest—Vasomotor tone.
- The constriction and relaxation of the arterioles in any organ is responsible for the tissue perfusion.
- The resistance is decided by the vasoconstriction or dilatation. There are other factors also which decide the total peripheral resistance (TPR).
- Important factors responsible for TPR are—radius of the vessel and viscosity of blood.

*Radius of the Vessel*

- Radius of the vessel is decided by the sympathetic nerves.
- Decrease in radius increases TPR and increase will decrease TPR.
- Even a decrease in resistance by half will increase the TPR 16 times.
- When the radius is doubled the resistance is reduced by 6% of its previous value

*Viscosity of Blood*

- Viscosity of blood also affects TPR.
- But the most commonly occurring change in TPR is due to change in the radius of blood vessel.
- Viscosity of blood is decided by the cellular components of blood, especially RBCs and composition of plasma and resistance of cells to deformation. Temperature also affects viscosity.
  - *Hematocrit:* Viscosity of blood is mostly dependent on the hematocrit. In large vessels increase in hematocrit largely increases viscosity. In smaller vessels, the effect of viscosity on TPR is less due to difference in nature of blood flow. So the net effect of viscosity of blood on TPR is less in vivo than in vitro, unless there is severe polycythemia. In anemia, TPR is decreased as viscosity is decreased.
  - *Composition of plasma:* Viscosity is increased in diseases where there is marked increase in plasma proteins as in multiple myeloma.
  - *Effect of deformed cells:* There is rise viscosity in hereditary spherocytosis
  - *Effect of temperature:* Increase in body temperature decreases viscosity and decrease in body temperature increases viscosity.

---

**4. What are the neural mechanisms involved in spontaneous breathing? Discuss chemical regulation of respiration. Distinguish between the two types of respiratory failure.**

---

**Neural Regulation of Respiration**

- Breathing is an automatic process occurring throughout life without conscious effort.
- Respiration is a process which is highly regulated.

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- Spontaneous respiration is due to rhythmic discharge of neurons from respiratory centers supplying the inspiratory muscles.
- This is under cortical (voluntary) and medullary and pontine control (Automatic).
- These centers in turn are regulated by alterations in the $PCO_2$, $PO_2$ and pH of arterial blood and other nonchemical influences.

**Cortical Control or Voluntary Control of Respiration**

**Impulses from cerebral cortex**
↓
**Corticospinal tract**
↓
**Motor neurons supplying the inspiratory muscles**

- Impulses also reach the innervation of expiratory muscles.
- There is a reciprocal inhibition between the motor neurons supplying the I and E neurons.
- There is an exception for this at the start of expiration were the "I" neurons are active.
- The inspiratory muscles are active for some time during expiration to brake the elastic recoil of lungs and make expiration smooth.

**Automatic Control of Respiration**

Impulses from brainstem respiratory centers in pons and medulla → supplies neurons in intermediolateral horn cells of cervical and thoracic segments → supplies inspiratory muscles.

*Medullary Respiratory Centers*

- **Dorsal respiratory group of neurons (DRG):** Has 'I' neurons
- **Ventral respiratory group of neurons (VRG):** Has 'I' and E' neurons (*refer* **Fig. 4**)
- **Central pattern generator (CPG):** Pre-Bötzinger complex (*refer* **Fig. 5**)

*Dorsal Respiratory Group of Neurons*

- Located near nucleus tractus solitarius (NTS)
- Contains only inspiratory neurons
- They project to cell bodies of phrenic nerves in spinal cord.

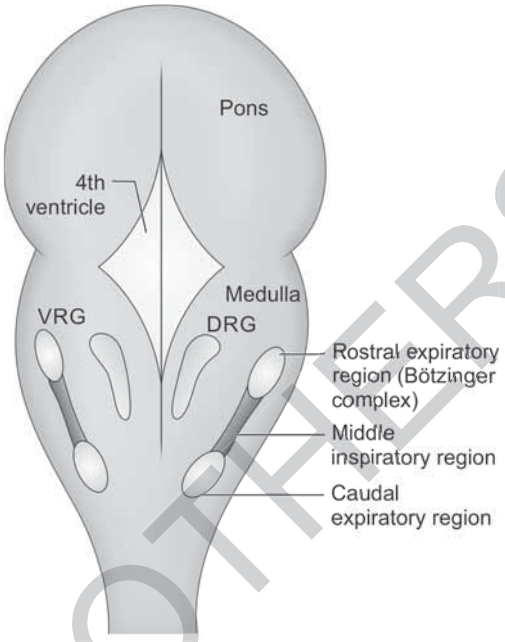


**Fig. 4:** Respiratory centers in medulla.
[DRG: dorsal respiratory group of neurons (has only inspiratory neurons); VRG: ventral respiratory group of neurons (has both inspiratory and expiratory neurons)]

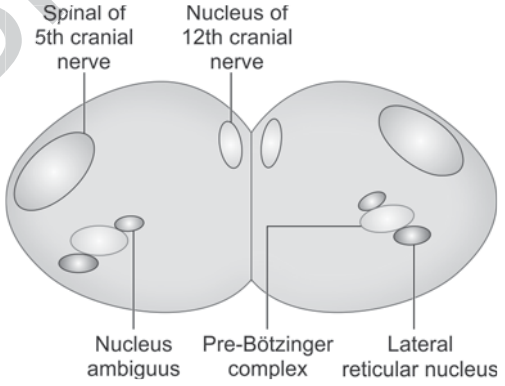


**Fig. 5:** Location of pacemaker (Pre-Bötzinger complex) for rhythm generation in medulla.

- Its activity is weaker to start with and gradually increases in a ramp fashion for 2 seconds and abruptly stops for 3 seconds.
- The ramp signal helps in steady increase in lung volume during inspiration
- Receives input from peripheral chemoreceptors through 9th and 10th cranial nerves.

*Ventral Respiratory Group of Neurons*

- Located in ventrolateral medulla in region of nucleus ambiguus (NA)

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- Contains both inspiratory and expiratory group of neurons
- 3 regions—Rostral, middle and caudal inspiratory regions
- Middle region has inspiratory neurons
- Rostral and caudal has expiratory neurons—supply expiratory muscles.
- They are active only during forceful respiration.
- I and E neurons reciprocally inhibit each other

***Pacemaker Cells for Respiration***

- Located close to DRG and VRG neurons
- Present in the pre-Bötzinger complex between nucleus ambiguus and lateral reticular nucleus.
- They are pacemaker cells and are responsible for generating respiratory rhythm → Rhythmically activate phrenic nerves.
- It receives input from higher centers

**Pontine Centers**

There are two centers:

1. **Pneumotaxic center:**
   - Located in nucleus parabrachialis and Kölliker-Fuse nucleus in the upper part of pons (*refer* **Fig. 6**)

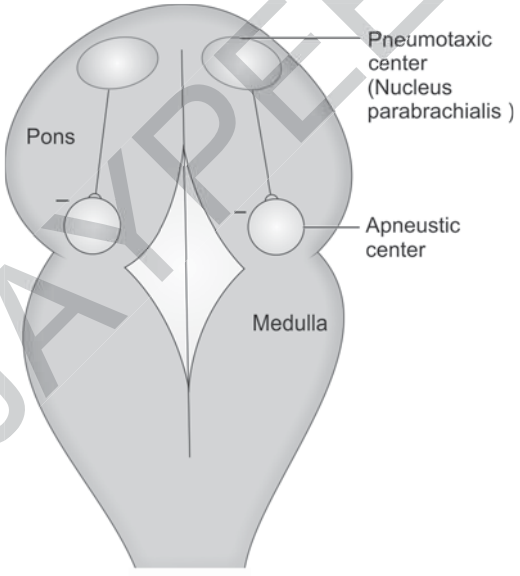


**Fig. 6:** Respiratory centers in pons, pneumotaxic and apneustic centers.

   - Active during both inspiration and expiration.
   - On stimulation, it shortens the duration of inspiration. When its activity is less, the duration of inspiration is longer.
   - Its major function is to limit inspiration, by inhibiting apneustic center
   - It coordinates switching between inspiration and expiration.
2. **Apneustic center:**
   - Present in lower part of pons (*refer* **Fig. 6**).
   - On stimulation → Leads to prolonged inspiratory spasms—APNEUSIS
   - It sends inputs to DRG to cause a prolonged inspiration
   - It is constantly stimulating DRG.
   - But its actions are kept in check by inputs from pneumotaxic center and vagal afferents from airways.

**Mechanism of Breathing**

- The neurons in DRG discharge steadily and spontaneously in a ramp like fashion for 2–3 seconds.
- Muscles of inspiration contract steadily resulting in expansion of the lung and chest wall resulting in inspiration.
- As the inspiration happens, the stretch receptors in lungs and airways are stimulated and impulses from here travel through vagus nerve to stop the firing of DRG neurons.
- Impulses from pneumotaxic center through inhibition of apneustic center stops the firing of DRG (*refer* **Fig. 7**).
- Inspiratory muscles relax and the lung and chest wall recoils and induces expiration.
- At the end of expiration, the next cycle starts.

**Factors Influencing Respiratory Centers**

- **Afferents from higher centers:** Cerebral cortex, limbic system, hypothalamus
- **Afferents from peripheral receptors:** Baroreceptors, chemoreceptors, J receptors, pain receptors, proprioceptors, pulmonary stretch receptors and thermoreceptors.

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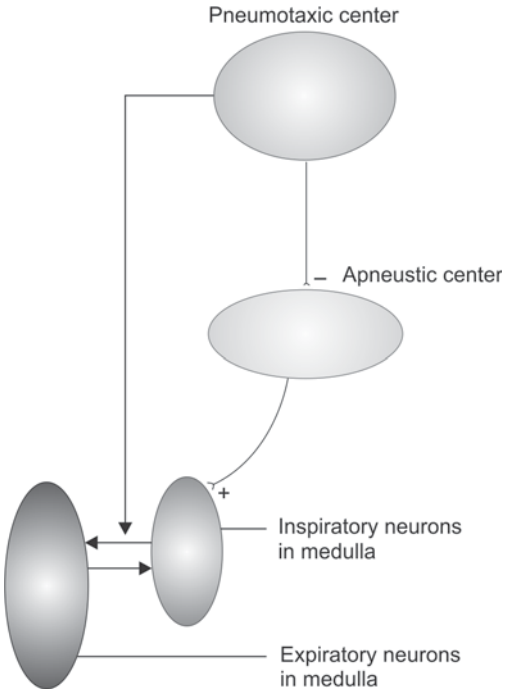


**Fig. 7:** Interaction of medullary and pontine centers in regulation of respiration.

- **Reflexes:** Hering-Breuer reflex, sneezing reflex, swallowing reflex, cough reflex, speech

### Chemical Regulation of Respiration

- Chemical regulation of respiration is also through the modulation of activities of neural centers through chemoreceptors.
- There are 2 sets of chemoreceptors responding to changes in arterial $PO_2$, $PCO_2$ and pH
- They are peripheral and central chemorecptors.

#### *Peripheral Chemoreceptors*

- Carotid and aortic bodies (*refer* **Fig. 8**).
- They have high blood flow (2000 mL/100 g/min) and high metabolic rate.
- Their metabolic needs are met by dissolved $O_2$.
- So they are highly sensitive to $\downarrow PO_2$, $\uparrow PCO_2$ and $\downarrow$pH.
- These receptors are not stimulated in anemia or carbon monoxide poisoning.
- They are stimulated in $\downarrow PO_2$, vascular stasis, cyanide poisoning

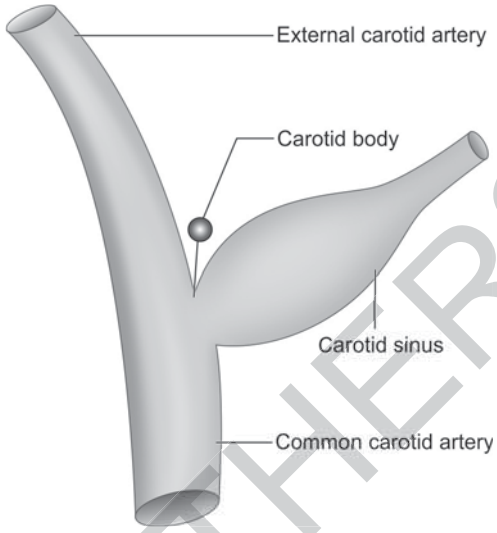


**Fig. 8:** Location peripheral chemoreceptors at bifurcation of common carotid artery; the carotid body. Carotid sinus is the dilatation in internal carotid artery; the baroreceptor.

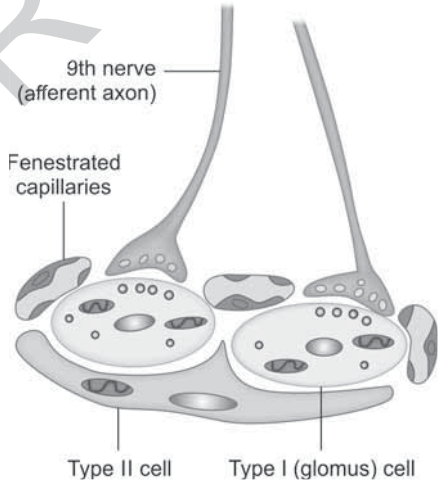


**Fig. 9:** Cells in carotid body—Type I (glomus cells) and Type II (supporting cells). Glomus cells are supplied by 9th cranial nerve and are close to fenestrated capillaries.

- They have 2 types of cells—glomus and sustentacular cells.

#### *Sensitivity of Peripheral Chemoreceptors (PCR)*

- Glomus cells are the sensors (*refer* **Fig. 9**)
- Hypoxia is the major stimulus. Neurotransmitter is Dopamine
- PCRs also responds to $\uparrow PCO_2$ and $\downarrow$pH

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5. **Last rapid filling:**
   - Duration 0.1 sec.
   - Atria contracts and pumps the last 25% of blood into ventricles.
   - At rest, this volume is not essential.
   - But in tachycardia (as in exercise) when the duration of diastole decreases this volume assumes importance.

## II. SHORT NOTES

### 1. cAMP signaling pathway, with an example

- ❒ cAMP is an important second messenger for peptide hormones.
- ❒ Many chemicals, such as norepinephrine, ACTH, ADH, angiotensin II, calcitonin, CRH, FSH, LH, etc., act via cAMP.
- ❒ It is said to be the cAMP-Adenylyl cyclase pathway.

**The following is the signaling pathway:**

- ❒ Peptide hormone binds to the membrane bound receptor
- ❒ The receptor is bound to G-protein.
- ❒ On binding of hormone to receptor, GDP is released from G-protein and replaced by GTP and G-protein is activated.
- ❒ Activated G-protein in turn stimulates or inhibits the membrane bound enzyme adenylyl cyclase.
- ❒ Adenylyl cyclase catalyzes the conversion of cytoplasmic ATP to cAMP. When the G-protein is stimulatory (Gs) the cAMP levels increase and when the G-protein is inhibitory (Gi) The cAMP is decreased (*refer* **Fig. 17**).
- ❒ cAMP in turn activates a cascade of enzyme system like protein kinase A.
- ❒ Protein kinase A catalyzes the phosphorylation of proteins, changing their confirmation and functions.
- ❒ cAMP is inactivated to 5'AMP by the enzyme phosphodiesterase and activity is terminated.

**For example**, the activation of phosphoryl kinase enzyme in the liver by epinephrine via cAMP and protein kinase A is given below:

Epinephrine
↓
β2 receptor
↓
Gs
↓
Adenylyl cyclase

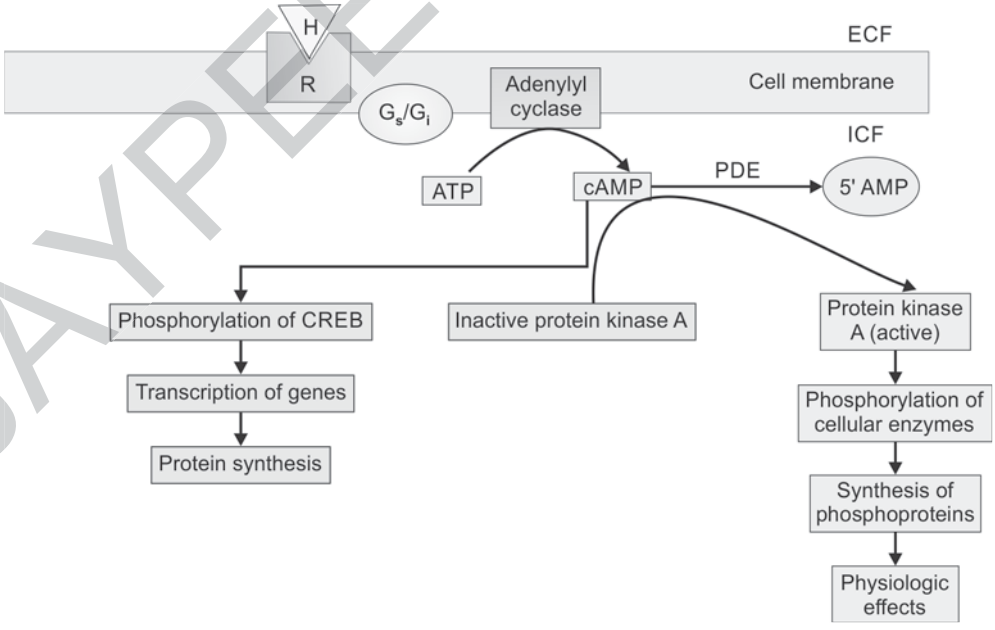


**Fig. 17:** Activation Gs-adenylyl cyclase-cAMP pathway signaling pathway.

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↓
ATP → cAMP
↓
Protein kinase A activated
↓
Inactive phosphorylase
b kinase +ATP → Activated phosphorylase b kinase
↓
Phosphorylase b +ATP → Phosphorylase A
↓
Glycogen → Glucose-1-phosphate

## 2. Colloid oncotic pressure and its importance.

- Colloidal oncotic pressure is the osmotic pressure generated by larger molecules, especially proteins in plasma.
- The oncotic pressure exerted by plasma proteins is 26–28 mm Hg.
- It is the important force involved in fluid movement across the capillaries.
- Among the plasma proteins, albumin is the major protein to exert the oncotic pressure.
- The pores in between the endothelial cells are smaller in size and therefore do not allow the movement of proteins and other colloids across them.
- The colloids have a high molecular weight and are present in large amounts.
- Therefore, the capillary membrane is like an impermeable membrane to colloids and they exert an osmotic pressure of an average of 25 mm Hg.
- The rate of filtration of fluid across the capillary wall at any time is dependent upon the balance of Starling's forces.
- One of the Starling's forces is the plasma oncotic pressure and other forces are—Capillary hydrostatic pressure, interstitial hydrostatic pressure and osmotic pressure in the interstitium.
- So the hydrostatic pressure gradient (Hydrostatic pressure in capillary—hydrostatic pressure in interstitium) and the osmotic pressure gradients (Colloidal oncotic pressure in capillary—colloidal osmotic pressure in interstitium) decide filtration across capillaries.
- In the arteriolar end of the capillaries, the hydrostatic pressure is 36 mm Hg and the interstitial hydrostatic pressure varies in different tissues, it is subatmospheric in subcutaneous tissues, positive in liver and 6 mm Hg in the brain.
- The oncotic pressure in capillary is 25 mm Hg and in the interstitium it is negligible.
  - So fluid movement = K[(Pc + πi) – (Pi + πc)]
  - K = Filtration co-efficient
  - Pc = Hydrostatic pressure in capillary (37 mm Hg in arteriolar end and 17 mm Hg in venous end))
  - Pi = Hydrostatic pressure in interstitium (1 mm Hg)
  - πc = Colloidal oncotic pressure in plasma (25 mm Hg)
  - πi = Colloidal osmotic pressure in interstitium (0 mm Hg)
  - Pc and πi favors filtration and Pi and πc opposes filtration.
- In the arteriolar end of capillaries the hydrostatic pressure in capillaries exceeds the oncotic pressure and therefore filtration of fluid happens here.
- Along with the fluid filtered, nutrients also enter the tissues.
- As fluid moves out of the capillaries, in the venous end, the hydrostatic pressure in capillaries drops to 17 mm Hg and the colloid oncotic pressure is 25 mm Hg and this favors reabsorption in the venous end of capillaries along with the metabolic wastes.
- In the glomerulus, throughout length of the capillary, the hydrostatic pressure exceeds the oncotic pressure and therefore fluid moves out of the capillaries throughout the length of the capillary.

***Applied Aspects***

In hypoproteinemia due to malnutrition, liver diseases (synthesis of protein is less) and in nephrosis (albumin is lost in urine), the plasma colloidal oncotic pressure decreases resulting in edema.

## 3. Excitation contraction coupling in skeletal muscle.

The process by which the muscle membrane is excited and followed by muscle contraction is given as excitation-contraction coupling.

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**The steps are given below:**

- The action potential generated following the end-plate potential (EPP) moves through the T-tubule and activates the voltage-gated dihydropyridine receptors (DHPRs) which are present in the T tubule membrane.
- The activated DHPRs open the $Ca^{2+}$ channels named as the Ryanodine receptors which are located in the terminal cisterns of the sarcoplasmic reticulum (SR).
- This releases $Ca^{2+}$ from the SR into the cytoplasm and the $Ca^{2+}$ concentration in cytosol rises by 2000 times that of the resting state.
- The $Ca^{2+}$ now gets attached to the troponin C subunit.
- Troponin C-$Ca^{2+}$ complex now induce a conformational change in the tropomyosin and the tropomyosin is lifted up, exposing the myosin binding sites on actin molecules.
- So the myosin heads bind to the actin molecules and cross-bridge cycling starts resulting in muscle contraction.

### 4. Types of polycythemia and complications due to this condition

Polycythemia is increase in the RBC count above the normal value. It can be physiological or pathological.

**Physiological Polycythemia**

- **Age:** At birth, the count is high (6–7 million cells/cumm) and after birth it starts decreasing due to destruction of RBCs.
- **High altitude:** Hypoxia in high altitude stimulates production of erythropoietin which in turn stimulates RBC production.
- Excessive exercise

**Pathological Polycythemia—2 Types**

1. **Primary polycythemia or polycythemia vera:** It is said to be a member of myeloproliferative syndrome and is due to the cancerous condition of the bone marrow. The counts increase more than 10 million cells/cumm. A high WBC count may also be seen. In this condition, erythropoietin levels may be very low.
2. **Secondary polycythemia:** It happens due to hypoxic conditions in the body as in lung (Emphysema, COPD) and cardiovascular diseases (Cyanotic heart diseases). The hypoxia stimulates synthesis of erythropoietin and thereby increased production of RBCs.

**Complications of Polycythemia**

Increase in RBC count, increases the viscosity of blood and thereby increases the peripheral resistance leading to hypertension and left heart failure.

### 5. Findings of 'tests of hemostasis' in hemophilia.

Hemophilia is a bleeding disorder due to the absence of clotting factor VIII. It is a genetic disorder and is X-linked recessive disease. The males are always affected and the females are careers.

**The laboratory tests in hemophilia:**

- **Bleeding time** is normal as the platelet count and the capillary integrity is normal
- **Capillary fragility test** is normal
- **Platelet count** is also normal
- **Coagulation time:** It is the time taken for the blood to clot in a capillary tube by formation of fibrin thread. It is prolonged in hemophilia.
- **Prothrombin time:** It is done by Quick's one stage method. Here the anticoagulated plasma of the patient is mixed with commercially available tissue thromboplastin and calcium chloride solution and is incubated at 37°C. The time at which a gel is formed is taken to be the PT. Normally it is around 11–16 seconds. Only extrinsic pathway is assessed by this method and therefore it is normal in hemophilia.
- **Partial thromboplastin time (PTT):** Here to the anticoagulated plasma, Kaolin (surface agent), calcium chloride and cephalin are added and incubated at 37°C. The time taken to form the gel is the PTT. It gives an idea about the intrinsic pathway.

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**V Suganthi** MD (Physiology) is Professor and Head (Department of Physiology) at Vinayaka Mission's Kirupananda Variyar Medical College and Hospitals, Salem, Tamil Nadu, India, and Executive Editor at National Journal of Basic Medical Sciences. She is working in the Institution for the past 16 years. She is involved in teaching the undergraduate and postgraduate students and she is also guiding the PhD scholars. She has received "Dr Srinivasan Award for best research paper" for postgraduates in the APPI conference in 2004. She has also received the best Poster Award in ASSOPICON conducted in JIPMER, Puducherry, India in the year 2014. She is the Executive Editor in the National Journal of Basic Medical Sciences for the past 4 years. She has published more than 30 papers in indexed international and national journals. She has co-authored a textbook "A Concise Textbook of Physiology".

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