

# Respiratory System

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## Questions

- 400. Muscles of expiration - (MAHE 98)**
- Diaphragm
  - Internal intercostal
  - External intercostal
  - Rectus Abdominis
- 401. Vital capacity is a measure of (Kerala 91)**
- Tidal volume
  - Inspiratory reserve volume plus expiratory reserve volume
  - Tidal volume plus inspiratory reserve volume plus expiratory reserve volume
  - Expiratory reserve volume plus reserve volume
- 402. The causes of pulmonary edema include all except - (AP 83)**
- Increased negative pressure
  - Increased pulmonary arteriolar pressure
  - Increased pulmonary capillary permeability
  - Increased plasma colloid pressure
- 403. The alveolar ventilation in an individual with tidal volume: 600ml, dead space 150ml and respiratory rate of 15/minute is- (JIPMER 93)**
- 2.5 lit/min
  - 4.0 lit/min
  - 6.75 lit/min
  - 9.0 lit/min
- 404. In normal adult, the lung is kept dry because of (Kerala 91)**
- Osmotic pressure
  - Surfactant
  - Hydrostatic pressure
  - Tidal volume
- 405. Limitation of inspiration by vagal lung inflation signals is called the (AP 85)**
- Autonomic reflex
  - Bainbridge reflex
  - Hering - Breur reflex
  - Dynamic stretch reflex

- 406. During the initial part of inspiration, which of the following does not occur (DNB 90)**
- Intrapulmonary pressure falls
  - Intrathoracic pressure rises
  - Intraabdominal pressure rises
  - The partial pressure of  $O_2$  in dead space rises
- 407. Rise of pulmonary arterial pressure is caused by - (PGI 88)**
- Hypoxia
  - Acidosis
  - Alkalosis
  - All
- 408. Total dead space can be calculated from - (UPSC 80)**
- $PCO_2$  of expired air
  - $PCO_2$  arteriolar blood
  - Tidal volume
  - All of the above
- 409.  $FEV_1$  is decreased in (TN 95)**
- Pulmonary TB
  - Fibrosing alveolitis
  - Chronic bronchitis
  - Bronchogenic carcinoma
- 410. Intrapleural pressure at the end of deep inspiration is - (AIIMS 98)**
- 4mm Hg
  - +4mm Hg
  - 18mm Hg
  - +18mm Hg
- 411. Effort during normal respiration is done due to- (DELHI 90)**
- Lung elasticity
  - Respiratory air passages
  - Alveolar air spaces
  - Creating negative pleural pressure
- 412. Oxygen affinity decreases in - (CU 2000)**
- Hypoxia
  - Hypothermia
  - HbF
  - Increase pH

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**413. Function of mucociliary action of upper respiratory tract is - (Kerala 94)**

- a. Protective
- b. Increase the velocity of inspired air
- c. Traps the pathogenic organisms in inspired air
- d. Has no physiological role

**414. Which of the following does not stimulate alveolar hyperventilation - (TN 94)**

- a. Hypoxia
- b. Hypercapnia
- c. Acidosis
- d. Stretching of airways

**415. Respiratory acidosis can cause - (JIPMER 91)**

- a. Decreased  $\text{PCO}_2$  and decreased pH
- b. Increased  $\text{PCO}_2$  and decreased pH
- c. Increased  $\text{PCO}_2$  and increased pH
- d. Decreased  $\text{PCO}_2$  and increased pH

**416. Alveolar  $\text{O}_2$  tension is (KAR 94)**

- a. Increased by hyperventilation
- b. Decreased by hyperventilation
- c. Increased  $\text{PCO}_2$  and increased pH
- d. Decreased  $\text{PCO}_2$  and increased pH

**417. Which occurs after hyperventilation with 6%  $\text{CO}_2$  - (AIIMS 91)**

- a. Apnea
- b. Continued hyperventilation
- c. Cheyne's stokes breathing
- d. Kussmaul's breathing

**418.  $\text{CO}_2$  affects respiratory center via - (JIPMER 80)**

- a. CSF  $\text{H}^+$  concentration
- b. Carotid body
- c. Inflation and deflation reflex
- d. Aortic body

**419. Increased fetal cortisol just before birth results in- (AI 88)**

- a. Uterine contraction
- b. Release of oxytocin
- c. Placental steroid biogenesis
- d. Fetal lung maturation

- 420. In strenuous exercise,  $PCO_2$  (mm Hg) falls from -**
- 40 to 15
  - 60 to 35
  - 25 to 10
  - 35 to 0
- 421. Hypoxia causes vasoconstriction in - (JIPMER 99)**
- Muscle
  - Lungs
  - Liver
  - Spleen
- 422. The most common form of hypoxia is (AIIMS 86)**
- Hypoxic
  - Stagnant
  - Anemic
  - Histotoxic
- 423. J receptor stimulation causes - (AIIMS 92)**
- Tachypnea
  - Apnea
  - Tachycardia
  - Hypotension
- 424. Structure through which  $O_2$  must diffuse in passing from alveolar lumen to hemoglobin (PGI 81)**
- Surfactant containing liquid
  - Alveolar membrane, basement membrane
  - Capillary endothelium, plasma and RBC membrane
  - All of the above
- 425. Hypoxia is characterised by - (AI 89)**
- Low arterial  $PO_2$
  - Intense chemoreceptor response
  - Favourable response to 100%  $O_2$
  - All of the above
- 426. An increase in ventilation occurs in all situations except - (AIIMS 84)**
- Fall in plasma bicarbonate
  - Sleep
  - Fall in pH of CSF
  - Rise in blood adrenaline level

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**427. In a normal adult the ratio of physiological and anatomical dead space is - (PGI 84)**

- a. 2:1
- b. 1:3
- c. 3:1
- d. 1:1

**428. Ventilation perfusion ratio is maximum in - (AI 98)**

- a. Base of lung
- b. Apex of lung
- c. Post. lobe of lung
- d. Middle lobe of lung

**429. Dissolved oxygen is not dependent on – (Jharkand 03)**

- a. Hb
- b. Atmospheric pressure
- c. Alveolar pressure
- d. Arterial tension of O<sub>2</sub>

**430. In Caissons disease all seen except – (Jharkand 03)**

- a. Myonecrosis
- b. Lymphedema
- c. Paraplegia
- d. None

**431. Surfactant is – (PGI 04)**

- a. Secreted by 26<sup>th</sup> week of GA
- b. Deficiency causes R.D.S in new born
- c. Composed of cytokeratin
- d. It can be used therapeutically
- e. Increased in bronchoalveolar carcinoma

**432. Surfactant is – (PGI 04)**

- a. Secreted by Type I pneumocytes
- b. Secreted by bronchial glands
- c. Contains mucin
- d. Necessary for alveolar stability
- e. Secreted as eosinophilic nodules

**433. Least amount of CO<sub>2</sub> is in – (Jipmer 04)**

- a. Anatomical dead space-end inspiration phase
- b. Anatomical dead space-end expiration phase
- c. Alveoli-end inspiration phase
- d. Alveoli-end expiration phase

- 434. Functional residual capacity of lung is defined as – (PGI 97)**
- Volume expired after normal expiration
  - Volume remaining after forced expiration
  - ERV+RV
  - Tidal volume + volume inspired forcefully
- 435. Arterial blood O<sub>2</sub> in ml of O<sub>2</sub> per dL – (PGI 02)**
- 12.1
  - 19.8
  - 15.6
  - 27.8
- 436. Administration of pure O<sub>2</sub> to hypoxic patients is dangerous because- (PGI 99)**
- Apnea occurs due to hypostimulation of peripheral chemoreceptors
  - Pulmonary edema
  - DPG
  - Convulsions
- 437. In high altitude mountain sickness, feature of pulmonary edema is – (PGI 99)**
- Decreased pulmonary capillary permeability
  - Increased pulmonary capillary pressure
  - Normal left atrial pressure
  - Increases left ventricular back pressure
- 438. Hyperbaric oxygen is dangerous because it – (PGI 99)**
- Decreases displacement of O<sub>2</sub> from Hb
  - Decreases respiratory drive
  - Enzyme damage
  - Is toxic to tissues
- 439. Central cyanosis is seen if – (PGI 01)**
- Methemoglobin 0.5 gm/dl
  - O<sub>2</sub> saturation < 85%
  - O<sub>2</sub> saturation < 94%
  - Reduced Hb – 4 gm%
- 440. Oxygen dissociation curve shift to right in – (PGI 02)**
- Hypothermia
  - Hypercarbia
  - Fetal Hb
  - Sickle cell Hb

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**441. Which of the following is seen in high altitude climbers – (PGI 01)**

- a. Hyperventilation
- b. Decreased PaCO<sub>2</sub>
- c. Pulmonary edema
- d. Hypertension
- e. Bradycardia

**442. Carbon dioxide retention is seen in the following condition- (PGI 01)**

- a. Carbon monoxide poisoning
- b. Lung failure
- c. Drowning
- d. Ventilatory failure
- e. High altitude

**443. A mountaineer ascents 18,000 feet in 2 days without supplemental oxygen. At the height of ascent the changes are- (PGI 03)**

- a. ↑ed Pa CO<sub>2</sub>
- b. ↓ed Barometric pressure
- c. ↓ ed inspired O<sub>2</sub>
- d. ↓ ed PaO<sub>2</sub>
- e. ↑ ed pH

**444. Peripheral chemoreceptors are stimulated maximally by – (TN 2002)**

- a. Cyanide
- b. Anaemia
- c. Hypocapnia
- d. Alkalosis

**445. If we cut the spinal cord above medulla, what happens to respiration- (AIIMS 2K)**

- a. It becomes slower and deeper
- b. Apneustic breathing
- c. Breathing ceases
- d. Irregular & gasping

**446. Hypoxia causes vasoconstriction in – (JIPMER 99)**

- a. Muscle
- b. Lungs
- c. Liver
- d. Spleen

**447. CO<sub>2</sub> is transported in plasma – (AI 99)**

- a. Bicarbonate
- b. Carbamino compounds
- c. Dissolved form
- d. CO

**448. Non-respiratory function of the lung is – (MAHE 98)**

- a. Dopamine metabolism
- b. Adrenaline metabolism
- c. Serotonin metabolism
- d. PGE<sub>2</sub> production

**449. In moderate exercise stimulation of respiration is due to – (MP 98)**

- a. Stimulation of J receptor
- b. Stimulation of lung receptor
- c. Joint Proprioception receptor
- d. Stimulation of medullary center

**450. Ventilation perfusion Ratio is maximum in – (AI 98)**

- a. Base of lung
- b. Apex of lung
- c. Post lobe of lung
- d. Middle lobe of lung

**451. Lung diffusion capacity is measured with – (NMS 96)**

- a. CO<sub>2</sub>
- b. CO
- c. O<sub>2</sub>
- d. H<sub>2</sub>

**452. Pulmonary surfactant reduces the following except – (Karnataka 96)**

- a. The filtration forces from pulmonary capillaries
- b. The surface tension in the Lungs
- c. Transpulmonary pressure
- d. Alveolar radius

# Respiratory System

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**400. (b) Internal intercostals  
(d) Rectus Abdominis**

Ref: Ganong 653

- ◆ A decrease in intrathoracic volume and forced expiration result when the expiratory muscles contract
- ◆ The internal intercostals have this action because they pass obliquely downward and posteriorly from rib to rib and therefore pull the rib cage downward when they contract
- ◆ Contractions of the muscles of the anterior abdominal wall also aid expiration by pulling the rib cage downwards and inward & by increasing the intra-abdominal pressure, which pushes the diaphragm upward

**401. (c) Tidal volume + inspiratory reserve volume + expiratory reserve volume**

Ref: Ganong 652 fig 34-7

		VOLUME (L)			
		MEN	WOMEN		
Vital Capacity	[	IRV	3.3	1.9	] Inspiratory Capacity
	]	TV	0.5	0.5	
	]	ERV	1.0	0.7	] Functional residual Capacity
		RV	1.2	1.1	
Total lung capacity		6.0	4.2		

**402. (d) Increased plasma colloid pressure**

Ref: Ganong 662 for c, 594, Table 30.4

Table 3-4 Causes of Increased interstitial fluid volume and edema

Increased filtration pressure :-

- ◆ Arterial dilatation
- ◆ Venular constriction
- ◆ Increased venous pressure (heart failure, incompetent valves, venous obstruction, increased total ECF volume, effect of gravity, etc.,)

Decreased osmotic pressure gradient across capillary

- ◆ Decreased plasma protein level
- ◆ Accumulation of osmotically active substances in interstitial space

Increased capillary permeability

- ◆ Substance P
- ◆ Histamine and related substances
- ◆ Kinins, etc.,

Inadequate lymph flow

- ◆ Pulmonary capillary pressure is about 10mmHg, where as the oncotic pressure is 25mm of Hg, so that an inward-directed pressure gradient of about 15mm Hg keeps the alveoli free of all but a thin film of fluid
- ◆ When the pulmonary capillary pressure is more than 25mmHg - as it may be, for example, in “backward failure” of the left ventricle - pulmonary congestion and edema result

**403. (c) 6.75 lit/min**

Ref: *Ganong 659, Table 34.4*

Table 34-4. Effect of variations in respiratory rate and depth on alveolar ventilation

Respiratory rate	30/min	10/min
Tidal volume	200ml	600ml
Minute volume	6L	6L
Alveolar ventilation	= (200-150) x 30 = 1500ml	(600-150) x 10 = 4500ml

Similarly in our subject (person)

$$\begin{aligned} \text{Alveolar ventilation} &= (\text{Total volume} - \text{dead space}) \times \text{breath/min} \\ &= (600-150) \times 15 \\ &= 6750\text{ml/min} \\ &= 6.75 \text{ Lt/min} \end{aligned}$$

**404. (a) Osmotic pressure****(c) Hydrostatic pressure**

Ref: *Ganong - 592, 594, Table 30-4*

$$\text{Fluid movement} = K[(P_c - P_i) - (\pi_c - \pi_i)]$$

Where

- K = Capillary filtration coefficient
- P<sub>c</sub> = Capillary hydrostatic pressure
- P<sub>i</sub> = Interstitial hydrostatic pressure
- π<sub>c</sub> = Capillary colloid osmotic pressure
- π<sub>i</sub> = interstitial colloid osmotic pressure

- ◆ π<sub>i</sub> is usually negligible, so the osmotic pressure gradient (π<sub>c</sub> - π<sub>i</sub>) usually equals the oncotic pressure
- ◆ The capillary filtration co-efficient takes into account, and is proportionate to, the permeability of the capillary wall and the area available for filtration

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- ◆ Fluid moves into the interstitial space at the arteriolar end of the capillary, where the filtration pressure across its wall exceeds the oncotic pressure, and into the capillary at the venular end, where the oncotic pressure exceeds the filtration pressure

### 405. (c) Hering - Breur reflex

*Ref: Ganong - 678*

- ◆ The shortening of inspiration produced by vagal afferent activity is mediated by slowly adapting receptors
- ◆ So are the Hering - Breur reflexes
- ◆ The Hering - Breur inflation reflex is an increase in the duration of expiration produced steady lung inflation, and the Hering - Breur deflation reflex is a decrease in the duration of expiration produced by marked deflation of the lung
- ◆ Because the rapidly adapting receptors are stimulated by chemicals such as histamine, they have been called irritant receptors

### 406. (b) Intrathoracic pressure rises

*Ref: Ganong - 651, 655 fig 34-10, 659 for d*

- ◆ Inspiration is an active process. The contraction of the inspiratory muscles increases intrathoracic volume
  - ◆ The intrapleural pressure at the base of the lungs, which is normally about - 2.5mm Hg (relative to atmospheric) at the start of inspiration, decreases to about - 6mm Hg.
  - ◆ The lungs are pulled into a more expanded position
- Pg - 658
- ◆ Normally, the volume of this anatomic dead space is approximately equal to the body weight in pounds. Thus in a man who weights 150 lb(68kgs), only the first 350ml of the 500ml inspired with each breath at rest mixes with the air in the alveoli
  - ◆ Conversely, with each expiration, the first 150ml expired is gas that occupied the dead space, and only the last 350ml is gas from the alveoli.

### 407. (a) Hypoxia

#### (b) Acidosis

*Ref: Ganong - 663 & 664*

- ◆ When a bronchus (or) bronchiole is obstructed, hypoxia develops in the underventilated alveoli beyond the obstruction
- ◆ The O<sub>2</sub> deficiency apparently acts directly on the vascular smooth muscle in the area to produce constriction, shunting blood away from the hypoxic area.
- ◆ Accumulation of CO<sub>2</sub> leads to a drop in pH in the area, and a decline in pH also produces vasoconstriction in the lungs, as opposed to the vasodilation it produces in other tissues

- ◆ Conversely, reduction of the blood flow to a portion of the lung lowers the alveolar  $\text{PCO}_2$  in that area, and this leads to constriction of the bronchi supplying it, shifting ventilation away from the poorly perfused area
- ◆ Systemic hypoxia also causes the pulmonary arterioles to constrict, with a resultant increase in pulmonary arterial pressure.

**408. (d) All of the above**

*Ref: Ganong - 659*

- ◆ The total dead space can be calculated from the  $\text{PCO}_2$  of expired air, the  $\text{PCO}_2$  of arterial blood and the tidal volume
- ◆ The tidal volume ( $V_T$ ) times the  $\text{PCO}_2$  of expired gas ( $P_{\text{ECO}_2}$ ) equals the arterial  $\text{PCO}_2$  ( $\text{PaCO}_2$ ) times the difference between the Tidal volume and the dead space ( $V/D$ ) plus the  $\text{PCO}_2$  of inspired air ( $P_{\text{ICO}_2}$ ) times  $V/D$  (Bohr's equation)

$$P_{\text{ECO}_2} \times V_T = \text{Pa CO}_2 \times (V_T - V_D) + P_{\text{ICO}_2} \times V_D$$

The term  $P_{\text{ICO}_2} \times V/D$  is so small that it can be ignored and the equation solved for  $V/D$ : If for example;

$$P_{\text{ECO}_2} = 28\text{mm Hg}$$

$$\text{PaCO}_2 = 40\text{mm Hg}$$

$$V_T = 500\text{ml}$$

then

$$V_D = 150\text{ml}$$

- ◆ The equation can also be used to measure the anatomic dead space if one replaces  $\text{PaCO}_2$  with alveolar  $\text{PCO}_2$  ( $P_A \text{CO}_2$ ), Which is the  $\text{PCO}_2$  of the last 10ml of expired gas.

**409. (c) Chronic bronchitis**

*Ref: Ganong - 652*

The fraction of the vital capacity expired during the first second of a forced expiration ( $\text{FEV}_1$ , timed vital capacity) gives additional information.

The vital capacity may be normal but the  $\text{FEV}_1$  reduced in diseases such as asthma, in which airway resistance is increased because of bronchial constriction.

**410. (c) -18mm Hg**

*Ref: Ganong - 651*

- ◆ The intrapleural pressure at the base of the lungs, which is normally about - 2.5mm Hg (relative to atmospheric) at the start of inspiration, decreases to about - 6mm Hg.
- ◆ The pressure in the airway becomes slightly negative, and air flows into the lungs
- ◆ At the end of inspiration, the lung recoil begins to pull the chest back to the expiratory position where the recoil pressures of the lung & chest wall balance

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- ◆ The pressure in the airway becomes slightly positive, and airflows out of the lungs
- ◆ Strong inspiratory efforts reduce intrapleural pressure to values as low as - 30mm Hg, producing correspondingly greater degrees of lung inflation. Since in the question we are not dealing with normal inspiration which reduces the intrapleural pressure to - 6mm Hg, but with deep inspiration which reduces the intrapleural pressure even more, and with regards to the reference given above which states that the intrapleural pressure reduces upto - 30mm Hg with strong inspiratory efforts. I definitely think that - 18mm Hg is the right answer.

### 411. (a) Lung elasticity

*Ref: Ganong - 650- 651*

- ◆ The lungs and the chest wall are elastic structures. Normally, no more than a thin layer of fluid is present between the lungs and chest wall (intrapleural space)
- ◆ The lungs slide easily on the chest wall but resist being pulled away from it in the same way that two moist pieces of glass slide on each other but resist separation.
- ◆ The pressure in the intrapleural space is subatmospheric
- ◆ The lungs are stretched when they expand at birth, and at the end of quiet expiration their tendency to recoil from the chest wall is just balanced by the tendency of the chest wall to recoil in the opposite direction
- ◆ Inspiration is an active process
- ◆ Expiration during quiet breathing is passive in the sense that no muscles, that decrease intrathoracic volume, contract. Since it is only during inspiration that effort is needed to stretch the lung, the force that needs to be overcome in order to do this is provided by the elasticity of lung.

### 412. (a) Hypoxia

*Ref: Ganong - 667*

Factors affecting the Affinity of Hemoglobin for oxygen

Three important conditions affect the oxygen - hemoglobin-dissociation curve -

- ◆ pH
- ◆ temperature
- ◆ 2,3 - biphosphoglycerate (2,3 - BPG)
- ◆ A rise of temperature (or) fall in pH shifts the curve to right, hence a higher  $PO_2$  is required for hemoglobin to bind a given amount of  $O_2$ . i.e. the Affinity decreases
- ◆ A fall in temperature (or) a rise in pH shifts the curve to left, and a lower  $PO_2$  is required to bind a given amount of  $O_2$  i.e. Affinity increases

O<sub>2</sub> Affinity of Hb increases in (Curve shifts to the right)

- ◆ Hypothermia
- ◆ Increase in pH

O<sub>2</sub> Affinity of Hb decreases in (Curve to the left)

- ◆ Hyperthermia
- ◆ decrease in pH
- ◆ Hypoxia → CO<sub>2</sub> ↑ → ↓pH

**413. (c) Traps the pathogenic organisms in inspired air**

*Ref: Ganong - 665*

"Ciliary escalator"

- ◆ The epithelium of the respiratory passages from the anterior third of the nose to the beginning of the respiratory bronchioles is ciliated, and the cilia, which are covered by mucus, beat in a co-ordinated fashion at a frequency of 1000-1500 cycles per minute.
- ◆ The ciliary mechanism is capable of moving particles away from the lungs at a rate of at least 16mm/min. Particles less than 2µm in diameter generally reach the alveoli, where they are ingested by the macrophages
- ◆ When ciliary motility is defective, mucus transport is virtually absent. This leads to :-
  - Chronic sinusitis
  - Recurrent lung infections
  - Bronchiectasis
- ◆ In Kartagener's syndrome, in which the axonemal dynein, the ATPase molecular motor that produces ciliary beating is absent.
- ◆ Patients with this condition are infertile because they lack mobile sperm, and they often have situs inversus, presumably because the cilia necessary for rotating the viscera are non-functional during embryonic development.

**414. (b) Hypercapnia**

**(d) Stretching of airways**

*Ref: Ganong - 675, 676*

Not so sure about (d)

- ◆ The chemoreceptors that mediate the hyperventilation produced by increases in arterial PCO<sub>2</sub> after the carotid and aortic bodies are denervated are located in the medulla oblongata and consequently are called medullary chemoreceptors
- ◆ They are separate from the dorsal and ventral respiratory neurons and are located on the ventral surface of the medulla  
Recent evidence indicates that additional chemoreceptors are located in the vicinity of the solitary tract nuclei, the locus ceruleus, and the hypothalamus

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- ◆ The chemoreceptors monitor the  $H^+$  concentration of CSF, including the brain interstitial fluid
- ◆  $CO_2$  readily penetrates membranes, including the blood-brain barrier, where as  $H^+$  and  $HCO_3^-$  penetrate slowly
  - The  $CO_2$  that enters the brain and CSF is promptly hydrated
  - The  $H_2CO_3$  dissociates, so that the local  $H^+$  concentration rises
  - The  $H^+$  concentration in brain interstitial fluid parallels the arterial  $PCO_2$
  - Any increase in spinal fluid  $H^+$  concentration stimulates respiration
- ◆ The magnitude of the stimulation is proportionate to the rise of  $H^+$  concentration. Thus, the effects of  $CO_2$  on respiration are mainly due to its movement into the CSF and brain interstitial fluid, where it increases the  $H^+$  concentration and stimulates receptors sensitive to  $H^+$ .  
Thus,
  - Acidosis
  - Hypoxiawill cause stimulation of respiration

Pg - 676

When the  $CO_2$  content of the inspired gas is more than 7%, the alveolar and arterial  $PCO_2$  begin to rise abruptly in spite of hyperventilation.

The resultant accumulation of  $CO_2$  in the body - hypercapnia depresses the central nervous system, including the respiratory center, and produces headache, confusion and eventually coma -  $CO_2$  narcosis (for option d)

Pg 679:- Table 36-2 gives. Airway & lung receptors

<b>Vagal Innervation</b>	<b>Type</b>	<b>Location in Interstitium</b>	<b>Stimulus</b>	<b>Response</b>
Myelinated	Slowly adapting	Among airway smooth muscle cells (?)	Lung Inflation	<ul style="list-style-type: none"> <li>- Inspiratory time shortening</li> <li>- Hering - Breur inflation &amp; deflation reflex</li> <li>- Bronchodilation</li> <li>- Tachycardia</li> </ul>
	Rapidly adapting (irritant receptors)	Among airway epithelial cells	Lung hyper-inflation. Exogenous & Endogenous Substances, (eg. histamine, prostaglandins)	<ul style="list-style-type: none"> <li>Hyper apnea</li> <li>Cough</li> <li>Bronchoconstriction</li> <li>Mucus secretion</li> </ul>
Unmyelinated	Pulmonary C fibres. Bronchial C fibres (J - juxta-capillary receptors)	Close to blood vessels	Lung hyper inflation Exogenous & Endogenous Substances (eg. Capsaicin, bradykinin, serotonin)	<ul style="list-style-type: none"> <li>- Apnea followed by rapid breathing</li> <li>- Broncho constriction</li> <li>- Bradycardia</li> <li>- Hypertension</li> <li>- Mucus secretion</li> </ul>

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### 415. (b) Increased $\text{PCO}_2$ and decreased pH

Ref: Ganong - 734

- ◆ A rise in arterial  $\text{PCO}_2$  due to decreased ventilation causes respiratory acidosis
- ◆ The  $\text{CO}_2$  that is retained is in equilibrium with  $\text{H}_2\text{CO}_3$ , which in turn is in equilibrium with  $\text{HCO}_3^-$ , so that plasma  $\text{HCO}_3^-$  rises and a new equilibrium is reached at a lower pH
- ◆ Conversely a decline in  $\text{PCO}_2$  causes respiratory alkalosis

### 416. (a) Increased by hyperventilation

Ref: Ganong - 692, 693

When a normal individual hyperventilates for 2-3 minutes, then stops and permits respiration to continue without exerting any voluntary control over it, a period of apnea occurs.

- ◆ This is followed by a few shallow breaths and then by another period of apnea, followed again by a few breaths - periodic breathing
- ◆ The cycles may last for some time before normal breathing is resumed
- ◆ The apnea apparently is due to  $\text{CO}_2$  lack because it does not occur following hyperventilation with gas mixtures containing 5%  $\text{CO}_2$ .
- ◆ During the apnea, the alveolar  $\text{PO}_2$  falls and the  $\text{PCO}_2$  rises

### 417. (b) Continued hyperventilation

Ref: Ganong 692, 693, fig 37.9

- ◆ The concept of periodic breathing is explained in Q.388 and it occurs in various disease states and is often called cheyne-stokes respiration
- ◆ When someone hyperventilates there is  $\text{CO}_2$  washout which reduces the  $\text{H}^+$  ion concentration (respiratory alkalosis) and thus reduces the drive for respiration  
A fact to remember in cases when we give  $\text{O}_2$  therapy, as in some cases if increased concentration of  $\text{O}_2$  are given, the sole reason for the patient to respire is lost and respiration can be lost completely  
Pg - 692 - gives the following with respect to why apnea occurs in periodic breathing, when one hyperventilates.
- ◆ The apnea apparently is due to  $\text{CO}_2$  lack because it does not occur following hyperventilation with gas mixture containing 5%  $\text{CO}_2$ .  
That means that the person would continue to hyperventilate, and this would also happen at 6%  $\text{CO}_2$  gas mixture concentration.

### 418. (a) C.S.F $\text{H}^+$ conc

Ref: Ganong 675

- ◆ The chemoreceptors monitor the  $H^+$  concentration of C.S.F, including the brain interstitial fluid
  - ◆  $CO_2$  readily penetrates membranes, including the blood-brain barrier where as  $H^+$  and  $HCO_3^-$  penetrate slowly.
  - ◆ The  $CO_2$  that enters the brain and CSF is promptly hydrated
  - ◆ The  $H_2CO_3$  dissociated so that  $H^+$  concentration rises
  - ◆ The magnitude of the stimulation is proportionate to the rise in  $H^+$  concentration
- 419. (d) Fetal lung maturation**  
*Ref: Ganong - 657*  
"Maturation of surfactant in the lungs is accelerated by glucocorticoid hormones. Fetal and maternal cortisol near term, and the lungs are rich in glucocorticoid receptors".
- 420. (a) 40 to 15**  
*Ref: Ganong 692*  
Hypocapnia is a result of hyperventilation. During voluntary hyperventilation (as in strenuous exercise), the arterial  $PCO_2$  falls from 40 to as low as 15mm Hg while alveolar  $PO_2$  rises to 120-140mm Hg
- 421. (b) Lungs**  
*Ref: Ganong - 674*
- ◆ The smooth muscle of pulmonary arteries contain  $O_2$ -sensitive  $K^+$  channels, which mediate the vasoconstriction caused by hypoxia (they reduce  $K^+$  efflux)
  - ◆ This is in contrast to systemic arteries, which contain ATP-dependant  $K^+$  channels that permit more  $K^+$  efflux with hypoxia and consequently cause vasodilation instead of vasoconstriction
- 422. (a) Hypoxic**  
*Ref: Ganong - 686*
- ◆ Hypoxic hypoxia is the most common form of Hypoxia seen clinically
  - ◆ The diseases that cause it can be roughly divided into those in which the gas exchange apparatus fails, those such as congenital heart disease in which large amounts of blood are shunted from the venous to the arterial side of the circulation, and those in which the respiratory pump fails.
- 423. (b) Apnea**  
**(a) Tachypnea**  
**(d) Hypotension**  
*Ref: Ganong - 678*

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- ◆ Because the C fiber endings are close to pulmonary vessels they have been called J (juxtacapillary) receptors.
- ◆ They are stimulated by hyperinflation of the lung but they respond as well to intravenous (or) intracardiac administration of chemicals such as capsaicin
- ◆ The reflex response that is produced is apnea followed by rapid breathing, bradycardia, and hypotension (pulmonary chemoreflex)  
I think this question should have been framed as an "except" type of question in which case the answer would have been (c) Tachycardia

### 424. (d) All of the above

Ref: Ganong - 660

- ◆ Gases diffuse from the alveoli to the blood in the pulmonary capillaries (or) vice-versa across the thin alveolocapillary membrane made up of the pulmonary epithelium, the capillary endothelium, and their fused basement membranes
- ◆ Whether or not substances passing from the alveoli to the capillary blood reach equilibrium in the 0.75s that blood takes to traverse the pulmonary capillaries at rest depends on their reaction with substances in the blood
- ◆ Surfactant is secreted by type II pneumocytes in the alveoli, and RBC are the O<sub>2</sub> carriers (As in Hb carriers), thus it is obvious that O<sub>2</sub> diffuses through them too.

### 425. (d) All of the above

Ref: Ganong - 683 for a, 691 for c, 674 & 675 for b

#### Hypoxia

- ◆ Hypoxia is O<sub>2</sub> deficiency at the tissue level. It is more correct term than anoxia, there rarely being no O<sub>2</sub> at all left in the tissues

There are four categories :-

- (1) Hypoxic hypoxia (anoxic anoxia) in which PO<sub>2</sub> of arterial blood is reduced
- (2) Anaemic hypoxia in which arterial PO<sub>2</sub> is normal but the amount of hemoglobin available to carry O<sub>2</sub> is reduced
- (3) Stagnant (or) ischemic hypoxia in which the blood flow to the tissues is so low that adequate O<sub>2</sub> is not delivered to it despite a normal PO<sub>2</sub> and hemoglobin concentration
- (4) Histotoxic hypoxia, in which the amount of O<sub>2</sub> delivered to a tissue is adequate but, because of action of toxic agent the tissue cells cannot make use of the O<sub>2</sub> supplied to them

Pg - 691 - When 100% O<sub>2</sub> is first inhaled, respiration may decrease slightly in normal individuals, suggesting that there is normally some hypoxic chemoreceptor drive

- ◆ However, the effect is minor and can be demonstrated only by special techniques

- ◆ In addition, it is offset by a slight accumulation of  $H^+$  ions, since the concentration of deoxygenated hemoglobin in the blood is reduced and Hb is a better buffer than  $HbO_2$ .

Table 36-1. stimuli affecting the respiratory center

Chemical control:

$CO_2$  ( via CSF and brain interstitial fluid  $H^+$  concentration)

$O_2$

$H^+$  ( via carotid & aortic bodies)

Non - chemical control

Vagal afferents from receptors in the airways & lung

Afferents from the pons, hypothalamus and limbic system

Afferents from proprioceptors

Afferents from baroreceptors, arterial, ventricular, pulmonary.

$\uparrow H^+$  concentration in C.S.F  $\rightarrow$  stimulates chemoreceptors in brain  
 $\rightarrow$  stimulate respiration

$\uparrow CO_2$  concentration  $\rightarrow$   $\uparrow H^+$  concentration

The glomus cells in carotid and aortic bodies contain type I and type II cells. The type I cells have  $O_2$  - sensitive  $K^+$  channels, whose conductance is reduced in proportion to the degree of hypoxia to which they are exposed.

**426. (b) Sleep**

*Ref: Ganong - 680*

- ◆ Respiration is less rigorously controlled during sleep than in the waking state, and brief periods of apnea occur in normal sleeping adults
- ◆ Changes in the ventilatory response to hypoxia vary
- ◆ If the  $PCO_2$  falls during the waking state, various stimuli from proprioceptors and the environment maintain respiration, but during sleep, these stimuli are decreased and a decrease in  $PCO_2$  can cause apnea.
- ◆ During R.E.M. sleep, breathing is irregular and the  $CO_2$  response is highly variable.

**427. (d) 1:1**

*Ref: Ganong - 659*

- ◆ In healthy individuals, the two dead spaces (anatomical & physiological) are identical Thus the ratio will be 1:1
- ◆ But in disease states, no exchange may take place between the gas in some of the alveoli and the blood, and some of the alveoli may be overventilated

**428. (b) Apex of lung**

*Ref: Ganong - 663*

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- ◆ Ventilation as well as perfusion, in the upright position declines in a linear fashion from the bases to the apices of the lungs
- ◆ However, the ventilation/ perfusion ratios are high in the upper portions of the lungs
- ◆ It is said that the high ventilation/perfusion ratios at the apices account for the predilection of tuberculosis for this area because the relatively high alveolar  $PO_2$  that results, provides a favourable environment for the growth of the tuberculosis bacteria.

### 429. (a) Hb.

Ref: (Guyton 11<sup>th</sup> Ed/Pg 546) fig 44-2)

- ◆ In the normal range of alveolar  $PO_2$  i.e. below 120 mm Hg almost none of the total oxygen is accounted for by dissolved oxygen.
- ◆ As the pressure rises to thousands of millimeters of mercury, a large portion of the total oxygen is the dissolved portion in the water of the blood.
- ◆ Hb is an option which is saturable, i.e. as far as the dissolved  $O_2$  in blood is concerned and any changes in hemoglobin do not affect its concentration after being saturated.
- ◆ But the other three options affect the concentration of dissolved  $O_2$  to a greater extent independent of the hemoglobin concentration.

### 430. (b) Lymphedema.

Ref: (Ganong 22<sup>nd</sup> Ed/ Pg 695, Guyton 11<sup>th</sup> Ed/Pg 549,548)

- ◆ **Caisson's disease** has many synonyms : as:-
  - Bends
  - Decompression sickness
  - Diver's paralysis
  - Dysbarism.
- ◆ It occurs in divers, compressed air is taken in order to match the pressures at deep sea.
- ◆ The air contains Nitrogen in almost 70 % of the mixture.
- ◆ At high pressures in the sea, the pressure over the body is balanced by the compressed air delivered to the lungs and the  $N_2$  stays dissolved in the tissues, more in fat than in other compartments.
- ◆ But if the diver ascends very quickly then the  $N_2$  is converted into its gaseous form as the pressure outside the body drops down to 1 atm. (760 mm Hg), at first they are smaller bubbles which later coalesce and progressively larger vessels are affected.
- ◆ Tissue ischemia and sometimes tissue death are the result.
- ◆ Coronaries getting blocked, cause myocardial damage.
- ◆ Most people with **bends** have **joint pains** and muscles of the arms and legs affecting 85-90%.

- ◆ 5-10% - Nervous system affection- dizziness (5%) collapse and unconsciousness 3%, the paralysis may be temporary, the damage can be permanent in some instances.
  - ◆ 2% - massive number of micro bubbles plugging the capillaries of the lung, "the chokes" → pulmonary edema → death (Occasionally).
- 431. (a) Secreted by 26<sup>th</sup> week of G.A.**  
**(b) Deficiency causes R.D.S in Newborn**  
**(c) It can be used therapeutically.**  
*Ref: (Ganong 22<sup>nd</sup> Ed/ Pg 656,657, Gray's Anatomy 39<sup>th</sup> Ed/ Pg. 1089)*
- o In the saccular stage, i.e. (24 weeks to birth) the surfactant production matures, which increases the chances of survival of fetus.
  - o Surfactant deficiency is an important cause of (I.R.D.A) Infant respiratory distress syndrome (also called hyaline membrane disease). Prolonged immaturity of the epithelial Na<sup>+</sup> channels cause Na<sup>+</sup> absorption by the pulmonary epithelial cells and thus fluid is also retained & contributes to I.R.D.S.
  - o In I.R.D.S, synthetic surfactant and a surfactant preparation derived from bovine lungs used prophylactically at birth & as replacement therapy decrease the severity of I.R.D.S and the severity of chronic lung disease in survivors, but does not affect its incidence.
  - o Surfactant maturity is accelerated by glucocorticoids. Therefore, in recent years premature babies and those who needed to be delivered preterm have been given cortisol, which releases fibroblast – pneumocyte factor accelerating lung maturity.
  - o There is sexual dimorphism in lung development, male type II cells are less mature than female counterparts. Androgen block effects of cortisol.
- 432. (d) Necessary for alveolar stability.**  
*Ref: (Ganong 22<sup>nd</sup> Ed/ Pg 655-657)*
- o Surfactant is a mixture of dipalmitoylphosphatidylcholine and other lipids & proteins. Formation of the phospholipid film is facilitated by proteins SP-A, SP-B, SP-C and SP-D.
  - o It is secreted by type II pneumocytes.
  - o Typical lamellar bodies, membrane-bound organelle containing whorls of phospholipid are formed in these cells and secreted into the alveolar lumen by exocytosis.
- 433. (a) Anatomical dead space at the end inspiration phase.**  
*Ref: (Ganong 22<sup>nd</sup> Ed/ Pg 660 fig 34-18)*

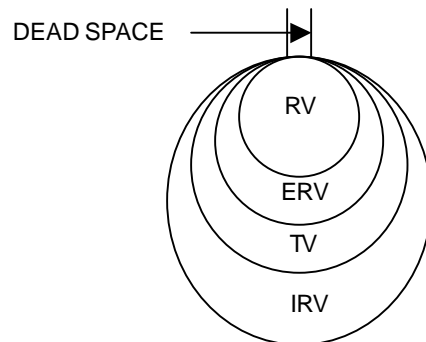
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- o In a normal healthy individual the anatomical dead space is equal to the physiological dead space which is **150ml in volume of the air**.
- o The anatomical dead space is due to the air passages, the trachea, the bronchi which are normally participating in transport of gases.
- o The pressure exerted by presence of CO<sub>2</sub> in inhaled air is minimum (0.3 mm Hg) and at the end of inspiration this is the concentration in the anatomical dead space, though there is a slight mixing at the interface with the air in the perfused part of the lungs.
- o The air in the alveoli is subject to a constant state of the pressures exerted by the constituent gases irrespective of the phase of respiration in a steady state, i.e. in absence of pathology. So they are maintained due to ventilation and perfusion to more or less constant values in a steady state in the alveoli.
- o At the end expiration phase the CO<sub>2</sub> from the alveoli comes to the anatomical dead space and its concentration is increased.

### 434. (c) ERV+RV.

Ref: (Ganong 22<sup>nd</sup> Ed/Pg 652 fig 34-7)

- ◆ Best way to remember it, is this fig.



- ◆ RV- Residual volume
- ◆ ERV- Expiratory reserve volume.
- ◆ TV- Tidal volume
- ◆ IRV – Inspiratory reserve volume.  
(which tells you which is a part of what )

$$\begin{aligned}\text{Vital Capacity} &= \text{IRV} + \text{TV} + \text{ERV} \\ &= 3.3\text{L} + 0.5\text{L} + 1.0\text{L} = \mathbf{4.8\text{ L}} \text{ (in men)} \\ &= 1.9\text{L} + 0.5\text{L} + 0.7\text{L} = \mathbf{3.1\text{ L}} \text{ (in women)} \\ \text{Inspiratory capacity} &= \text{IRV} + \text{TV}\end{aligned}$$

$$= 3.3\text{L} + 0.5\text{L} = \mathbf{3.8\text{L}}$$
 (in men)

$$= 1.9\text{L} + 0.5\text{L} = \mathbf{2.4\text{L}}$$
 (in women)

Functional residual capacity = TV + ERV

$$= 1.2\text{L} + 1.0\text{L} = \mathbf{2.2\text{L}}$$
 (men)

$$= 0.7\text{L} + 1.1\text{L} = \mathbf{1.8\text{L}}$$
 (women)

**435. (b) 19.8 ml/dL**

*Ref: (Ganong 22<sup>nd</sup> Ed/Pg 666,667)*

- ◆ When fully saturated each gram of normal hemoglobin contains 1.39 ml of O<sub>2</sub>, the blood normally contains small amount of inactive hemoglobin in derivatives. Thus the measured in vivo value is lower. The traditional figure is **1.34 ml of O<sub>2</sub>/ gram of Hb.**
- ◆ Men – 16 g/dL of Hb and women – 14 g/dL of Hb.
- ◆ On an average 15 g/dL of Hb.
- ◆ Thus, 1 dL of blood contains
  - 15 x 1.34 ml = 20.1 ml of O<sub>2</sub>
- ◆ But since due to the physiological shunt i.e. the slight admixture with venous blood that bypasses the pulmonary capillaries the hemoglobin in systemic arterial blood is only 97% saturated and hence the arterial blood therefore contains 19.8 ml/dL of O<sub>2</sub>, out of which, 0.29 ml is in solution and 19.5ml is bound to Hb.
- ◆ Venous blood – Hb is 75% saturated (at rest) O<sub>2</sub> content
  - 15.2 ml/dL
  - 0.12 ml in solution.
  - 15.1 ml bound to Hb.
- ◆ Tissues remove - 4.6 ml from each dL (at rest).
- ◆ 250 ml of O<sub>2</sub>/ minute is transported from blood to tissues at rest.

**436. (a) Apnea occurs due to hypostimulation of peripheral chemoreceptors.**

*Ref: (Ganong 22<sup>nd</sup> Ed/Pg 691)*

- ◆ “ In hypercapnic patients, in severe pulmonary failure, the CO<sub>2</sub> level may be so high that it depresses rather than stimulates respiration. Some of these patients keep breathing only because the carotid and aortic chemoreceptors drive the respiratory center.
- ◆ If the hypoxic drive is withdrawn by administering O<sub>2</sub>, breathing may stop.
- ◆ During the resultant apnea:-
- ◆ The arterial PO<sub>2</sub> drops but breathing may not start again because, the increase in PCO<sub>2</sub> further depresses the respiratory center.
- ◆ Therefore, O<sub>2</sub> therapy in this situation must be started with care.”

**437. (b) Increase pulmonary capillary pressure.**

*Ref: (Ganong 22<sup>nd</sup> Ed/Pg 685)*

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- ◆ High altitude illness not only includes mountain sickness but also two more serious syndromes that complicate it.

### 1) **High – altitude cerebral edema:-**

- ◆ In mountain sickness there is ;
  - irritability
  - headache
  - insomnia
  - breathlessness
  - nausea & vomiting
- ◆ The cause is thought to be cerebral edema. If the cerebral autoregulation does not compensate which is further aggravated to – frank brain swelling
  - ataxia
  - disorientation coma & death in some cases due to herniation of the brain through the tentorium.

### 2) **High altitude pulmonary edema:-**

- ◆ Patchy edema of lung related to marked pulmonary hypertension.
- ◆ Occurs because not all pulmonary arteries have enough smooth muscle to constrict in response to hypoxia.
- ◆ Thus the capillaries supplied by these arteries, there is high pressure due to rapid blood flow, causing damage to their walls and this results in the pulmonary edema.

### 438. (d) **Is toxic to tissues**

*Ref: (Ganong 22<sup>nd</sup> Ed/Pg 691)*

- ◆ Administration of 100% O<sub>2</sub> at increased pressure accelerates the onset of O<sub>2</sub> toxicity.
- ◆ Effects of Toxicity
  - Tracheobronchial irritation
  - Muscle twitching
  - Ringing in ears.(tinnitus)
  - Dizziness
  - Convulsions
  - Coma.
- ◆ The speed of symptom appearance is directly proportional to the pressure at which O<sub>2</sub> is administered.
- ◆ At 4 atmosphere, symptoms develop in 30 mins.
- ◆ At 6 atmosphere, convulsions develop in few minutes.

### 439. (b) & (d)

*Ref: (Harrison's 16<sup>th</sup> Ed/Pg 210)*

- Cyanosis refers to the bluish colour of the skin and mucous membranes resulting from an increased quantity of reduced hemoglobin, or of hemoglobin derivatives, in small blood vessels of those areas.

- o It is of mainly two types:
  - Central type
  - Peripheral type
- o Central type:-
  - ◆ Oxygen saturation ( $\text{SaO}_2$ ) is reduced due to
    - $\downarrow \text{FiO}_2$
    - Impaired pulmonary function
    - Shunting
  - ◆ Hemoglobin derivatives
    - Meth-hemoglobin
    - Sulfahemoglobin

Central cyanosis can be detected readily when the  $\text{SaO}_2$  has fallen to **85%**, in dark skinned individuals it may not be detected until it has declined to **75%**.

In general, cyanosis becomes apparent when the mean capillary concentration of reduced hemoglobin exceeds **40 g/L (4g/dL)**.

**440. (b) hypercarbia and (d) sickle cell Hb**

*Ref: (Ganong 22<sup>nd</sup> Ed/Pg.536, 667, 669)*

Three important conditions affect the oxygen-hemoglobin dissociation curve.

- ◆ pH
- ◆ Temperature
- ◆ 2,3-BPG
- ◆ A rise in temperature or  $\uparrow$  in  $\text{H}^+$  ions shifts the curve to right, since pH of blood falls as its  $\text{CO}_2$  content increases, it also causes a shift towards right.
- ◆ This is so because the deoxygenated blood binds  $\text{H}^+$  ions more avidly than the oxygenated form.
- ◆ The decrease in  $\text{O}_2$  affinity of hemoglobin, when pH falls is called as the **Bohr effect**.
- ◆ Also, since deoxygenated hemoglobin binds more  $\text{H}^+$  ions than oxygenated Hb does and forms carbamino compounds more readily, binding of  $\text{O}_2$  to hemoglobin reduces its affinity for  $\text{CO}_2$  - This is called **Haldane effect**.
- ◆ In sickle cell anemia, the Hb gets polymerized at a lower  $\text{O}_2$  tension and causes sickling of cells and blocking of small arterioles, both of these processes affect the saturation of Hb, shifting the curve to right. Since also, because of the blocking of arterioles, emergencies like acute abdomen due to ischemia of the intestinal loops can result.

**441. (a) Hyperventilation (b) Decreased PaCO<sub>2</sub>.**

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### (c) Pulmonary edema.

Ref: (Ganong 22<sup>nd</sup> Ed/Pg 686).

- ◆ Acclimatization is a process by which a person becomes adapted to the surrounding environment due to adjustments in the physiology, which are needed for survival.
- ◆ Acclimatization to altitude is due to operation of variety of compensatory mechanisms, mainly:-
  - Respiratory alkalosis caused by hyperventilation, which is due to decrease in the fraction of oxygen that is inhaled, which is responsible for decreased PaCO<sub>2</sub>
  - Increase in the red blood cell 2,3-BPG, which tends to decrease the O<sub>2</sub> affinity of hemoglobin.
- ◆ Hyperventilation steadily increases over the next 4 days because of active transport of H<sup>+</sup> in CSF or due to production of lactic acidosis in the brain. This increases the response to hypoxia. After 4 days the ventilatory response declines slowly.
- ◆ The increase in red blood cells triggered by the erythropoietin begins in 2-3 days and is sustained as long as the individual remains at high altitude.
- ◆ High altitude pulmonary edema is a complication of the mountain sickness.

### 442. (b) lung failure

#### (c) drowning

#### (d) Ventilatory failure.

Ref: (Ganong 22<sup>nd</sup> Ed/ Pg 686, 690, 692)

- Lung failure indicates a gas exchange failure
  - eg- pulmonary fibrosis.
  - Ventilation-perfusion imbalance.
- Ventilatory failure (pump failure) may be due to
  - Fatigue
  - Mechanical defects
  - Depression of respiratory controller of brain.
- Drowning is asphyxia caused by immersion usually in water.
  - In 10% of cases, due to the last effort of not to breathe, triggers laryngospasm and water does not enter lungs.
  - In rest the glottic muscles eventually relax and water enters. Now depending on whether it is fresh water or Ocean water the movement of fluid in lungs is decided, as is also the lysis of red blood cells in plasma in case of fresh water drowning.
  - In cases that are saved, resuscitation is the immediate protocol but subsequently due to sequelae of edema and inflammation there are areas of ventilation perfusion irregularities.
  - Thus all the three conditions result in carbon dioxide retention.
  - In carbon monoxide poisoning-

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- Hb has 210 times affinity for CO than for O<sub>2</sub>
  - Cherry red discolouration of COHb visible in skin, nails beds & mucous membranes.
  - The dissociation curve of remaining HbO<sub>2</sub> shifts to left, decreasing the amount of O<sub>2</sub> released.
  - Since there is no pathology affecting the gas exchange at the lungs there is no retention of CO<sub>2</sub>.
  - Arterial PO<sub>2</sub> remains normal and the carotid and aortic chemoreceptors are not stimulated.
- o At high altitude there is hyperventilation that causes respiratory alkalosis and decreased PaCO<sub>2</sub>.

**443. (b) ↓ ed Barometric pressure.**

**(d) ↓ ed PaO<sub>2</sub>**

**(e) ↓ ed pH**

*Ref: (Ganong 22<sup>nd</sup> Ed/Pg 686). Guyton 11<sup>th</sup> Ed/Pg 538, 539, table 43-1) (Ganong 22<sup>nd</sup> Ed/Pg 686)*

- o It takes almost four days for the respiration to steadily decrease towards the actual respiration that occurs at sea level, that too complete normalization takes residence at high altitude for many years.
- o This mountaineer ascended in 2 days and hence he did not give enough time to get acclimatized that too without supplemental oxygen.
- o Some of the important acute effects of hypoxia in the unacclimatized person breathing air, beginning at an altitude of 12,000 ft are drowsiness, lassitude, mental and muscle fatigue, sometimes headache. Nausea and sometimes euphoria. These effects progress to a stage of twitching or seizures above 18,000 feet and, end above 23,000 feet in the unacclimatized person, in coma and then followed by death.

ALTITUDE (FEET)	BAROMETRIC PRESSURE (mm Hg)	PO <sub>2</sub> IN AIR (mm Hg)	ARTERIAL OXYGEN SATURATION (%)
0	760	159	97
10,000	523	110	90
20,000	349	73	3

- o Respiratory alkalosis is produced due to hyperventilation AND decreased oxygen content of the inhaled oxygen.

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### 444. (a) Cyanide.

Ref: (Ganong 22<sup>nd</sup> Ed/Pg. 675)

- ◆ Peripheral receptors are located in the carotid bodies and aortic bodies called the glomus, containing islands of two types of cells. Type I and type II glomus cells.
- ◆ The type I glomus cells have O<sub>2</sub> sensitive K<sup>+</sup> channels, i.e. the K<sup>+</sup> efflux is reduced in presence of hypoxia, depolarizing the cells and causing Ca<sup>+2</sup> influx which triggers action potentials and transmitter release.
  - The afferents from carotid body are carried in glossopharyngeal nerves.
  - The afferents from aortic body carried within vagi.
- ◆ 2 mg carotid body is subject to a blood flow of 2000 ml/100g/min compared with a blood flow per 100 g/min of 54ml in brain and 420 ml in kidney.
- ◆ Thus receptors are satisfied with the dissolved arterial PO<sub>2</sub> itself, and thus are unaffected by conditions as anemia and carbon monoxide poisoning.
- ◆ Powerful stimulation is also produced by cyanide, which prevents O<sub>2</sub> utilization at the tissue level.

### 445. (d) Irregular and gasping.

Ref: (Ganong 21<sup>st</sup> Ed/ Pg 675)

- " The main components of the RESPIRATORY CONTROL PATTERN GENERATOR responsible for automatic respiration are located in the medulla, since spontaneous respiration continues, albeit somewhat irregular and gasping after transection of the brain stem at the inferior border of the pons."
- Rhythmic respiration is initiated by a small group of synaptically coupled pacemaker cells in Pre-Botzinger complex on either side of the medulla between the nucleus ambiguus and lateral reticular nucleus.
- These neurons discharge rhythmically and also cause rhythmic discharges in phrenic nerve motor neurons and are abolished by sections between the Pre-Botzinger complex and these motor neurons.
- They also contact the hypoglossal nuclei and the tongue is involved in the regulation of airway resistance.

### 446. (b) Lungs

Ref: (Ganong 22<sup>nd</sup> Ed/ Pg 674)

- As the O<sub>2</sub>- sensitive K<sup>+</sup> channels in the type I glomus cells that reduces K<sup>+</sup> efflux due to Hypoxia, the same O<sub>2</sub>- sensitive K<sup>+</sup>

channels cause vasoconstriction of the pulmonary arteries caused by hypoxia.

- o The systemic arteries contain ATP- dependant K<sup>+</sup> channels that permit more K<sup>+</sup> efflux with hypoxia and consequently cause vasodilatation instead of vasoconstriction.
- o Causes other than Hypoxia include :-
  - Inhalation of Cocaine.
  - Dexfenfluramine & related appetites suppressing drugs that increase extracellular serotonin.
  - Systemic lupus erythematosus.
- o Treatment with vasodialators as prostacyclin and prostacyclin analogues is effective, and had to be administered by continuous interavenous infusion,
- o Aerosolized preparations appear to be effective.

**447. (a) Bicarbonate**

**(b) Carbamino compounds**

**(c) Dissolved form**

Ref: (Ganong 22<sup>nd</sup> Ed/Pg 670, Table 35-2)

o **Fate of CO<sub>2</sub> in blood:-**

◆ In plasma :-

- 1. Dissolved
- 2. Formation of Carbamino compounds with plasma protein.
- 3. Hydration, H<sup>+</sup> buffered, HCO<sub>3</sub><sup>-</sup> in plasma

◆ In red blood cells:-

- 1. Dissolved
- 2. Formation of Carbamino –Hb
- Hydration, H<sup>+</sup> buffered, 70% of HCO<sub>3</sub><sup>-</sup> enters the plasma.
- Cl<sup>-</sup> shifts into cells, mosm in cells increases.

Of approximately 49 ml of CO<sub>2</sub> in blood in each deciliter of arterial blood:

- o 2.6ml –dissolved
- o 2.6ml –in carbamino compounds
- o 43.8ml-in HCO<sub>3</sub><sup>-</sup>.

**448. (c) Serotonin metabolism**

Ref: (Ganong 22<sup>nd</sup> Ed/Pg 665 Table 34-6)

- o Biologically active substances metabolized by the lungs.
- o Synthesized and used in lungs.
  - Surfactant
- o Synthesized or stored and released into blood.
  - Prostaglandin
  - Histamine
  - Kallikrein

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- o Partially removed from the blood
  - Prostaglandin
  - Bradykinin
  - Adenine nucleotides
  - Serotonin
  - Norepinephrine
  - Acetylcholine
- o Activated in the lungs
  - Angiotensin I → angiotensin II.
  - ◆ Large amounts of angiotensin-converting enzyme responsible for this activation are located on the surface of the endothelial cells of the pulmonary capillaries.
  - ◆ This reaction occurs in other tissues as well, but it is particularly prominent in lungs.
  - ◆ Removal of serotonin and norepinephrine reduces the amounts of these vasoactive substances reaching the blood circulations.

### 449. (c) joint proprioception receptors.

*Ref: (Ganong 22<sup>nd</sup> Ed/Pg 681)*

- ◆ Ventilation increases abruptly with the onset of exercise followed after a brief pause by a further, more gradual increase.
- ◆ With moderate exercise there is increase, due mostly to an increase in the depth of respiration also accompanied by increased rate of respiration when exercise is more strenuous.
- ◆ The PO<sub>2</sub> of blood falls from 40 to 25 mm Hg thus increasing the O<sub>2</sub> gradients
- ◆ The rate of blood flow is increased from 5.5 L/min to 20-35 L/min.
- ◆ The total amount entering the blood increases from 250ml/min to 4000ml/min.
- ◆ CO<sub>2</sub> excretion increases from 200ml/min to as much as 8000ml/min.
- ◆ The abrupt increase at the start of the exercise is presumably due to psychic stimuli and afferent impulses from the **proprioceptors in muscles, Tendons and joints.**

### 450. (b) Apex of lung

*Ref: (Ganong 22<sup>nd</sup> Ed/ Pg.662)*

- ◆ Gravity has a marked effect on the pulmonary circulation.
- ◆ In the upright position, the upper portions of the lungs are well above the level of the heart and the bases are at or below it.
- ◆ Consequently in the upper part of the lungs, the blood flow is less, the alveoli are larger, and ventilation is less than at the base.
- ◆ The pressure in the capillaries at the top of the lungs is close to the atmospheric pressure in the alveoli.

- ◆ Pulmonary arterial pressure is normally just sufficient to maintain perfusion, but if it is reduced or if alveolar pressure is increased, some of the capillaries collapse.
- ◆ It is said that the high ventilation/perfusion ratios at the apices account for the predilection of tuberculosis for this area because the relatively high alveolar  $PO_2$  that results, and provides a favourable environment for the growth of the tuberculosis bacteria.

451. (b) CO

*Ref: (Ganong 22<sup>nd</sup> Ed/ Pg.661)*

- ◆ CO is taken up by the Hb in the red blood cells at such a high rate that the partial pressure of CO in the capillaries stays very low and equilibrium is not reached in the 0.75 s, the blood takes to traverse the pulmonary capillaries at rest. Therefore, the transfer of **CO** is not limited by perfusion at rest and instead **is diffusion-limited**.
- ◆ The diffusing capacity for a given gas is directly proportionate to the surface area of the alveolo-capillary membrane and inversely proportionate to its thickness.
- ◆ The diffusing capacity for CO ( $DL_{CO}$ ) is measured as an index of diffusing capacity because its uptake is diffusion-limited.

452. (c) & (d)

*Ref: (Ganong 22<sup>nd</sup> Ed/ Pg.655)*

- ◆ The low surface tension when the alveoli are small is due to the presence, in the fluid lining the alveoli, of surfactant.
- ◆ It has been calculated that if it were not present, the unopposed surface tension in the alveoli would produce a 20mm Hg force favouring transudation of fluid from the blood in the alveoli.
- ◆ The surface tension is inversely proportional to the concentration of surfactant molecules per unit area. They move farther apart during inspiration and surface tension increases, it decreases when they move closer during expiration.