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20th Edition of Harrison's Principles of Internal Medicine

MCQs
for
Medical
Professionals



Prof. Ajay Mathur

www.drajaymathur.com

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CARDIOLOGY

Dyspnea

1. Which of the following about dyspnea is false?

Harrison's 20th Ed. Chapter 33 Page 226

- A. Self-reported
- B. Subjective experience of breathing discomfort
- C. Qualitatively distinct sensations that vary in intensity
- D. None of the above

Dyspnea is a symptom. American Thoracic Society (ATS) defines dyspnea as a "subjective experience of breathing discomfort that consists of qualitatively distinct sensations that vary in intensity. Dyspnea can be perceived only by the person experiencing it and, therefore, must be self-reported.

2. Which of the following features of breathing define dyspnea?

S Afr Med J. 2016;106(1):32-36

- A. Abnormal
- B. Uncomfortable
- C. Awareness
- D. All of the above

Normally, at rest, one is unaware of the act of breathing. With exercise, though aware of breathing, discomfort is expected to be transient. Dyspnea is defined with prefixes before awareness of breathing i.e. abnormally uncomfortable.

3. Which of the following is a sign of increased work of breathing?

Harrison's 20th Ed. Chapter 33 Page 226

- A. Tachypnea
- B. Use of accessory muscles of respiration
- C. Intercostal retraction
- D. All of the above

4. Which of the following can cause dyspnea?

Harrison's 20th Ed. Chapter 33 Page 226

- A. Pulmonary diseases
- B. Cardiac diseases
- C. Neurologic diseases
- D. All of the above

Dyspnea, like hunger or thirst, is a "synthetic sensation" as it often arises from multiple sources of information rather than from stimulation of a single neural receptor.

5. Which of the following best relates to dyspnea?

Harrison's 20th Ed. Chapter 33 Page 226

- A. Efferent afferent mismatch
- B. Efferent reafferent mismatch
- C. Afferent reefferent mismatch
- D. All of the above

Three main components contribute to dyspnea - afferent signals, efferent signals and central information processing. Dyspnea arises when there is a mismatch in integrative signaling between afferent signals from the respiratory system and efferent signals from the CNS. This is termed as "neuro-mechanical" or "efferent-reafferent mismatch".

6. Neural signal termed "corollary discharge" is sent to?

Harrison's 20th Ed. Chapter 33 Page 226

- A. Sensory cortex
- B. Motor cortex
- C. Ventilatory muscles
- D. All of the above

The sensory cortex is simultaneously activated when motor signals are sent to the chest wall, resulting in the conscious sensation of muscular effort and breathlessness. When there is increased work of breathing, increased neural output from the motor cortex is sensed via a neural signal, termed corollary discharge, is sent to the sensory cortex at the same time that motor output is directed to the ventilatory muscles. Corollary discharges are important in shaping the sense of respiratory effort.

Chemoreceptors in the carotid bodies and medulla are activated by?

Harrison's 20th Ed. Chapter 33 Page 226

- A. Hypoxemia
- B. Acute hypercapnia
- C. Acidemia
- D. All of the above

Chemoreceptors in the carotid bodies and medulla are activated by hypoxemia, acute hypercapnia, and acidemia.

8. The sense of air hunger arises from?

Harrison's 20th Ed. Chapter 33 Page 226

- A. Stimulation of chemoreceptors
- B. J-receptors
- C. Pulmonary vascular receptors
- D. All of the above

Sensation of air hunger arises from a combination of stimuli that increase drive to breathe such as hypoxemia or hypercapnia (mediated by signals from chemoreceptors in the carotid body and aortic arch), acute hypercapnia or acidemia (mediated by signals from peripheral & central chemoreceptors), airway & interstitial inflammation (mediated by pulmonary afferents), and pulmonary vascular receptors.

J (juxtacapillary) receptors are found in?

N Engl J Med. 2008;358:1054-9

- A. Bronchi
- B. Terminal brochiole
- C. Alveolar interstitial space
- D. All of the above

Pulmonary "J Receptors" are sensory cells located within alveolar septa and are "juxtaposed" to the pulmonary capillaries. These cells are activated by physical engorgement of the pulmonary capillaries (left heart dysfunction) or increased pulmonary interstitial volume (pulmonary edema). Dyspnea results from transudation of pulmonary fluid & stretching of pulmonary interstitial juxtacapillary receptors due to combination of increased pulmonary arterial blood flow in concert with elevated pulmonary venous pressures.

10. Irritant receptors around the epithelial cells of bronchial walls are activated by?

Am J Respir Crit Care Med. 1999;159:321-340

- Tactile stimulation in the bronchial mucosa
- B. High rates of air flow
- C. Increases in bronchial smooth muscle tone
- D. All of the above

Pulmonary irritant receptors are sensors present within the respiratory epithelium which can sense and respond to a variety of chemical irritants.

11. J-receptors are sensitive to?

Harrison's 19th Ed. 47e-1

- A. Changes in pulmonary artery pressure
- B. Acidemia
- C. Interstitial edema
- D. Hypercapnia

J-receptors or pulmonary C-fiber receptors are sensitive to interstitial edema.

12. The sensation of chest tightness results from?

Harrison's 20th Ed. Chapter 33 Page 226

- A. Chemoreceptors in medulla
- B. Mechanoreceptors in lungs
- C. Chemoreceptors in aortic and carotid bodies
- D. Afferent fibers in the phrenic nerves

Mechanoreceptors in the lungs, when stimulated by bronchospasm, lead to a sensation of chest tightness.

13. Metaboreceptors are located in?

Harrison's 20th Ed. Chapter 33 Page 226, J Physiol. 2007;585(Pt 1):165-174

- A. Medulla
- B. Carotid bodies
- C. Skeletal muscle
- D. All of the above

Metaboreceptors are located in skeletal muscle. During exercise, accumulation of metabolites within the active skeletal muscle, stimulates group III & IV afferent neurons which evoke a reflex increase in muscle sympathetic nerve activity (MSNA), known as the muscle metaboreflex.

14. Which of the following is an ischemic metabolite?

J Physiol. 2007;585(Pt 1):165-174

- A. Lactic acid
- B. Adenosine
- C. Bradykinin
- D. All of the above

Muscle metaboreceptors regulate sympathetic activation during exercise. This reflex is activated by lactic acid, phosphate, K+, H+, adenosine, prostaglandins, and bradykinin released from exercising skeletal muscle. These ischemic metabolites stimulate groups III and IV chemosensitive afferents.

15. Which of the following is used to measure dyspnea?

Am J Respir Crit Care Med. 1999;159:321-340

- A. Medical Research Council (MRC) Scale
- B. Baseline dyspnea index
- C. Oxygen Cost Diagram (OCD) scale
- D. All of the above

16. Modified Medical Research Council Dyspnea Scale classifies dyspnea into how many grades?

Harrison's 20th Ed. Chapter 33 Page 228 Table 33-1

- A. 3
- B. 4
- C. 5
- D. 6

Modified Medical Research Council Dyspnea Scale classifies dyspnea into 5 grades i.e. 0 to 4. This scale has been incorporated into GOLD 2017 guidelines as a tool for rating dyspnea in COPD.

17. What is the grade of dyspnea if one has to stop to rest after walking 100 meters on level ground?

Harrison's 20th Ed. Chapter 33 Page 228 Table 33-1

- A. ´
- B. 2
- C. 3
- D. 4

18. Chronic dyspnea is defined as symptoms lasting longer than?

Harrison's 20th Ed. Chapter 33 Page 226

- A. 1 month
- B. 3 months

- C. 6 months
- D. 12 months

Chronic dyspnea is defined as symptoms lasting longer than 1 month.

19. What proportion of patients have multifactorial reasons underlying dyspnea?

Harrison's 20th Ed. Chapter 33 Page 226

- A. 10%
- B. 25%
- C. 33%
- D. 50%

As many as one-third of patients may have multifactorial reasons underlying dyspnea.

20. Breathing discomfort during exercise in anemia is due to stimulation of?

Harrison's 20th Ed. Chapter 33 Page 228 Table 33-2

- A. Chemoreceptors
- B. Mechanoreceptors
- C. Metaboreceptors
- D. All of the above

21. Increased work of breathing (WOB) is the possible mechanism underlying dyspnea in?

Harrison's 20th Ed. Chapter 33 Page 228 Table 33-2

- A. COPD
- B. Interstitial lung disease
- C. Kyphoscoliosis
- D. All of the above

22. Orthopnea is seen in?

Harrison's 20th Ed. Chapter 33 Page 227

- A. Congestive heart failure
- B. Asthma triggered by esophageal reflux
- C. Mechanical impairment of diaphragm in obesity
- D. All of the above

23. "Nocturnal dyspnea" is a feature of which of the following?

Harrison's 20th Ed. Chapter 33 Page 227

- A. Chronic heart failure
- B. Myocardial ischemia
- C. Interstitial lung disease
- D. COPD

CHF or asthma cause nocturnal dyspnea. Circadian variations increase bronchial sensitivity between 2 AM & 4 AM in asthma patients leading to episodes of nocturnal dyspnea.

24. Which of the following is most characteristic feature of severe paroxysmal dyspnea of left ventricular failure?

N Engl J Med. 2010; 363:1464-1469

- A. Nocturnal episodes
- B. Sudden and unexpected
- C. Orthopnea
- D. All of the above

Nocturnal episodes of dyspnea are a typical feature of left ventricular failure. Sudden & unexpected dyspneic episodes at rest is more typical of pulmonary embolization, spontaneous pneumothorax, anxiety. Orthopnea is characteristic of congestive heart failure. The differential diagnosis of orthopnea and paroxysmal nocturnal dyspnea includes ischemic heart disease, nonischemic cardiomyopathies, valvular heart disease, constrictive pericarditis, and infiltrative cardiomyopathies.

25. Acute intermittent episodes of dyspnea are due to?

Harrison's 20th Ed. Chapter 33 Page 227

- A. Myocardial ischemia
- B. Bronchospasm
- C. Pulmonary embolism
- D. All of the above

Acute intermittent episodes of dyspnea are more likely to reflect episodes of myocardial ischemia, bronchospasm or pulmonary embolism.

26. Chronic persistent dyspnea is typical of?

Harrison's 20th Ed. Chapter 33 Page 227

- A. COPD
- B. Interstitial lung disease
- C. Chronic thromboembolic disease
- D. All of the above

Chronic persistent dyspnea is typical of COPD, interstitial lung disease and chronic thromboembolic disease.

27. Platypnea is dyspnea that occurs in which position?

Harrison's 20th Ed. Chapter 33 Page 228

- A. Upright
- B. Sitting
- C. Supine
- D. Lateral

Platypnea is dyspnea that occurs only in upright position with relief in supine position.

28. Platypnea is a feature of?

Harrison's 20th Ed. Chapter 33 Page 227

- A. Hepatopulmonary syndrome
- B. Emphysema
- C. Ankylosing spondylitis
- D. Psychogenic

29. Platypnea is a feature of?

Harrison's 20th Ed. Chapter 33 Page 227

- A. Obesity
- B. Left atrial myxoma
- C. Interstitial lung disease
- D. Chronic thromboembolic disease

Left atrial myxoma and hepatopulmonary syndrome are the causes of platypnea.

30. Trepopnea most often occurs in patients with?

N Engl J Med. 1970; 283:266

- A. Asthma
- B. COPD
- C. Heart disease
- D. Pleural effusion

Trepopnea is dyspnea that occurs only in a lateral decubitus position, most often in patients with heart disease due to positional alterations in ventilation-perfusion relationships. Most cardiac patients seem to prefer sleeping on the right side. The reasons given for this preference include "awareness of the heart" and dyspnea. The feature common to most patients seems to be cardiomegaly, and the mechanism of the dyspnea is thought to be positional compression of pulmonary veins. Perhaps trepopnea explains why unilateral pulmonary edema and unilateral pleural effusions, when they occur in these patients, are almost always just on the right.

31. Which of the following is called "cardiac asthma"?

Harrison's 20th Ed. Chapter 252 Page 1766

- A. Paroxysmal nocturnal dyspnea (PND)
- B. Orthopnea
- C. Platypnea
- D. Trepopnea

PND is also called cardiac asthma. During night, with recumbency, total blood volume is increased due to fluid mobilization from edematous areas leading to pulmonary congestion.

32. Presence of resting hypoxemia suggests?

Harrison's 20th Ed. Chapter 33 Page 228

- A. Ventilation-perfusion mismatch
- B. Shunt
- C. Impairment in diffusion capacity
- D. All of the above

33. Which of the following indicate increased work of breathing?

Harrison's 20th Ed. Chapter 33 Page 228

- A. Supraclavicular retractions
- B. Use of accessory muscles of ventilation
- C. Tripod position
- D. All of the above

Increased work of breathing is evidenced by supraclavicular retractions, use of accessory muscles of ventilation and tripod position (sitting with hands braced on knees). It is indicative of increased airway resistance or stiffness of the lungs & chest wall.

Paradoxical movement of the abdomen during breathing is suggestive of?

Harrison's 20th Ed. Chapter 33 Page 229

- A. Abdominal hernia
- B. Diaphragmatic weakness
- C. Pulmonary edema
- D. Acute asthma

35. Rounding of the abdomen during exhalation is suggestive of?

Harrison's 20th Ed. Chapter 33 Page 229

- Abdominal hernia
- B. Diaphragmatic weakness
- C. Pulmonary edema
- D. Acute asthma

Rounding of the abdomen during exhalation is suggestive of pulmonary edema.

36. Low lung volumes on a chest radiograph suggests?

Harrison's 20th Ed. Chapter 33 Page 229

- A. Interstitial edema or fibrosis
- B. Diaphragmatic dysfunction
- C. Impaired chest wall motion
- D. All of the above

37. On a chest radiograph, prominent pulmonary vasculature in the upper lung zones indicates?

Harrison's 20th Ed. Chapter 33 Page 229

- A. Pulmonary arterial hypertension
- B. Pulmonary venous hypertension
- C. Pulmonary embolism
- D. Any of the above

38. On a chest radiograph, enlarged central pulmonary arteries indicates?

Harrison's 20th Ed. Chapter 33 Page 229

- A. Pulmonary arterial hypertension
- B. Pulmonary venous hypertension
- C. Pulmonary embolism
- D. Any of the above

39. In cardiopulmonary exercise test (CPET), which of the following suggests respiratory system as the cause of dyspnea?

Harrison's 20th Ed. Chapter 33 Page 229

- A. Patient achieves predicted maximal ventilation
- B. Demonstrates an increase in dead space or hypoxemia
- C. Develops bronchospasm
- D. All of the above

40. In cardiopulmonary exercise test (CPET), which of the following suggests cardiovascular system as the cause of dyspnea?

Harrison's 20th Ed. Chapter 33 Page 229

- A. If the heart rate is >85% of the predicted maximum
- B. If the anaerobic threshold occurs early
- C. If the BP is excessively high or decreases during exercise
- D. All of the above

At peak exercise in CPET, if heart rate is >85% of the predicted maximum, if anaerobic threshold occurs early, if BP becomes excessively high or decreases during exercise, if O₂ pulse falls, or if there are ischemic changes on ECG, an abnormality of cardiovascular system is likely cause of dyspnea.

41. Cardiopulmonary exercise testing (CPET) provides assessment of the integrative exercise responses involving?

Postgrad Med J. 2007;83(985):675-682

- A. Pulmonary
- B. Cardiovascular
- C. Skeletal muscle systems
- D. All of the above

Cardiopulmonary exercise testing (CPET) provides assessment of the integrative exercise responses involving the pulmonary, cardiovascular, hematopoietic, neuropsychological, and skeletal muscle systems, which are not adequately reflected through the measurement of individual organ system function

42. Exercise tolerance is determined by?

Postgrad Med J. 2007;83(985):675-682

- Pulmonary gas exchange
- B. Cardiovascular performance
- C. Skeletal muscle metabolism
- D. All of the above

Peak exercise capacity is defined as "the maximum ability of the cardiovascular system to deliver oxygen to exercising skeletal muscle and of the exercising muscle to extract oxygen from the blood".

3. Oxygen uptake (VO₂) is calculated by?

Postgrad Med J. 2007;83(985):675-682

- A. Bernoulli equation
- 3. Fick equation
- C. Friedewald equation
- D. Henderson-Hasselbalch equation

44. One metabolic equivalent (MET) is the resting oxygen uptake in a sitting position and equals?

Postgrad Med J. 2007;83(985):675-682

- A. 1.5 mL/kg/minute
- B. 2.5 mL/kg/minute
- C. 3.5 mL/kg/minute
- D. 4.5 mL/kg/minute

Oxygen uptake (VO₂) equals cardiac output times the arterial minus mixed venous oxygen content or $VO_2 = (SV \times HR) \times (CaO_2 - CvO_2)$, where SV is the stroke volume, HR is the heart rate, CaO_2 is the arterial oxygen content, and CvO_2 is the mixed venous oxygen content.

45. In dyspnea, supplemental O₃ should be administered if?

Harrison's 20th Ed. Chapter 33 Page 230

- A. Resting O₂ saturation is ≤ 88%
- B. If patient's saturation drops to ≤ 88% with activity
- C. If patient's saturation drops to ≤ 88% with sleep
- D. All of the above

46. Which of the following may have a role in managing dyspnea?

Harrison's 20th Ed. Chapter 33 Page 230

- A. Opioids
- B. Anxiolytics
- C. Inhaled furosemide
- D. All of the above

Cardiology

Hypoxia and Cyanosis

47. Pasteur's effect relates to?

Harrison's 20th Ed. Chapter 36 Page 234

- A. Switch from aerobic to anaerobic metabolism
- B. Abnormal hemoglobin derivative
- C. Pulmonary arteriovenous fistulae
- D. Flow rate in vessels

Pasteur's effect refers to switch from aerobic to anaerobic metabolism.

48. Severe hypoxia with inadequate production of ATP leads to?

Harrison's 20th Ed. Chapter 36 Page 234

- A. Uncontrolled Ca++ influx in cells
- B. Activation of Ca++ dependent phospholipases
- C. Activation of Ca++ dependent proteases
- D. All of the above

49. Which of the following gene is upregulated in adaptation to hypoxia?

Harrison's 20th Ed. Chapter 36 Page 234

- A. Phosphoglycerate kinase
- B. Phosphofructokinase
- C. Glucose transporters Glut-1 and Glut-2
- D. All of the above

Adaptations to hypoxia are mediated by upregulation of genes encoding glycolytic enzymes like phosphoglycerate kinase & phosphofructokinase & glucose transporters Glut-1 & Glut-2, and by growth factors like vascular endothelial growth factor (VEGF) & erythropoietin (EPO).

50. During hypoxia systemic arterioles dilate by opening of?

Harrison's 20th Ed. Chapter 36 Page 234

- A. NaATP channels in vascular smooth-muscle cells
- B. KATP channels in vascular smooth-muscle cells
- C. CIATP channels in vascular smooth-muscle cells
- D. All of the above

In hypoxia systemic arterioles dilate by opening of KATP channels in vascular smooth-muscle cells.

51. During hypoxia, pulmonary vascular smooth-muscle cells contract due to inhibition of?

Harrison's 20th Ed. Chapter 36 Page 234

- A. Na⁺ channels
- B. K+ channels
- C. Cl-channels
- D. All of the above

In pulmonary vascular smooth-muscle cells, inhibition of K⁺ channels causing contraction.

52. Acute hypoxia causes a clinical picture resembling?

Harrison's 20th Ed. Chapter 36 Page 235

- A. Partial seizure
- B. Peripheral neuropathy
- C. Acute alcoholism
- D. Migraine

Clinically, acute hypoxia resembles acute alcoholism (impaired judgment, motor incoordination).

53. Headache in high-altitude illness is caused by?

Harrison's 20th Ed. Chapter 36 Page 235

- A. Cerebral vasodilation
- B. Pulmonary arterial constriction
- C. Pulmonary venous constriction
- D. All of the above

High-altitude illness is characterized by headache secondary to cerebral vasodilation, gastrointestinal symptoms, dizziness, insomnia, fatigue or somnolence. High-altitude cerebral edema (HACE) is manifest by severe headache and papilledema and can cause coma.

54. In severe hypoxia, death usually results from?

Harrison's 20th Ed. Chapter 36 Page 235

- A. Respiratory failure
- B. Cardiac arrhythmia
- C. Seizure
- D. Autonomic failure

In severe hypoxia, centers of brainstem are affected & death results from respiratory failure.

55. When hypoxia occurs consequent to respiratory failure, hemoglobin-oxygen dissociation curve is displaced to?

Harrison's 20th Ed. Chapter 36 Page 235

- A. Right
- B. Left
- C. Center
- D. Any of the above

When hypoxia occurs due to respiratory failure, PaO₂ declines, PaCO₂ rises & Hb-O₂ dissociation curve is displaced to right, with greater quantities of O₂ released at any level of tissue PO₂.

6. Most common cause of respiratory hypoxia is?

Harrison's 20th Ed. Chapter 36 Page 235

- A. Hypoventilation
- B. Ventilation-perfusion mismatch
- C. Intrapulmonary right-to-left shunting
- D. None of the above

Most common cause of respiratory hypoxia is ventilation-perfusion mismatch.

57. High-altitude illness develops when a person ascends rapidly to a height of?

Harrison's 20th Ed. Chapter 36 Page 235

- A. 1000 meters
- B. 2000 meters
- C. 3000 meters
- D. 4000 meters

High-altitude illness develops when a person ascends rapidly to a height of 3000 meters or ~10,000 feet. At this altitude, the reduction of O_2 content of inspired air (FIO₂) leads to a decrease in alveolar PO₂ to ~60 mm Hg.

58. In which of the following conditions, PaO_a cannot be restored to normal with inspiration of 100% O.?

Harrison's 20th Ed. Chapter 36 Page 235

- Tetralogy of Fallot (TOF)
- Transposition of great arteries (TGA) B.
- C. Eisenmenger's syndrome
- D All of the above

Hypoxia due to congenital cardiac malformations (TOF, TGA & Eisenmenger's syndrome) resembles intrapulmonary right-to-left shunting & PaO, cannot be restored to normal with 100% O,

59. In anemic hypoxia, the PaO, is?

Harrison's 20th Ed. Chapter 36 Page 235

- Α Normal
- R Decreased
- C. Increased
- D Any of the above

In anemic hypoxia, PaO is normal but due to reduction of Hb concentration, absolute quantity of O transported per unit volume of blood is diminished.

60. Which of the following is false about in anemic hypoxia?

Harrison's 20th Ed. Chapter 36 Page 235

- Absolute quantity of O₂ transported per unit volume of blood is diminished
- B. Usual quantity of O₂ is removed from blood in capillaries
- C. PO₂ & saturation in venous blood decline to a greater extent
- None of the above

In which of the following hypoxia's, venous blood tends to 61. have a high O₃?

Harrison's 20th Ed. Chapter 36 Page 235

- A. Excercise induced
- B. Circulatory hypoxia
- C. Cyanide poisoning
- Carbon monoxide intoxication

62. Example of "Histotoxic hypoxia" is?

Harrison's 20th Ed. Chapter 36 Page 235

- A. Severe exercise
- B. Cyanide poisoning
- Raynaud's phenomenon C.
- Π High altitude hypoxia

Cyanide causes cellular hypoxia because tissues are unable to utilize O₂. As a result, venous blood tends to have a high O, tension. This condition is called histotoxic hypoxia.

Special chemosensitive cells that determine respiratory 63. response to hypoxia are located in?

Harrison's 20th Ed. Chapter 36 Page 235

- Carotid bodies
- В. Aortic bodies
- Respiratory center in the brainstem
 - All of the above

Which of the following is a feature of chronic mountain sickness? Harrison's 20th Ed. Chapter 36 Page 236

- Reduced ventilation
- Cvanosis

- Right ventricular enlargement
- All of the above D

In persons with chronic hypoxemia secondary to prolonged residence at a high altitude (>13000 feet or 4200 meters), chronic mountain sickness develops. This is characterized by a blunted respiratory drive, reduced ventilation, erythrocytosis, cyanosis, weakness, right ventricular enlargement secondary to pulmonary hypertension and even stupor.

65. Cyanosis is usually most marked in?

Harrison's 20th Ed. Chapter 36 Page 236

- A. Nail beds
- B. Fars
- C. Malar eminences
- D All of the above

Cyanosis is usually most marked in the lips, nail beds, ears, and malar eminences.

The degree of cyanosis is modified by?

Harrison's 20th Ed. Chapter 36 Page 236

- Color of the cutaneous pigment
- Thickness of the skin
- State of the cutaneous capillaries
- All of the above

67. Cyanosis is apparent when the mean capillary concentration of reduced hemoglobin exceeds?

Harrison's 20th Ed. Chapter 36 Page 236

- 2 gram / dL
- 3 gram / dL
- 4 gram / dL
- D. 5 gram / dL

It is the absolute rather than relative quantity of reduced Hb that produces cyanosis. As concentration of total Hb is markedly reduced in severe anemia, absolute quantity of reduced Hb is still small and patients may not become cyanotic even with marked arterial desaturation.

Central and peripheral cyanosis may be present in?

Harrison's 20th Ed. Chapter 36 Page 236

- Α Shock
- Congestive failure
- C. Peripheral vascular disease
- Cardiogenic shock with pulmonary edema

69. Cyanosis occurs upon ascent to an altitude of?

Harrison's 20th Ed. Chapter 36 Page 236

- 2000 meters
- В. 3000 meters
- C. 4000 meters
- D. 5000 meters

Cyanosis is manifest in an ascent to 4000 m (13,000 ft). At this height, FIO₂ & alveolar PO₂ are about 85 & 50 mm Hg, respectively & SaO, is ~75% leaving more reduced Hb in arterial blood.

Cyanosis can be observed in all except?

Harrison's 20th Ed. Chapter 36 Page 237

- Α Marked polycythemia
- Carboxyhemoglobin (COHb) B.
- C. Methemoglobin
- Sulfhemoglobin

Patients with marked polycythemia become cyanotic at higher levels of SaO_a than patients with normal hematocrit values. Cyanosis is also observed when nonfunctional hemoglobin (methemoglobin or sulfhemoglobin) is present in blood.

71. Most common congenital cardiac lesion associated with cyanosis in adult is?

Harrison's 20th Ed. Chapter 234 Page 1667

- Tetralogy of Fallot
- B. Patent ductus arteriosus
- C. Ventricular septal defect
- D Atrial septal defect

Most common congenital cardiac lesion with cyanosis in the adult is tetralogy of Fallot.

72. Differential cyanosis is a feature of?

Harrison's 20th Ed. Chapter 234 Page 1667

- Tetralogy of Fallot
- Patent ductus arteriosus B.
- C. Ventricular septal defect
- D. Atrial septal defect

In patent ductus arteriosus, pulmonary hypertension and right-to-left shunt, differential cyanosis results, that is, cyanosis occurs in the lower but not in the upper extremities.

73. Which of the following is suspected when blood remains brown after mixing in test tube & exposed to air?

Harrison's 20th Ed. Chapter 94 Page 695

- Α. Marked polycythemia
- В. Carboxyhemoglobin (COHb)
- C. Methemoglobin
- D. Sulfhemoglobin

Diagnosis of methemoglobinemia is suspected if blood remains brown after mixing in a test tube & exposure to air. Spectroscopy confirms the diagnosis.

74. Acquired methemoglobinemia may occur on exposure to?

N Engl J Med. 2000;343:337

- A. Dapsone
- B. **Nitrates**
- C. Sulfonamides
- D All of the above

Acquired methemoglobinemia is a rare consequence of the exposure of normal red cells to oxidizing drugs such as benzocaine, dapsone, nitrates, primaquine and sulfonamides.

75. Symptomatic methemoglobinemia should be treated with?

N Engl J Med. 1992;327:1461

- Methylcobalamin
- B. Hexamethylene diisocyanate
- C. Methylene blue
- Toluene diisocyanate

Symptomatic methemoglobinemia should be treated with intravenous injection of methylene blue (1 to 2 mg per kilogram) or oral methylene blue (60 mg three to four times each day) or ascorbic acid (300 - 600 mg/day).

76. Which of the following is false in Eisenmenger syndrome?

Harrison's 20th Ed. Chapter 264 Page 1838

- A. Cyanosis
- Elevated pulmonary vascular resistance
- C. Intracardiac communication
- D. Pulmonic stenosis

Elevated pulmonary vascular resistance that produces cyanosis in the presence of intra- & extracardiac communications without pulmonic stenosis is termed Eisenmenger syndrome

In peripheral cyanosis of extremities, the arterial blood is? 77.

Harrison's 20th Ed. Chapter 36 Page 237

- Normally saturated with oxygen
- В. Over saturated with oxygen
- Under saturated with oxygen
- Any of the above

78. Clubbing without cyanosis is frequent in?

Harrison's 20th Ed. Chapter 36 Page 237

- Infective endocarditis
- B. Inflammatory bowel disease
- C. Jackhammer operators
- D. All of the above

Clubbing without cvanosis is frequent in infective endocarditis, inflammatory bowel disease & in Jackhammer operators.

ardiology

Edema

79. Edema is defined as a clinically apparent increase in?

Harrison's 20th Ed. Chapter 37 Page 237

- A. Intracellular fluid volume
- B. Plasma volume
- C. Interstitial fluid volume
- D. All of the above

Edema is defined as a clinically apparent increase in interstitial fluid volume.

80. Which of the following statements is false?

Harrison's 20th Ed. Chapter 37 Page 237

- A. ~ Two-thirds of total body water is intracellular
- B. ~ One-third of total body water is extracellular
- C. ~ One-fourth of extracellular water is in the plasma and the remainder comprises the interstitial fluid
- D. None of the above

81. Which of the following about movement of water & diffusible solutes from plasma to interstitium is false?

Harrison's 20th Ed. Chapter 37 Page 237

- A. Promoted by hydrostatic pressure within the capillaries
- B. Promoted by colloid oncotic pressure in the interstitial fluid
- C. Most prominent at the arterial origin of the capillary
- D. None of the above

82. Fluid is returned from the interstitial space into the vascular system largely through?

Harrison's 20th Ed. Chapter 37 Page 237

- A. Arterial system
- B. Venous system
- C. Lymphatic system
- D. All of the above

Fluid is returned from the interstitial space into the vascular system largely through the lymphatic system.

83. Development of edema occurs due to?

Harrison's 20th Ed. Chapter 37 Page 237

- A. Inadequate lymphatic drainage
- B. Damage to capillary endothelial barrier
- C. Increase in the oncotic pressure in interstitial space
- D. All of the above

Development of edema occurs due to an increase in intracapillary hydrostatic pressure, inadequate lymphatic drainage, reductions in oncotic pressure in plasma, damage to capillary endothelial barrier and increases in the oncotic pressure in interstitial space.

84. Which of the following is referred to as "tissue tension"?

Harrison's 19th Ed. 250

- A. Hydrostatic pressure within the vascular system
- B. Colloid oncotic pressure within the vascular system
- C. Hydrostatic pressure within the interstitial fluid
- D. All of the above

Plasma & interstitial fluid are two components of extracellular fluid regulate by Starling forces. Hydrostatic pressure within interstitial fluid is referred to as the tissue tension which promotes the movement of fluid into the vascular compartment.

85. Movement of water & diffusible solutes from vascular space into the interstitial space occurs at?

Harrison's 20th Ed. Chapter 37 Page 237

- A. Arteriolar end of capillaries
- B. Venous end of capillaries
- C. Lymphatics
- D. All of the above

Movement of water & diffusible solutes from vascular space into interstitial space occurs at the arteriolar end of capillaries. Fluid is returned from interstitial space into vascular system at the venous end of capillary & by way of lymphatics.

86. Which of the following about renal juxtaglomerular cells is false?

Harrison's 20th Ed. Chapter 37 Page 237

- A. Secrete renin
- B. Specialized myoepithelial cells
- C. Surround the afferent renal arteriole
- D. None of the above

87. Which of the following best define renin?

Harrison's 20th Ed. Chapter 37 Page 237

- A. Enzyme
- B. Pro-hormone
- C. Hormone
- D. Cofactor

Renin is an enzyme with a molecular weight of ~40,000 secreted by juxtaglomerular cells.

8. Conditions that reduce effective arterial blood volume cause constriction of which of the following?

Harrison's 20th Ed. Chapter 37 Page 237

- A. Renal afferent arteriolar constriction
- B. Renal efferent arteriolar constriction
- C. Renal glomerular capillary constriction
- D. All of the above

Heart failure, nephrotic syndrome & cirrhosis reduce effective arterial blood volume and cause renal efferent arteriolar constriction.

9. Which of the following stimulates renin release?

Harrison's 20th Ed. Chapter 37 Page 237

- A. Diminished stretch of the juxtaglomerular cells
- B. Low sodium chloride load in distal renal tubules
- C. Circulating catecholamines
- D. All of the above

Diminished renal blood flow resulting in diminished stretch of juxtaglomerular cells lowers sodium chloride load reaching distal renal tubules signals juxtaglomerular cells to secrete renin. Activation of β-adrenergic receptors in juxtaglomerular cells by sympathetic nervous system & circulating catecholamines stimulates renin release.

90. Angiotensinogen is synthesized by?

Harrison's 20th Ed. Chapter 37 Page 237

- A. Kidney
- B. Liver
- C. Pancreas
- D. Lung

Angiotensinogen, an $\alpha 2$ globulin, is synthesized by liver. Renin converts angiotensinogen to a decapeptide angiotensin I, which is broken down to an octapeptide angiotensin II.

91. Renal effects of Angiotensin II are mediated by activation of which type of Angiotensin II receptors?

Harrison's 20th Ed. Chapter 37 Page 238

- A. Type 1
- B. Type 2
- C. Type 3
- D. Type 4

Angiotensin II produces renal vasoconstriction & salt and water retention. These renal effects are mediated by activation of Angiotensin II type 1 receptors.

92. Which of the following stimulates the production of aldosterone?

Harrison's 20th Ed. Chapter 37 Page 238

- A. Angiotensinogen
- B. Angiotensin I
- C. Angiotensin II
- D. All of the above

93. Aldosterone is produced by?

Harrison's 20th Ed. Chapter 37 Page 238

- A. Juxtaglomerular cells of kidney
- B. Macula densa cells of kidney
- C. Zona glomerulosa of adrenal cortex
- Zona reticularis of adrenal cortex

Aldosterone is produced by zona glomerulosa of adrenal cortex & its release is stimulated by Angiotensin II. It is metabolized by liver.

94. Site of action of aldosterone is?

Harrison's 20th Ed. Chapter 37 Page 238

- A. Proximal convoluted tubule
- B. Loop of Henle
- C. Distal convoluted tubule
- D. Collecting tubule

95. Blockade of the action of aldosterone can be done by?

Harrison's 20th Ed. Chapter 37 Page 238

- A. Spironolactone
- B. Eplerenone
- C. Amiloride
- D. All of the above

96. In heart failure, aldosterone secretion is elevated due to?

Harrison's 20th Ed. Chapter 37 Page 238

- A. Prolongation of biologic half-life
- B. Increased secretion
- C. Reduced hepatic catabolism
- D. All of the above

In heart failure, levels of aldosterone are raised due to increased secretion, prolonged biologic half-life & reduced hepatic catabolism due to reduced hepatic blood flow, secondary to reduction in cardiac output.

Activation of Renin-Angiotensin-Aldosterone (RAA) system is most striking in which of the following?

Harrison's 20th Ed. Chapter 37 Page 238

- A. Acute, severe heart failure
- B. Chronic heart failure
- C. Stable heart failure
- D. Compensated heart failure

Activation of RAA system is seen conspicuously in early phase of acute, severe heart failure & is less intense in patients with chronic, stable, compensated heart failure.

98. Mineralocorticoid escape phenomenon is best explained by?

Harrison's 20th Ed. Chapter 37 Page 238

- A. Deficit in effective arterial blood volume
- B. Aldosterone antagonism
- C. Pressure natriuresis
- D. Blocking of epithelial sodium channels

Administration of potent mineralocorticoids (deoxycorticosterone acetate or fludrocortisone) leads to salt & water referition. This accumulation is self-limiting, despite continued exposure to steroid, a phenomenon known as mineralocorticoid escape wherein edema does not develop. It is due to an increase in GFR (pressure natriuresis).

99. Arginine vasopressin (AVP) is best related to?

Harrison's 20th Ed. Chapter 37 Page 238

- A. V1 receptors
- B. V2 receptors
- C. V3 receptors
- D. V4 receptors

Secretion of arginine vasopressin (AVP) occurs in response to increased intracellular osmolar concentration. By stimulating V2 receptors, AVP increases the reabsorption of free water in the distal tubules and collecting ducts of the kidneys, thereby increasing total-body water.

100. Which of the following is an effect of Endothelin-1?

Harrison's 20th Ed. Chapter 37 Page 238

- A. Renal vasoconstriction
- B. Sodium retention
- C. Edema
- D. All of the above

Endothelin-1 is a potent peptide vasoconstrictor released by endothelial cells. Its concentration in the plasma is elevated in patients with severe heart failure and contributes to renal vasoconstriction, sodium retention and edema.

101. Atrial natriuretic peptide (ANP) is stored in secretory granules within?

Harrison's 20th Ed. Chapter 37 Page 238

- A. Sinoatrial node
- B. Atrial myocytes
- C. Pulmonary veins
- D. All of the above

Polypeptide ANP is secreted by atrial myocytes secondary to atrial distention and/or sodium load. Its actions are excretion of sodium & water by increasing GFR, inhibiting sodium reabsorption in PCT & inhibiting release of renin & aldosterone. It also antagonizes vasoconstrictor actions of Angiotensin II, AVP & sympathetic stimulation causing arteriolar & venous dilatation.

102. Brain natriuretic peptide (BNP) is present in?

Harrison's 20th Ed. Chapter 37 Page 238

- A. Cardiac ventricular myocardium
- B. Cerebral cortex
- C. Cerebellum
- D. All of the above

BNP is stored in cardiac ventricular myocardium & is released when ventricular diastolic pressure rises. Its actions are similar to ANP. Circulating levels of ANP & BNP are elevated in CHF.

103. Released ANP & BNP bind to?

Harrison's 20th Ed. Chapter 37 Page 238

- A. Natriuretic receptor-A
- B. Natriuretic receptor-B
- C. Natriuretic receptor-C
- D. Natriuretic receptor-D

Released ANP & BNP bind to the natriuretic receptor-A.

104. Which of the following is an action of ANP and BNP?

Harrison's 20th Ed. Chapter 37 Page 238

- A. Excretion of sodium & water
- B. Inhibiting sodium reabsorption
- C. Inhibiting release of renin & aldosterone
- D. All of the above

Released ANP and BNP lead to excretion of sodium & water by augmenting glomerular filtration rate, inhibiting sodium reabsorption in proximal tubule and inhibiting release of renin & aldosterone.

105. ANP and BNP antagonize the vasoconstrictor actions of?

Harrison's 20th Ed. Chapter 37 Page 238

- A. Angiotensin II
- B. Arginine vasopressin
- C. Sympathetic stimulation
- D. All of the above

Released ANP and BNP also cause dilation of arterioles & venules by antagonizing the vasoconstrictor actions of All, AVP and sympathetic stimulation. Thus, elevated levels of natriuretic peptides have the capacity to oppose sodium retention in hypervolemic and edematous states.

106. Which of the following is a form of edema?

Harrison's 20th Ed. Chapter 37 Page 238

- A. Anasarca
- B. Ascites
- C. Hydrothorax
- D. All of the above

Anasarca refers to gross, generalized edema. Ascites and hydrothorax refer to accumulation of excess fluid in the peritoneal and pleural cavities, respectively and are considered special forms of edema.

107. Which of the following about edema of renal disease is false?

Harrison's 20th Ed. Chapter 37 Page 239

- A. Hypertension
- B. Without considerable cardiac enlargement
- C. Develop orthopnea
- D. Normal cardiac output

108. NSAIDs and cyclosporine cause edema due to?

Harrison's 20th Ed. Chapter 37 Page 239

- A. Renal vasoconstriction
- B. Arteriolar dilation

- C. Augmented renal sodium reabsorption
- D. Capillary damage

109. Refeeding edema best relates to?

Harrison's 20th Ed. Chapter 37 Page 239

- A. Calcium
- B. Insulin
- C. Paratharmone
- D. Oxytocin

Refeeding edema may be linked to increased release of insulin which directly increases tubular sodium reabsorption.

110. Which of the following is involved in the edema of starvation?

Harrison's 20th Ed. Chapter 37 Page 239

- A. Hyponatremia
- B. Hypokalemia
- C. Hypocalcemia
- D. Hypomagnesemia

In addition to hypoalbuminemia, hypokalemia and caloric deficits may be involved in the edema of starvation.

111. Edema of hypothyroidism best relates to?

Harrison's 20th Ed. Chapter 37 Page 240

- A. Glutamic acid
- B. Aggrecan
- C. Hyaluronic acid
- D. Lubricin

Nonpitting edema of hypothyroidism (myxedema) due to deposition of hyaluronic acid.

112. Causes of facial edema include?

Harrison's 20th Ed. Chapter 37 Page 240

- A. Trichinosis
- B. Hypoproteinemia
- C. Myxedema
- D. All of the above

Apart from hypoproteinemia, causes of facial edema include trichinosis (Trichinella spiralis), allergic reactions & myxedema. Trichinosis can also lead to membranous glomerulopathy, proliferative glomerulonephritis & acute pericarditis. Trichinosis is a cause of FUO.

113. Antihypertensive agents associated with edema formation include all except?

Harrison's 20th Ed. Chapter 37 Page 240 Table 37-2

- A. Minoxidil
- B. Hydralazine
- C. Clonidine
- D. Atenolol

114. Antihypertensive agents associated with edema formation include all except?

Harrison's 20th Ed. Chapter 37 Page 240 Table 37-2

- A. Methyldopa
- B. Calcium channel antagonists
- C. ACE Inhibitors
- D. Alpha adrenergic antagonists

Harrison's 20th Ed. Chapter 37 Page 240 Table 37-2

MCQs FOR MEDICAL PROFESSIONALS BY PROF. AJAY MATHUR

A. Glucocorticoids

all except?

115.

- B. Mineralocorticoids
- C. Anabolic steroids
- D. Estrogens / Progestins

116. Which of the following is associated with edema formation?

Steroid hormones associated with edema formation include

Harrison's 20th Ed. Chapter 37 Page 240 Table 37-2

- Cyclosporine
- В. Growth hormone
- C. Interleukin 2
- D. All of the above

Which of the following is false about idiopathic edema?

N Engl J Med. 1960; 263:1342-1345

- Occurs in women
- B. Edema unrelated to menstrual cycle
- Occurs after upright posture
- D. None of the above

Idiopathic cyclic edema occurs almost exclusively in women, is characterized by periodic episodes of edema (unrelated to menstrual cycle), frequently accompanied by abdominal distention orthostatic retention of sodium & water after upright posture.

Venous pressure in upper extremities is elevated in all except?

Harrison's 20th Ed. Chapter 37 Page 240

- Advanced heart failure
- B. Constrictive pericarditis
- C. Tricuspid stenosis
- Cirrhosis liver

Venous pressure in upper extremities is elevated in advanced heart failure, constrictive pericarditis or tricuspid stenosis but is normal in cirrhosis. In hepatic cirrhosis, JVP is normal.

Ajay Mathur works as Senior Professor of Medicine at SMS Medical College and Hospital, Jaipur, Rajasthan, India.

Gold Medal recipient at undergraduate and postgraduate levels, Dr Mathur has keen academic interests. This book is an outcome of his passion for medical science and research spanning over the last three decades.

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