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Based on 19th Edition of Harrison's Principles of Internal Medicine

MCQs for Medical Professionals

Prof. Ajay Mathur
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Foreword

As a professional, who has been practicing medicine for over four decades now, I appreciate the value this book brings to the table in times like today. As we move from a largely descriptive era to the bullet-point generation, this academic initiative appears even more relevant than its first three earlier editions.

Many refinements have been made in this book bearing in mind the reception it has received in the last few years. The book has been a reference point for many medical entrance examinations and has left an impact on medical professionals who look for high quality of academic material.

Harrison's Principles of Internal Medicine, published by The McGraw-Hill Companies, Inc. is an epic in the world of medical science. This book serves as a faithful companion to the epic by assisting the readers draw most out of it in the service of mankind.

Knowledge is a more processed form of information. Prof. Ajay Mathur stays true to his pledge by presenting well-digested bytes of knowledge across different fields of medicine. He relies on good old word-of-mouth to make this book a success rather than blitzkrieg marketing. I recommend that you make this a must-have without an iota of doubt.

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Past President, Indian Society of Gastroenterology (2008)

Preface

Medicine, in all its vastness, needs to be understood in a way that makes most sense to how it is applied today. Memorizing each word is elusive and therefore, testing knowledge of a discipline remains an evergreen challenge.

It is a widely accepted fact that taking a quiz soon after studying helps one retain information and knowledge better. The brain works in mysterious ways but a sure way of holding onto what the mind has already digested is to put lessons to test. Multiple Choice Questions are a quick and effective way of remembering the gist of the matter. This is precisely the reason why most examinations today follow this format. This book is committed to hone your skills for retaining knowledge; it is only axiomatic that excellence will follow when you acquire knowledge properly.

In its fourth edition, this book incorporates the recent advances in medicine as well as my personal insights on how to learn better. Based on earlier and the 19th edition of Harrison's Principles of Internal Medicine, published by The McGraw-Hill Companies, Inc., this book also comprises relevant studies from the leading medical journals from the world over.

This book caters to medical professionals at all levels. Not only can this be used by aspiring doctors to prepare for medical entrance examinations but by seasoned medical professionals to update knowledge long after it has been acquired. The book is sign-posted with resources and references should the reader require elaboration on any given topic.

Over ten thousand questions and still counting; I take it upon myself to continually refine the content of the book and chronicle the advances of medical science.

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The chapters and page numbers mentioned is the same as in 19th edition of Harrison's Principles of Internal Medicine.

Contents

Cardiology 2

Chapter 47e. Dyspnea

1. Neural signal termed "corollary discharge" is sent to ?
Harrison's 19th Ed. 47e-1

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

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.

2. Chemoreceptors in the carotid bodies and medulla are activated by ?
Harrison's 19th Ed. 47e-1

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.

3. The sense of air hunger arises from ?
Harrison's 19th Ed. 47e-1

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

4. J (juxtacapillary) receptors are found in ?
Harrison's 19th Ed. 47e-1

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

5. 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 are sensitive to interstitial edema.

6. The sensation of chest tightness results from ?
Harrison's 19th Ed. 47e-1

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.

7. Metaboreceptors are located in ?
Harrison's 19th Ed. 47e-1

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

Metaboreceptors are located in skeletal muscle.

8. Which of the following is used to measure dyspnea ?
Harrison's 19th Ed. 47e-2

A. Modified Borg scale
B. Baseline dyspnea index
C. Chronic respiratory disease questionnaire
D. All of the above

9. Breathing discomfort during exercise in anemia is due to stimulation of ?
Harrison's 19th Ed. 47e-2

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

10. Metaboreceptors contribute to dyspnea in ?
Harrison's 19th Ed. 47e-2 Table 47e-2

A. Cardiogenic pulmonary edema
B. Anemia
C. Deconditioning
D. All of the above

11. Orthopnea is seen in ?
Harrison's 19th Ed. 47e-3

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

12. "Nocturnal dyspnea" is a feature of which of the following ?
Harrison's 19th Ed. 47e-3

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.

13. Acute intermittent episodes of dyspnea are due to ?
Harrison's 19th Ed. 47e-3

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.

14. Chronic persistent dyspnea is typical of ?
Harrison's 19th Ed. 47e-3

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.

15. Platypnea is dyspnea that occurs in which position ?
Harrison's 19th Ed. 47e-3

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

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

16. Platypnea is a feaature of ?
Harrison's 19th Ed. 47e-3

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

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

17. Which of the following indicate increased work of breathing ?
Harrison's 19th Ed. 47e-3

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.

18. Rounding of the abdomen during exhalation is suggestive of ?
Harrison's 19th Ed. 47e-3

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

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

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

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.

20. Sudden and unexpected dyspneic episodes at rest can be associated with all except ?

A. Pulmonary emboli
B. Spontaneous pneumothorax
C. Metabolic acidemia
D. Anxiety

Laboured breathing is not synonymous with dyspnea. Hyperventilation with metabolic acidemia is rarely accompanied by dyspnea. Sudden & unexpected dyspnea at rest occurs with pulmonary emboli, spontaneous pneumothorax, hypercapnea secondary to breath holding, or anxiety.

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

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 rests is more typical of pulmonary embolization, spontaneous pneumothorax, anxiety. Orthopnea is characteristic of congestive heart failure.

22. Trepopnea most often occurs in patients with ?

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.

23. In chronic bronchitis, which of the following is the predominant sensory experience ?

A. Inability to take in a sufficiently deep breath
B. Difficulty in exhaling
C. Difficulty in inhaling and exhaling
D. Any of the above

Despite the fact that severe limitation of expiratory flow & hyperinflation of lung are characteristic of chronic bronchitis, sensory experience is often that of an inability to take in a sufficiently deep breath rather than difficulty in exhaling.

24. Obstruction of airways is an invariable finding in ?

A. Asthma
B. Chronic bronchitis
C. Emphysema
D. All of the above

Emphysema is a parenchymal disease, it is invariably accompanied by obstruction of airways.

25. Chronic cor pulmonale & respiratory failure is more common in which of the following diseases ?

A. Severe kyphoscoliosis
B. Pectus excavatum
C. Ankylosing spondylitis
D. Rheumatoid arthritis

Severe kyphoscoliosis alters ventilation to produce chronic cor pulmonale & respiratory failure.

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

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.

27. Echocardiographically, which of the following is not a feature of left ventricular failure ?

A. Left atrial dilatation
B. Left ventricular hypertrophy
C. Reduced left ventricular ejection fraction
D. Reduced right ventricular ejection fraction

Left atrial &/or left ventricular dilatation, LVH, reduced LV ejection fraction & disorders of LV wall motion are clues to a left ventricular cardiac etiology. Right ventricular ejection fraction may be low at rest or may decline during exercise in patients with severe lung disease.

28. In neurocirculatory asthenia, the electrocardiographic changes are most often seen during ?

A. Depolarization
B. Repolarization
C. Depolarization + Repolarization
D. Any of the above

In neurocirculatory asthenia, ECG changes are most often seen during repolarization.

29. Frequent sighing respirations & irregular breathing pattern suggest which cause of dyspnea ?

A. Psychogenic
B. Cardiac
C. Pulmonary
D. Diseases of chest wall or respiratory muscles

Frequent sighing respirations & irregular breathing point to a psychogenic origin of dyspnea.

30. Pulmonary edema due to "imbalance of Starling forces" includes all of the following except ?

A. Increased pulmonary capillary pressure
B. Decreased plasma oncotic pressure
C. Endogenous vasoactive substances
D. Increased negativity of interstitial pressure

Imbalance of Starling forces leading to pulmonary edema can be produced by increased pulmonary capillary pressure, decreased plasma oncotic pressure or by increased negativity of interstitial pressure. Endogenous vasoactive substances (histamine, kinins) alter alveolar-capillary membrane permeability (acute respiratory distress syndrome).

31. Which of the following is termed "overperfusion pulmonary edema" ?

A. Increased pulmonary venous pressure without LVF
B. Increased pulmonary venous pressure secondary to LVF
C. Increased pulmonary capillary pressure secondary to increased pulmonary arterial pressure
D. All of the above

Increased pulmonary capillary pressure secondary to increased pulmonary arterial pressure is called overperfusion pulmonary edema.

32. Which of the following is a benign form of altitude illness ?
Harrison's 19th Ed. 476e-1

A. Acute mountain sickness (AMS)
B. High-altitude cerebral edema (HACE)
C. High-altitude pulmonary edema (HAPE)
D. None of the above

AMS is a benign form of altitude illness, whereas HACE and HAPE are life-threatening. AMS and HACE represent opposite ends of a continuum of altitude-related neurologic disorders.

33. High-altitude pulmonary edema (HAPE) is more common in persons of which age ?

A. Infants
B. < 25 years
C. 30 to 60 years
D. > 75 years

Exposure to high altitude in association with severe physical exertion causes pulmonary edema in healthy unacclimatized persons. It is common in persons under the age of 25 years.

34. Prophylactic inhalation of which of the following reduces the incidence of high-altitude pulmonary edema (HAPE) ?
Harrison's 19th Ed. 476e-4

A. β2 agonist salmeterol
B. Steroid
C. Ipratropium bromide
D. Chromolyn

Prophylactic inhalation of β2 agonist salmeterol, administration of oxygen and/or return to lower altitudes reduces the incidence of high-altitude pulmonary edema (HAPE).

35. Monge's disease best relates to ?
Harrison's 19th Ed. 476e-5

A. Severe asthma
B. Pregnant women
C. Chronic mountain sickness
D. High-altitude pulmonary hypertension

Monge's disease refers to chronic mountain sickness of long-term residents of altitudes above 2500 meters that is characterized by excessive erythrocytosis with moderate to severe pulmonary hypertension leading to cor pulmonale.

36. Chronic mountain sickness is characterized by ?
Harrison's 19th Ed. 248

A. Blunted respiratory drive
B. Reduced ventilation
C. Erythrocytosis
D. All of the above

Chronic mountain sickness is characterized by blunted respiratory drive, reduced ventilation, erythrocytosis, cyanosis, weakness, right ventricular enlargement secondary to pulmonary hypertension and stupor.

37. Which of the following diffuse pulmonary edema does not have a hemodynamic origin ?

A. Shock due to sepsis
B. Shock due to hemorrhagic pancreatitis
C. Shock following cardiopulmonary bypass
D. All of the above

Toxic insult to lungs, diffuse pulmonary infections, aspiration & shock, particularly due to sepsis, hemorrhagic pancreatitis & following cardiopulmonary bypass, are associated with diffuse pulmonary edema that clearly does not have a hemodynamic origin.

38. Neurogenic pulmonary edema has been described in ?

A. Central nervous system disorders
B. Peripheral nervous system disorders
C. Central + peripheral nervous system disorders
D. Any of the above

Neurogenic pulmonary edema has been described in patients with CNS disorders and without apparent preexisting left ventricular dysfunction.

39. Overdoses of which of the following heroin preparations is associated with pulmonary edema ?

A. Morphine
B. Methadone
C. Dextropropoxyphene
D. All of the above

Parenteral & oral overdoses of legitimate preparations of morphine, methadone and dextropropoxyphene can produce pulmonary edema.

40. Which of the following leads to the development of interstitial edema ?

A. Rapid evacuation of a large pneumothorax
B. Acute severe asthma
C. Lymphangitic carcinomatosis
D. All of the above

Rapid evacuation of a large pneumothorax causes increased negativity of interstitial pressure. Large negative intrapleural pressures during acute severe asthma may cause interstitial edema. Lymphatic blockade due to lymphangitic carcinomatosis may lead to interstitial edema.

41. Chest radiograph in cardiogenic pulmonary edema shows ?
Harrison's 19th Ed. 47e-4

A. Peribronchial thickening
B. Prominent vascular markings in upper lung zones
C. Kerley B lines
D. All of the above

The chest radiograph in cardiogenic pulmonary edema typically shows an enlarged cardiac silhouette, patchy alveolar filling, typically in a perihilar distribution, peribronchial thickening, prominent vascular markings in the upper lung zones, Kerley B lines, interstitial thickening and perihilar alveolar infiltrates. Pleural effusions are common.

42. Which of the following is false about chest radiograph in noncardiogenic pulmonary edema ?
Harrison's 19th Ed. 47e-4

A. Alveolar infiltrates distributed uniformly throughout lungs
B. Heart size is normal
C. Pleural effusions are uncommon
D. None of the above

In noncardiogenic pulmonary edema, heart size is normal, alveolar infiltrates are distributed more uniformly throughout the lungs and pleural effusions are uncommon. Hypoxemia of cardiogenic pulmonary edema is due to V/Q mismatch and responds to the administration of supplemental oxygen. Hypoxemia in noncardiogenic pulmonary edema is due to intrapulmonary shunting & typically persists despite high concentrations of inhaled oxygen.

Chapter 49. Hypoxia and Cyanosis

43. Pasteur's effect relates to ?
Harrison's 19th Ed. 247

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.

44. Which of the following gene is upregulated in adaptation to hypoxia ?
Harrison's 19th Ed. 247

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).

45. During hypoxia systemic arterioles dilate by opening of ?
Harrison's 19th Ed. 247

A. Na_{ATP} channels in vascular smooth-muscle cells
B. K_{ATP} channels in vascular smooth-muscle cells
C. Cl_{ATP} channels in vascular smooth-muscle cells
D. All of the above

In hypoxia systemic arterioles dilate by opening of K_{ATP} channels in vascular smooth-muscle cells.

46. During hypoxia, pulmonary vascular smooth-muscle cells contract due to inhibition of ?
Harrison's 19th Ed. 247

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

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

47. Acute hypoxia causes a clinical picture resembling ?
Harrison's 19th Ed. 247

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

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

48. Headache in high-altitude illness is caused by ?
Harrison's 19th Ed. 247

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.

49. In severe hypoxia, death usually results from ?
Harrison's 19th Ed. 247

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

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

50. When hypoxia occurs consequent to respiratory failure, hemoglobin-oxygen dissociation curve is displaced to ?
Harrison's 19th Ed. 247

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

When hypoxia occurs due to respiratory failure, PaO_2 declines, $PaCO_2$ rises & Hb-O_2 dissociation curve is displaced to right, with greater quantities of O_2 released at any level of tissue PO_2.

51. Most common cause of respiratory hypoxia is ?
Harrison's 19th Ed. 247

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.

52. In which of the following conditions, PaO2 cannot be restored to normal with inspiration of 100% O2 ?
Harrison's 19th Ed. 248

A. Tetralogy of Fallot (TOF)
B. Transposition of great arteries (TGA)
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_2 cannot be restored to normal with 100% O_2.

53. In anemic hypoxia, the PaO2 is ?
Harrison's 19th Ed. 248

A. Normal
B. Decreased
C. Increased
D. Any of the above

In anemic hypoxia, PaO_2 is normal but due to reduction of Hb concentration, absolute quantity of O_2 transported per unit volume of blood is diminished.

54. In which of the following hypoxia's, venous blood tends to have a high O2 ?
Harrison's 19th Ed. 248

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

55. Example of "Histotoxic hypoxia" is ?
Harrison's 19th Ed. 248

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

Cyanide causes cellular hypoxia because tissues are unable to utilize O_2. As a result, venous blood tends to have a high O_2 tension. This condition is called histotoxic hypoxia.

56. Which of the following is a feature of chronic mountain sickness ?
Harrison's 19th Ed. 248

A. Reduced ventilation
B. Cyanosis
C. Right ventricular enlargement
D. All of the above

In persons with chronic hypoxemia secondary to prolonged residence at a high altitude (>12000 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.

57. Cyanosis is apparent when the mean capillary concentration of reduced hemoglobin exceeds ?
Harrison's 19th Ed. 249

A. 2 gram / dL
B. 3 gram / dL
C. 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.

58. Cyanosis occurs upon ascent to an altitude of ?
Harrison's 19th Ed. 249

A. 2000 meters
B. 3000 meters
C. 4000 meters
D. 5000 meters

Cyanosis is manifest in an ascent to 4000 m (13,000 ft). At this height, FIO_2 & alveolar PO_2 are about 85 & 50 mmHg, respectively & SaO_2 is ~75% leaving more reduced Hb in arterial blood.

59. Cyanosis can be observed in all except ?
Harrison's 19th Ed. 249

A. Marked polycythemia
B. Carboxyhemoglobin (COHb)
C. Methemoglobin
D. Sulfhemoglobin

Patients with marked polycythemia become cyanotic at higher levels of SaO_2 than patients with normal hematocrit values. Cyanosis is also observed when nonfunctional hemoglobin (methemoglobin or sulfhemoglobin) is present in blood.

60. Most common congenital cardiac lesion associated with cyanosis in adult is ?
Harrison's 19th Ed. 400

A. 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.

61. Differential cyanosis is a feature of ?
Harrison's 19th Ed. 51e-6, Harrison's 19th Ed. 1442

A. Tetralogy of Fallot
B. Patent ductus arteriosus

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.

62. Which of the following is suspected when blood remains brown after mixing in test tube & exposed to air ?
Harrison's 19th Ed. 249

A. Marked polycythemia

B. 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.

63. Which of the following is false in Eisenmenger syndrome ?
Harrison's 19th Ed. 249

A. Cyanosis

B. 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.

64. In peripheral cyanosis of extremities, the arterial blood is ?
Harrison's 19th Ed. 249

A. Normally saturated with oxygen

B. Over saturated with oxygen

C. Under saturated with oxygen

D. Any of the above

65. Clubbing without cyanosis is frequent in ?
Harrison's 19th Ed. 250

A. Infective endocarditis

B. Inflammatory bowel disease

C. Jackhammer operators

D. All of the above

Clubbing without cyanosis is frequent in infective endocarditis, inflammatory bowel disease & in jackhammer operators.

Chapter 50. Edema

66. Edema is defined as a clinically apparent increase in ?
Harrison's 19th Ed. 250

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.

67. 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.

68. Movement of water & diffusible solutes from vascular space into the interstitial space occurs at ?
Harrison's 19th Ed. 250

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.

69. Conditions that reduce effective arterial blood volume cause constriction of which of the following ?
Harrison's 19th Ed. 250

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.

70. Which of the following stimulates renin release ?
Harrison's 19th Ed. 250

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.

71. Angiotensinogen is synthesized by ?
Harrison's 19th Ed. 250

A. Kidney

B. Liver

C. Pancreas

D. Lung

Angiotensinogen, an α_2 globulin, is synthesized by liver. Renin converts angiotensinogen to a decapeptide angiotensin I, which is broken down to an octapeptide angiotensin II.

72. Renin is which of the following kinds ?
Harrison's 19th Ed. 250

A. Enzyme

B. Pro-hormone

C. Hormone

D. Cofactor

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

73. Renal effects of Angiotensin II are mediated by activation of which type of Angiotensin II receptors ?
Harrison's 19th Ed. 250

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.

74. Aldosterone is produced by ?
Harrison's 19th Ed. 250

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

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

75. In heart failure, aldosterone secretion is elevated due to ?
Harrison's 19th Ed. 250

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.

76. Activation of Renin-Angiotensin-Aldosterone (RAA) system is most striking in which of the following ?
Harrison's 19th Ed. 250

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.

77. Mineralocorticoid escape phenomenon is best explained by ?
Harrison's 19th Ed. 250

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 retention. 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).

78. Arginine vasopressin (AVP) is best related to ?
Harrison's 19th Ed. 250

A. V_1 receptors
B. V_2 receptors
C. V_3 receptors
D. V_4 receptors

Secretion of arginine vasopressin (AVP) occurs in response to increased intracellular osmolar concentration and 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.

79. Which of the following is an effect of Endothelin-1 ?
Harrison's 19th Ed. 251

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

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

80. Atrial natriuretic peptide (ANP) is stored in secretory granules within ?
Harrison's 19th Ed. 251

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.

81. Brain natriuretic peptide (BNP) is present in ?
Harrison's 19th Ed. 251

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.

82. Released ANP & BNP bind to ?
Harrison's 19th Ed. 251

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.

83. Which of the following is an action of ANP and BNP ?
Harrison's 19th Ed. 251

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.

84. ANP and BNP antagonize the vasoconstrictor actions of ?
Harrison's 19th Ed. 251

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 AII, AVP and sympathetic stimulation. Thus, elevated levels of natriuretic peptides have the capacity to oppose sodium retention in hypervolemic and edematous states.